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THE AMERICAN JOURNAL OF PSYCHIATRY

(16)

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Presidential Papers: 1977

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Basic tool of primitive psychiatry



Herbalist's staff (pa osanyin), with birds (wrought iron), Yoruba tribe, Nigeria, 21 inches.

This staff is topped by a circle to which 16 small birds (a magical number) are attached. The single bird in the middle symbolizes the witch who causes sickness or death. The staff is placed next to the shrine of Osanyin (the god of the herbalist), along with eggs and kola, to neutralize the witch's evil spirit. Sometimes the staff is also placed next to the bed of a sick person to chase away the disease-causing spirit.

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Basic tool of modern psychiatry

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Tablets: 25 mg of the HCl

brand of chlorpromazine

- 'Thorazine' effectively controls psychotic symptoms
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- Unsurpassed clinical experience
- 18 convenient dosage forms and strengths

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Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings:

Caution is recommended when 'Thorazine' is administered for the treatment of vomiting in children.

Antiemetics are not recommended to treat uncomplicated short-term vomiting in children or vomiting of unknown etiology.

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Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudo-parkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis; contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

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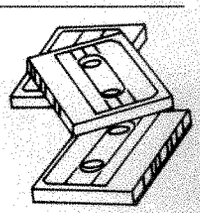
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Capsules 1 mg., 2 mg., 5 mg., 10 mg., 20 mg. Concentrate 5 mg./ml.—Intramuscular 2 mg./ml.

For prescribing information, including adverse reactions and contraindications, please see following page of this advertisement.

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Navane® (thiothixene) (thiothixene hydrochloride)

Capsules: 1 mg, 2 mg, 5 mg, 10 mg, 20 mg Concentrate: 5 mg/ml Intramuscular: 2 mg/ml

PRESCRIBING INFORMATION
Navane® (thiothixene)
Capsules 1 mg, 2 mg, 5 mg, 10 mg, 20 mg (thiothixene hydrochloride) Concentrate: 5 mg/ml, Intramuscular: 2 mg/ml

Actions. Navane is a psychotropic agent of the thioxanthene series. Navane possesses certain chemical and pharmacological similarities to the piperazine phenothiazines and differences from the aliphatic group of phenothiazines. Navane's mode of action has not been clearly established.

Indications. Navane is effective in the management of manifestations of psychotic disorders.

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy* – Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration – As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

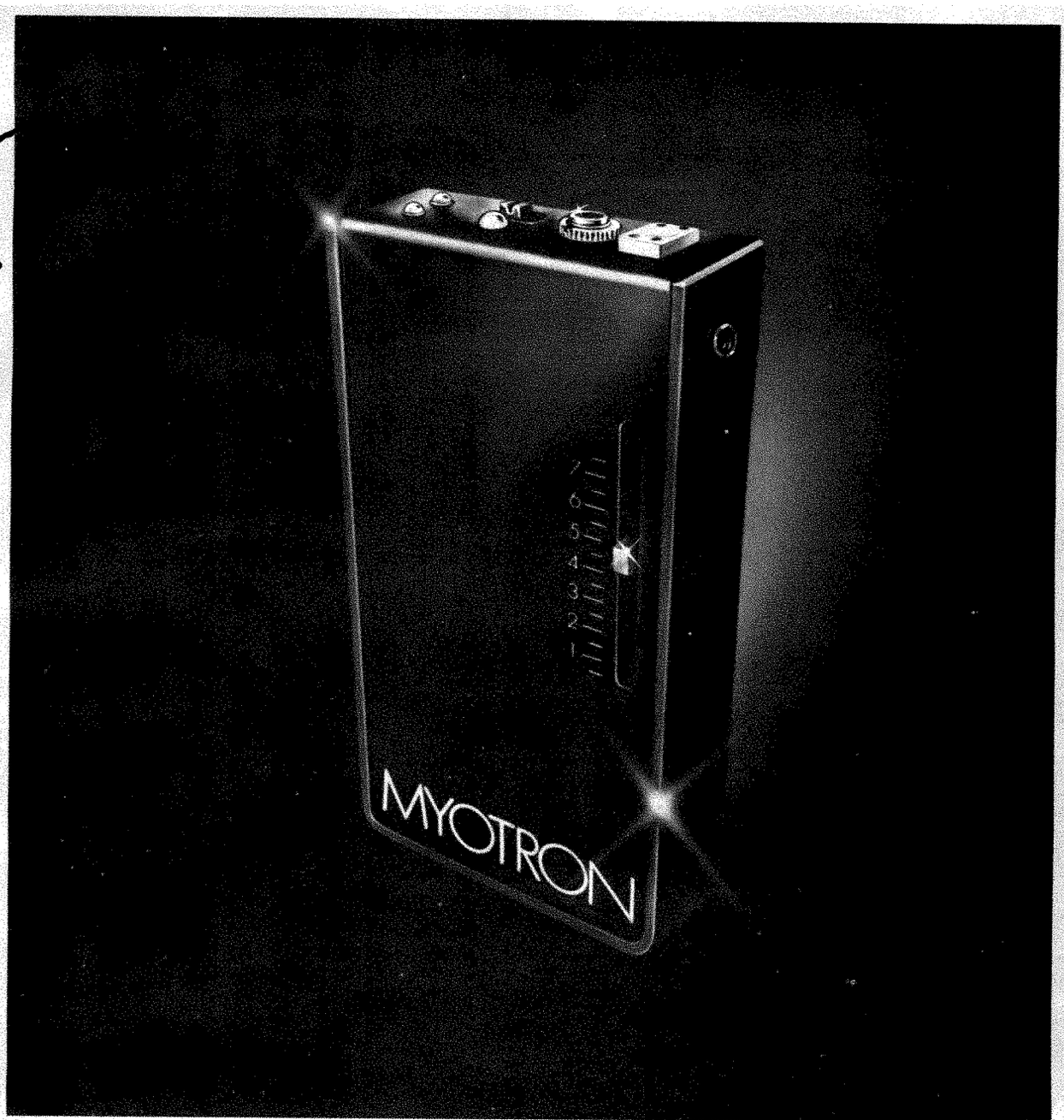
How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg. of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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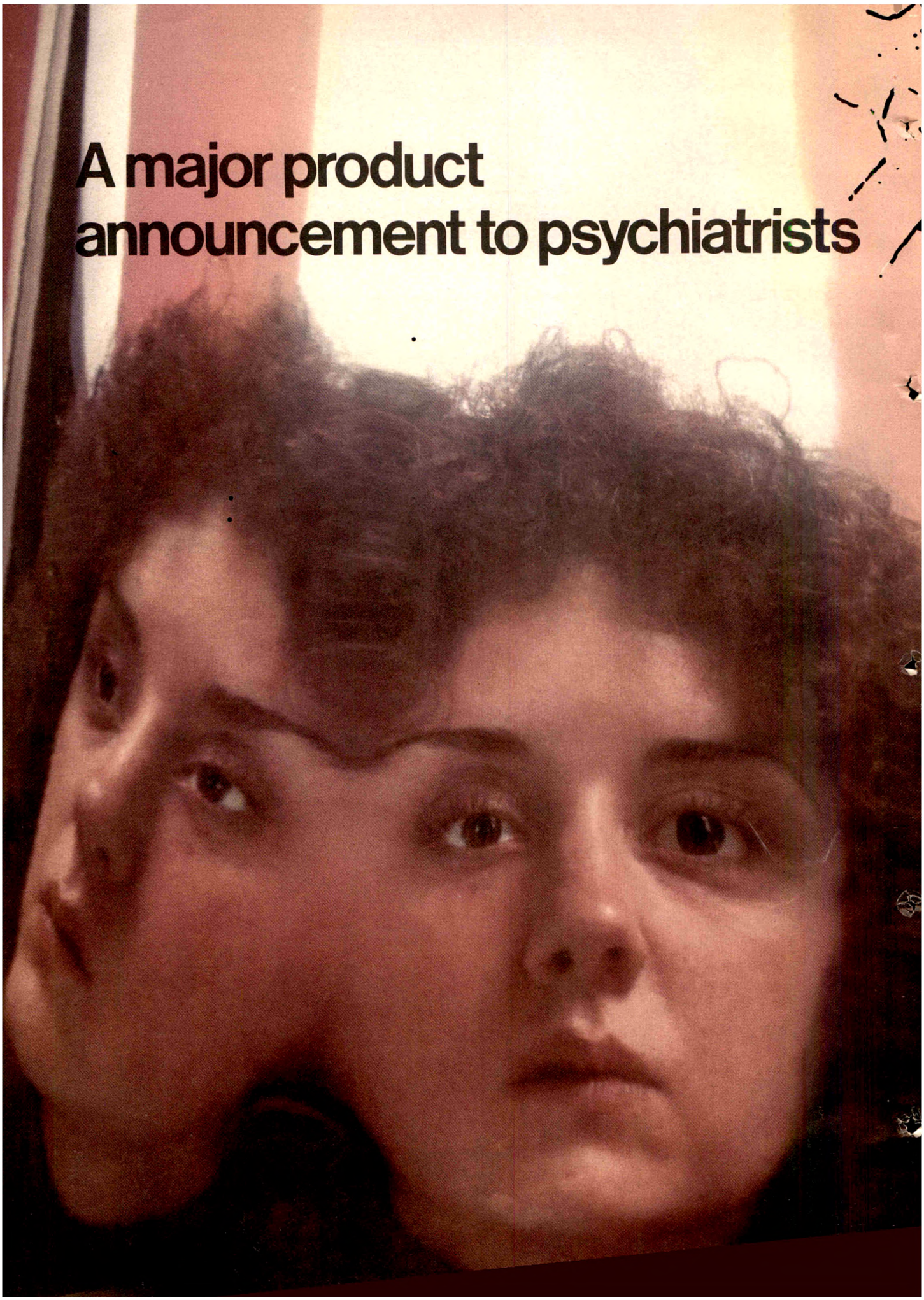
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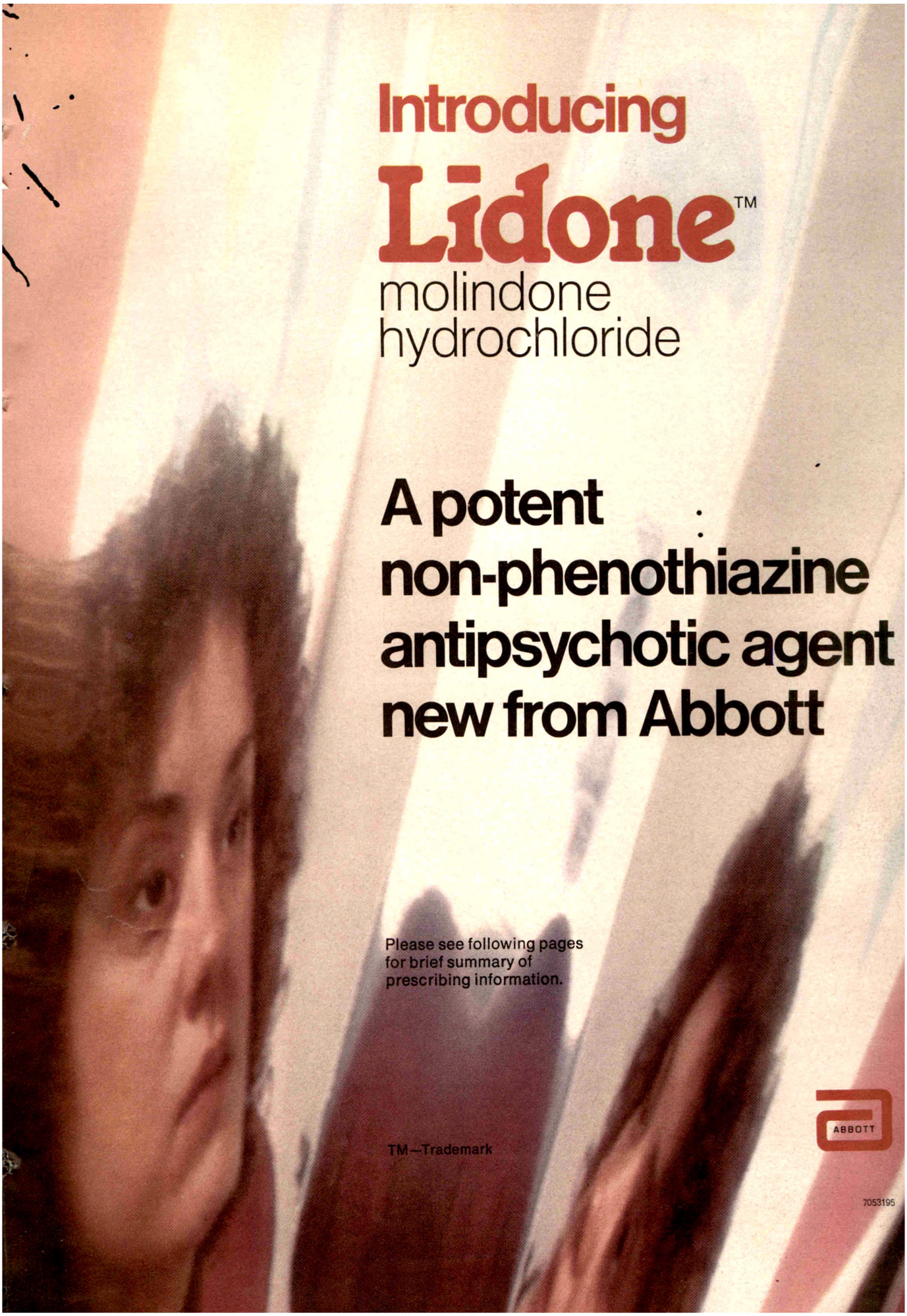
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Introducing
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hydrochloride
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non-phenothiazine
antipsychotic agent
new from Abbott
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for brief summary of
prescribing information.
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molindone
hydrochloride

Why Lidone™ belongs among your first-line antipsychotic agents

Because of its effectiveness

Demonstrated control of target symptoms. Lidone is a non-phenothiazine antipsychotic agent, indicated for management of the manifestations of schizophrenia. As such it offers a clinically proven oral treatment effective over a broad range of target symptoms. Improvement has been reported in:

- Conceptual disorganization[1,2,6,9]
- Unusual thought content[3,4,11,12]
- Hallucinations[3,6,9,11]
- Hostility[1,10,11]
- Bizarre motor behavior[3,11,12]
- Retardation[2,8,12]

Improvement in social outlook. Lidone has enabled patients to display progress in social behavior, and improved social competence and interest.[2,7,9,12,13] Interpersonal relationships have accordingly benefited.



Because of its degree of safety

Seldom causes problem sedation. Lidone is classed among the less-sedating group of major tranquilizers. Initial drowsiness usually subsides with continued use.

Extrapyramidal effects usually controlled. These usually respond to appropriate management without cessation of drug therapy.

Significant hypotension rare; hematologic effects limited. Less than 1% incidence of significant hypotension has been reported in clinical studies, and only rare reports of leucopenia and leucocytosis.

No serious ocular effects, impotence, or excessive weight gain. Unlike some antipsychotic agents, Lidone has not been associated with these conditions.

Persistent tardive dyskinesia not reported. Only a few cases of late-occurring dyskinesia have been noted (reversible upon discontinuation of therapy), and there have been no reports of persistent tardive dyskinesia.

Other side effects comparable to other agents. See Brief Summary for a complete statement of adverse reactions.



Lidone™

molindone hydrochloride

Because of its benefits

Patients accessible to psychotherapy. Patients are seldom over-sedated with Lidone. They tend to remain alert and therefore responsive to psychotherapy and rehabilitation.

Social relationships not disrupted. Improved social behavior has been noted, and side effects which are potentially disruptive to social or family relationships (e.g., weight gain, sexual dysfunction) have not been a problem.

Permits continuity of drug therapy. Few of the side effects require discontinuation of drug therapy.

Most economical non-phenothiazine. The relatively low milligram cost and high milligram potency of molindone make it least expensive of the potent new non-phenothiazine antipsychotics, and also less costly than many leading phenothiazines.[14]

Unique alternative for refractory patients. Molindone is unrelated to other antipsychotic drug families. Hence Lidone may benefit patients who are unresponsive to, or who cannot tolerate, other agents.

Please see following page for brief summary of prescribing information.

Because of its ease of administration

Simple Oral Regimen according to severity of presenting symptoms:

Dose strength	Schizophrenic manifestations	Dosage regimen
5 mg	mild	5 mg three or four times daily. (An increase to 15 mg three or four times daily may be required).
10 mg	moderate	10 mg three or four times daily. (An increase to 25 mg three or four times daily may be required.)
25 mg	severe	Daily dosage as high as 225 mg may be required.

Dosage Comparison at approximately equipotent dose levels:

Agent	Comparable doses[14]
Chlorpromazine	100 mg
Thioridazine	100 mg
Lidone	10 mg
Trifluoperazine	5 mg
Thiothixene	3 mg*
Haloperidol	2 mg
Fluphenazine	2 mg*

*Product not supplied in this exact strength.

Supplied in bottles of 100 capsules

Dose strength	Drug code	Color
5 mg	NDC 0074-5542-13	blue and cream
10 mg	NDC 0074-5543-13	red and cream
25 mg	NDC 0074-5544-13	brown and cream

7053195

Brief summary of
prescribing information for

Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose —
20 mg/kg/day — 2 weeks, no adverse effect
40 mg/kg/day — 2 weeks, no adverse effect

Pregnant Mice oral dose —
20 mg/kg/day — 9 days, slight increase resorptions
40 mg/kg/day — 9 days, slight increase resorptions

Pregnant Rabbits oral dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial and maintenance doses of LIDONE (molindone hydrochloride) should be individualized, and the minimal effective dose should be employed. Elderly and debilitated patients should be started on lower dosage.

DOSAGE SCHEDULE, BASED ON SEVERITY OF SYMPTOMATOLOGY

1. Mild — 5 mg three or four times a day; an increase to 15 mg three or four times a day may be required.
2. Moderate — 10 mg three or four times a day; an increase to 25 mg three or four times a day may be required.
3. Severe — daily dosage as high as 225 mg may be required.

DRUG INTERACTIONS — Potentiation of drugs administered concurrently with LIDONE (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when LIDONE is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE — Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of LIDONE (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of LIDONE (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by LIDONE in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized LIDONE from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of LIDONE is excreted unmetabolized in the urine.) However, poor response of the patient may justify use of these procedures.

While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized LIDONE in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

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TM — Trademark
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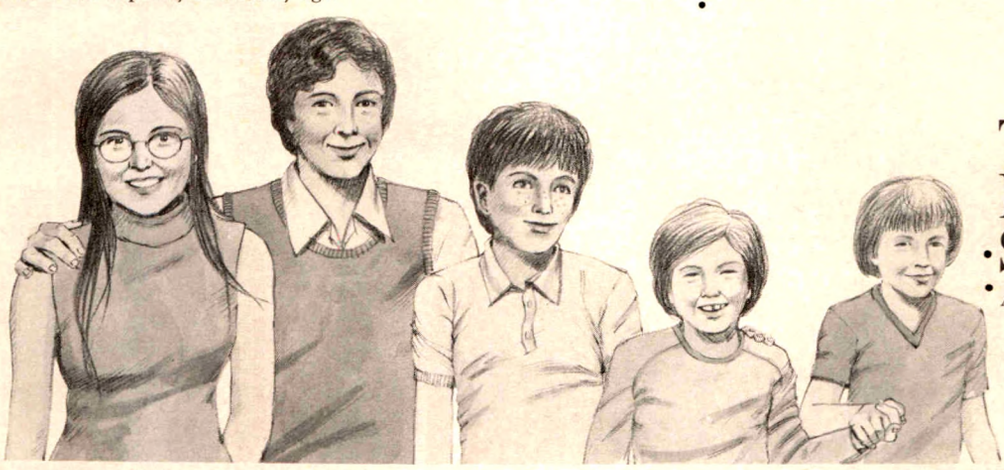
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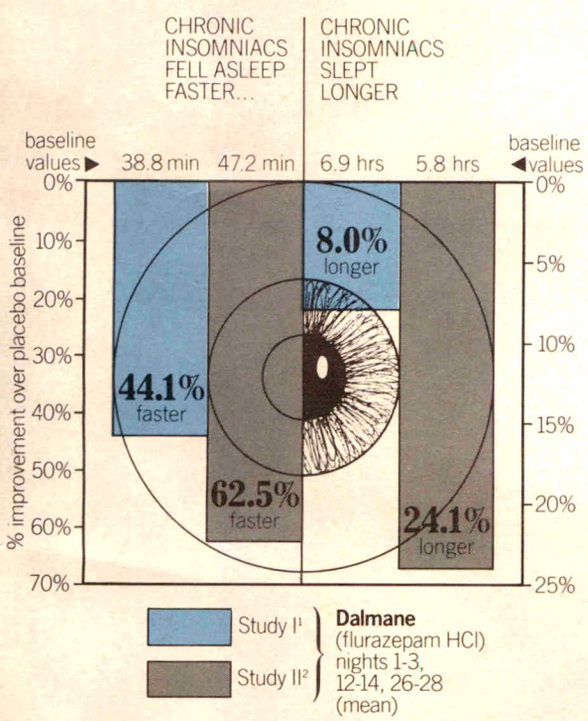
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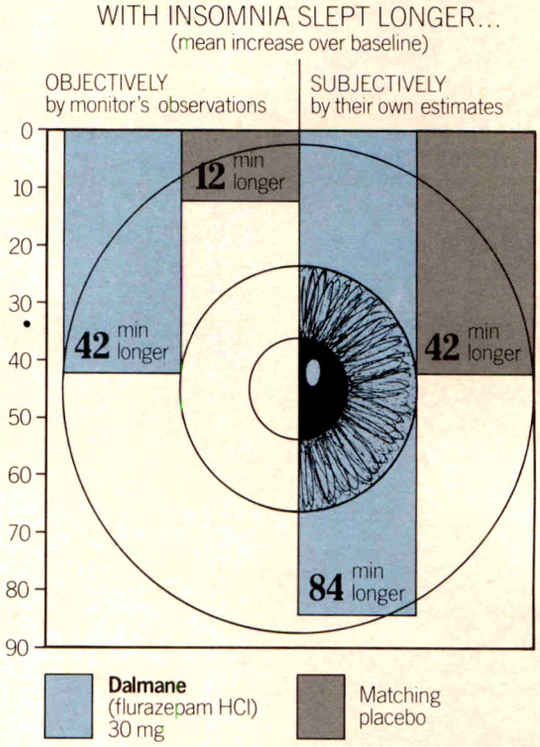
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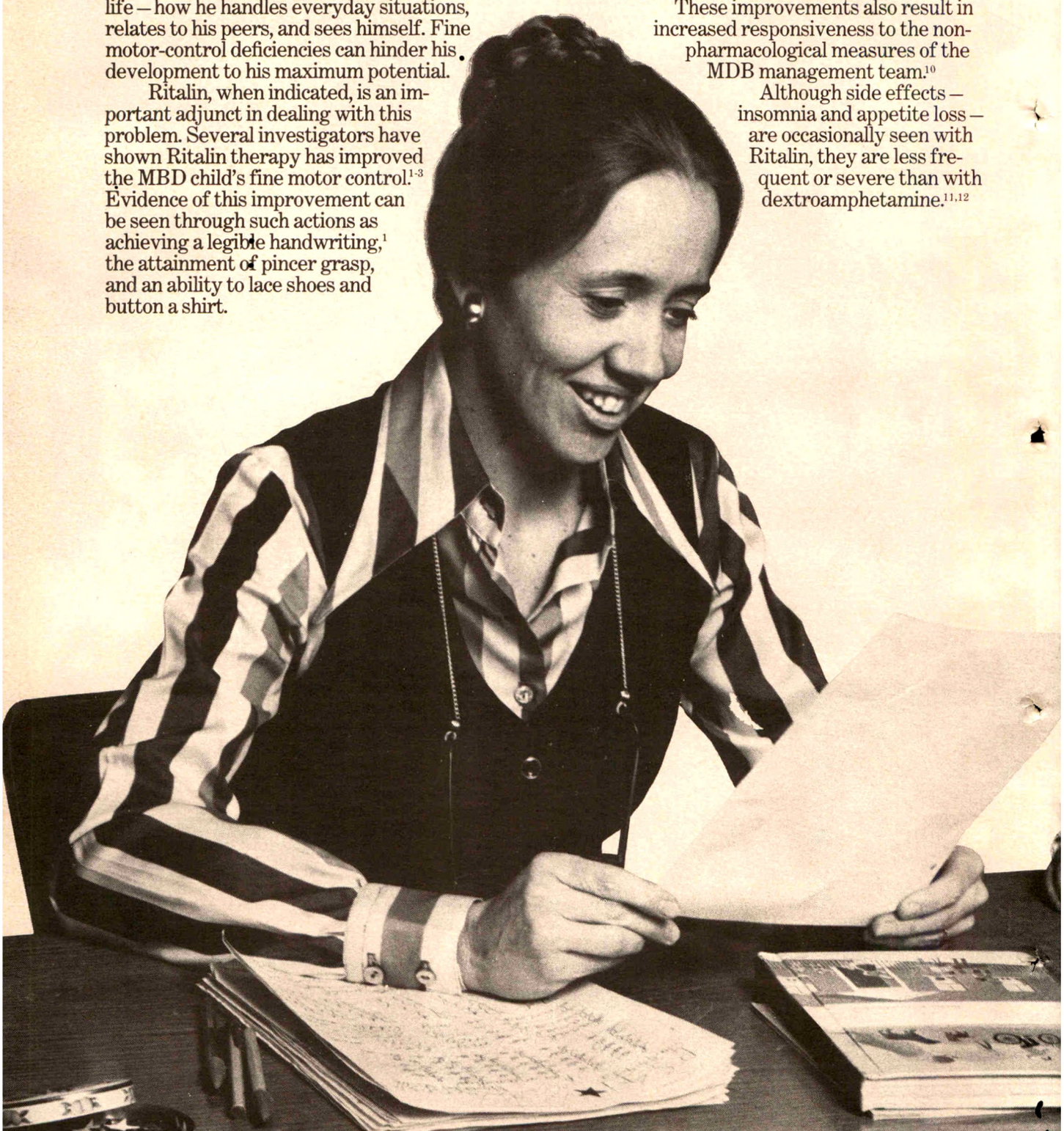
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These improvements also result in increased responsiveness to the non-pharmacological measures of the MDB management team.[10]

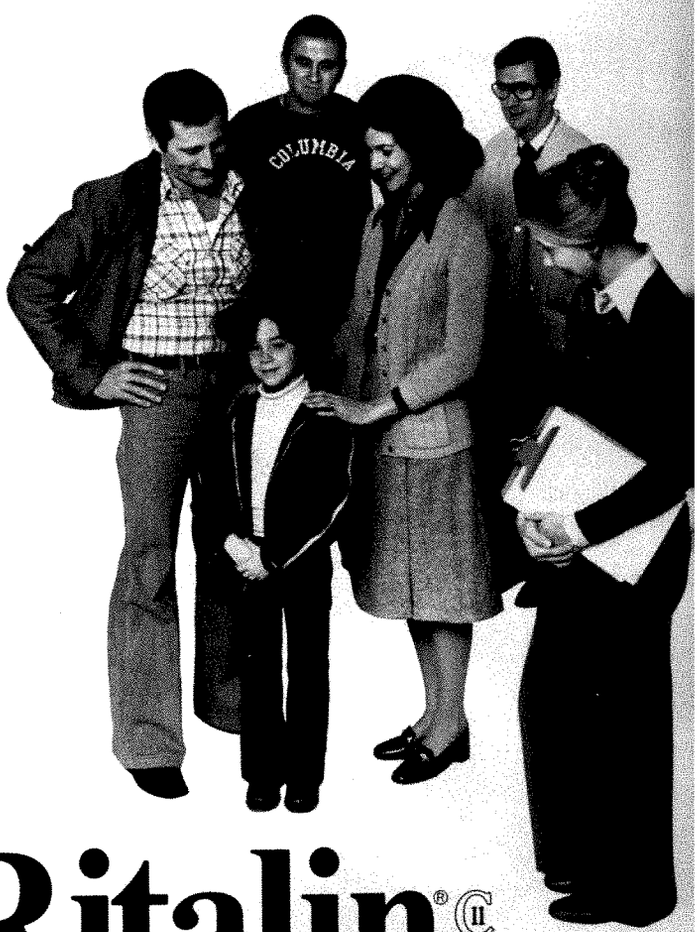
Although side effects – insomnia and appetite loss – are occasionally seen with Ritalin, they are less frequent or severe than with dextroamphetamine.[11,12]



like hen scratchings"

Therapy with Ritalin should be undertaken only after a medical diagnosis of MBD has been made. Dosage should be periodically interrupted. Often these interruptions reveal some "stabilization" in the child's behavior even without medication. In some MBD children they permit a reduction in dosage and eventual discontinuance of drug therapy.

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Ritalin® (methylphenidate) C II

An effective member of the MBD management team

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TABLETS

INDICATION
Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)
Special Diagnostic Considerations
Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.
Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.
Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS
Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS
Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.
Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.
Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.
Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.
Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.
Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions
Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy
Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence
Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.
Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS
Patients with an element of agitation may react adversely; discontinue therapy if necessary.
Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS
Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.
In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION
Children with Minimal Brain Dysfunction (6 years and over).
Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.
If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.
Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.
Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

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Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-Pak® blister units of 100.
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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

1. PSYCHIATRIC EDUCATION: PROLOGUE TO THE 1980's, Library Bound, 544 pages, $15.00.

This is a substantive, highly readable, report of the Conference, written by Anne H. Rosenfeld with an Editorial Board comprising: Ewald W. Busse, M.D., Chairperson of the Conference, Walter E. Barton, M.D., Bernard Holland, M.D., Donna Norris, M.D., Melvin Sabshin, M.D., Jeanne Spurlock, M.D., and Robert L. Robinson, M.A. (Staff Consultant).

The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

2. THE WORKING PAPERS OF THE 1975 CONFERENCE ON EDUCATION OF PSYCHIATRISTS Paper bound, 432 pages, $12.50.

Sixty two leading psychiatric educators served on seven preparatory commissions, one for each of seven topic areas, and prepared advance working papers as a basis for Conference deliberations. The topics and Commission Chairpersons were: Social Issues and Needs (James N. Sussex, M.D.); Goals and Objectives (Morton F. Reiser, M.D.); The Resident (David R. Hawkins, M.D.); Content, Methodology, Faculty and Environment (Milton Greenblatt, M.D.); Research (Daniel X. Freedman, M.D.); Economics (Herzl Spiro, M.D.); Evaluation and Quality Control (Eric Pfeiffer, M.D.). The Chairpersons, together with the Conference Chairperson, served as the Editorial Board for this volume.

Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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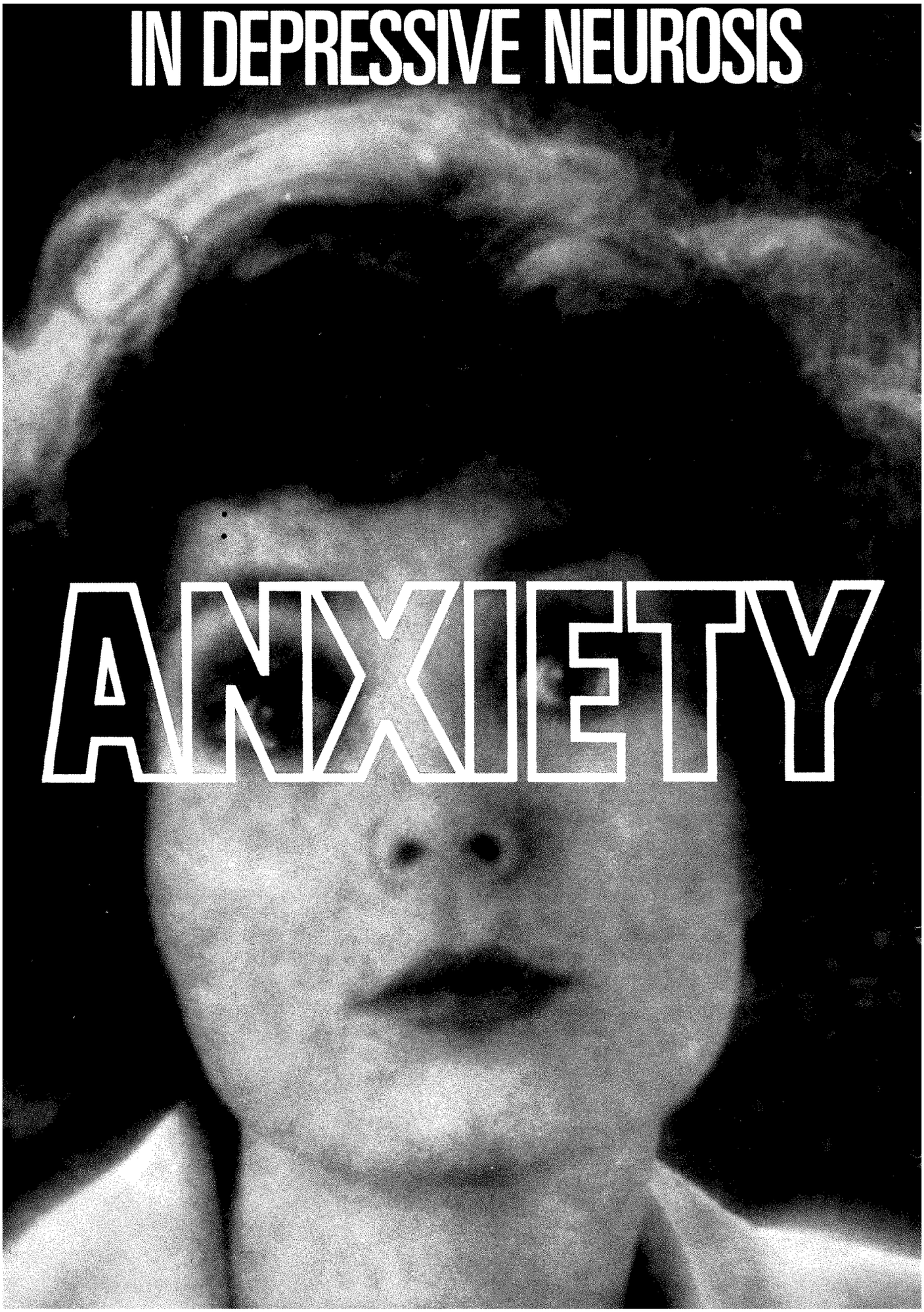
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in short-term therapy of moderate to marked depression
with variable degrees of anxiety in patients with depressive neurosis

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TABLETS: 10 mg, 15 mg, 25 mg, and 50 mg thioridazine HCl, USP

RESULTS OFTEN SEEN IN A WEEK

For Brief Summary, please see following page.

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in short-term therapy of moderate to marked depression with variable degrees of anxiety in patients with depressive neurosis

MELLARIL®
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RESULTS OFTEN SEEN IN A WEEK

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient. In adults with depressive neurosis the usual starting dosage is 25 mg t.i.d. and the dosage ranges from 10 mg b.i.d. to q.i.d. in milder cases to 50 mg t.i.d. or q.i.d. for more severely disturbed patients; the total daily dose ranges from 20 mg to a maximum of 200 mg.

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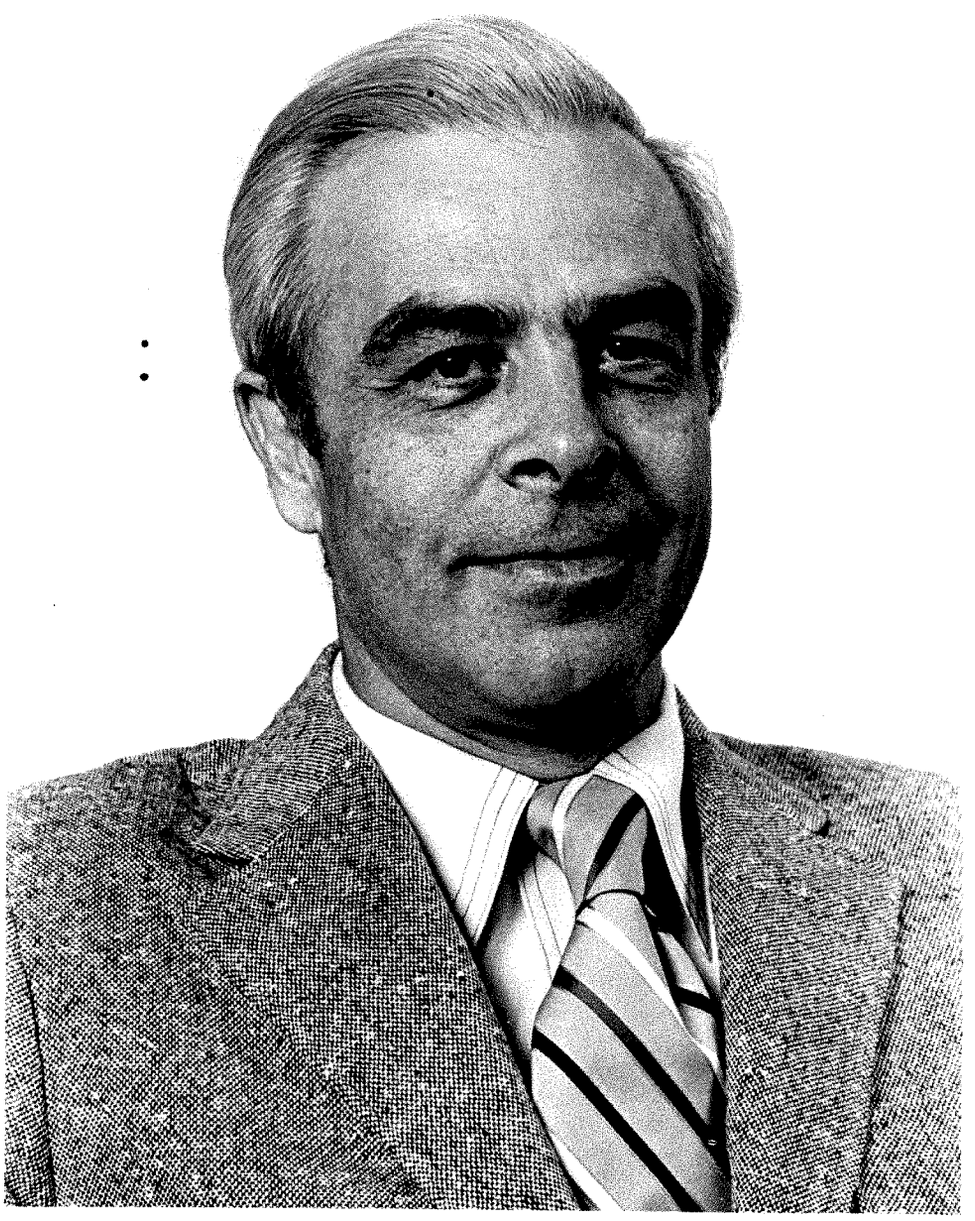
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ROBERT W. GIBSON, M.D.

Presidential Address: A Profession Worthy of the Public Trust

BY ROBERT W. GIBSON, M.D.

THE THREEFOLD escalation of health care expenditures from 1965 to 1975 has prevented the United States from making good on the commitment that adequate health care is a right. Government leaders, third-party payers, and the consumer, responding to inflationary pressures, have served notice that the amount and allocation of resources—dollars, facilities, personnel, and supplies—will be contingent on the ability of those of us who provide care to demonstrate that treatment services not only are effective but are delivered efficiently.

The leadership role of health care professionals has steadily eroded. Government planners and managers are setting health policy and establishing priorities. In many instances they lack clinical experience, and sometimes they seem insensitive to the needs of the mentally ill. If we as mental health professionals fail to act, we will lose our ability to influence the quality and character of care provided to our patients.

Protestations of outrage may provide catharsis but are not enough. We must acknowledge that abuses do occur and that the health care delivery system has many flaws. Because health is influenced by many social forces, psychiatry interacts with a multitude of systems, making rational control difficult and complex.

But increased government regulation is not the solution; more often it is the problem. In my judgment it is our professional responsibility to strengthen our own systems of quality assurance, show that external regulation is only minimally necessary, and reaffirm the public trust. This is why I requested that the theme of this Toronto meeting be "Professional Responsibility and the Public Trust."

In this address I hope to demonstrate that: 1) while the national concern about increasing health care costs is valid, simply adding government regulation is *not* the solution—in fact, inappropriate regulation can cause many problems; 2) APA has moved into many new areas and has attempted to strengthen its activities in other areas so as to enhance prospects for a more rational and effective mental health care system; and 3) we recognize that a new level of accountability is expected of us and we stand prepared, through our peer review and related efforts, to show that we are equal to the challenge.

Presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2–6, 1977.

Dr. Gibson was 105th President of the American Psychiatric Association, 1976–1977. He is Medical Director, Sheppard and Enoch Pratt Hospital, Towson, Md. 21204.

THE PROBLEM OF RISING HEALTH COSTS

In 1976, total expenditures for health in the United States amounted to $139.3 billion, or $638 per person; these expenses claimed 8.6% of the gross national product, with 42.2% coming from tax dollars. These 1976 expenditures represented a 14% increase over fiscal year 1975, and inflation continues at close to 10% annually. At 8.6% of the GNP health care expenditures have a significant impact on the overall economy, and with over 42% of the funds coming from tax dollars the cost of health care has become a significant political issue and provides the government with enough leverage to control the system.

Alarmed by runaway costs, legislators are hedging on the original declaration that health care is a right. For example, the statement of purpose of the Health Planning Act (PL 93-641) stipulates: "The achievement of equal access to health care at a reasonable cost is the priority of the federal government." Equal access is now a priority, not a right; even that is conditional on "reasonable cost." The determination of what is a reasonable cost will be a political decision, not a medical decision.

National health insurance is still a significant political issue but is being pushed with less vigor because of concern about costs. Instead, the current government emphasis is on efforts to control and reshape the health care system and presumably to make it more efficient and cost effective.

For example, appropriations to assist health maintenance organizations are intended to promote organized systems of health care as opposed to individual private practice. Legislation authorizing Professional Standards Review Organizations is designed to increase participation by physicians in utilization review activities. The Health Planning Act establishes a network of local, state, and national citizen groups to plan, coordinate, and control health care resources. The Health Professions Educational Assistance Act of 1976 provides the mechanisms to control that most important of all health resources—trained personnel.

It appears that the strategy is to move cautiously on national health insurance until the various controls are in place and working effectively. This reflects the conviction of government planners that simply adding dollars and benefits will not improve the health of our nation. The message is plain. Payers, whether they be federal or state government or insurance carriers, will make resources available only to the degree that providers can demonstrate they can control these resources and use them effectively and efficiently. Evaluation and quality assurance are now preconditions that must be met before mental health benefits will be increased.

SPECIAL CHALLENGES FOR PSYCHIATRY

Satisfying such expectations of accountability is difficult for all segments of the health care industry. Regrettably, mental health is often singled out for special challenge while other types of care are not. Consider that the National Ambulatory Medical Care Services (NAMCS) estimates that in 1973–1974, visits to physicians numbered 14 million for colds, 18 million for back problems, 12 million for headaches, 12 million for fatigue, and so forth. Many of these conditions were probably psychosomatic, many were not amenable to definitive medical treatment, and outcome and cost benefit would be impossible to judge. But expenditures for such care are not challenged to the same degree as mental health services.

Some of the more difficult tasks confronting psychiatry are: 1) completion of valid outcome studies; 2) refinement of standards and criteria; 3) achieving a balance between humanistic concerns and cost effectiveness; and 4) preservation of confidentiality. Psychiatrists have not been derelict; as we interact with other social systems the increased exposure inevitably opens us to new challenges and new demands to justify our practice.

BUREAUCRATIC INTRUSIONS

The demands frequently take the form of bureaucratic intrusion seemingly to the point of harassment. A task force on regulation created by the Hospital Association of New York State to assemble information on the scope of the problem found that no less than 164 different regulatory agencies had some jurisdiction over hospitals in the state (1). Acknowledging that government has the right to regulate hospitals, the task force, in its study of the impact of regulation, wondered why 25 separate agencies must review hospital admitting procedures, why 33 agencies protect patients' rights, and why 31 agencies insure patients' safety. The task force found that during the past 10 years, while the total number of employees in the state health department went from 2,000 to 3,500, the number of accountants went from 2 to 170 and the number of attorneys went from 3 to 30. But the number of physicians declined from 69 to 60. The state's new austerity budget calls for 568 new regulatory positions in the department!

Most of the regulatory efforts are well intentioned. But unfortunately the controls overlap, are sometimes contradictory, and add to the cost of care. Preoccupation with these multiple requirements may distract mental health professionals from their main goal: providing high quality treatment services appropriate to the patient's need. In exasperation, staff protest that it has become more important to document that some arbitrary requirement has been met than it is to do something that will be really helpful to the patient.

Despite the heavy emphasis on reducing length of stay, an October 1976 report commissioned by the Department of Health, Education, and Welfare (2) estimated that utilization controls would reduce the cost of additional services under the American Medical Association's national health insurance proposal by $.3 billion (less than .2%) and under the Health Security Bill by $1.4 billion (only .7%). Regulation adds to costs through special reports, legal services, requirements for documentation, and the like. Beyond that comes the time of clinicians diverted from patient care as they are kept busy meeting the requirements. It would not surprise me if these activities add 5% to 10% to the health care budget. Surely our profession can develop a better system.

CONSEQUENCES OF REGULATION BY THIRD-PARTY PAYERS

The funding mechanism is seen as a tool to manage the health care system through planning, organizing, coordinating, staffing, motivating, and controlling. Although other nations have established a national health service under which the government owns the facilities, hires the personnel, chooses locations for dispensing care, and by taxation pays whatever costs are required, there appears to be no significant support in this country for such a solution. Instead, the approach will probably be one of voluntary participation by providers and possibly by consumers. More liberal benefits and coverage for participants will be strong inducements to consumers to seek care from those providers who are a part of the system. Government planners,

by reshaping the health care system, hope to overcome maldistribution of providers, inaccessibility of services, and inappropriate utilization of personnel.

A troublesome consequence is that as our health care system becomes more complex, the decision-making process is removed further and further from those providing direct patient care. Clinicians find it increasingly difficult to participate in top management decisions such as the allocation of resources and the setting of priorities.

Third-party payers, both government and private, have already gained strong control over the health care system. Under Medicare and Medicaid, government officials decide what benefits should be available, set standards for providers, and establish criteria for the services themselves. All national health insurance proposals contain schemes of economic incentives and disincentives to reshape the system.

The techniques used by large private health insurance carriers, such as the federal employees programs, provide insight into the way the funding mechanism can influence psychiatric care. Faced with escalating costs, insurance carriers have initiated claims review: a patient seeks help, a practitioner provides the service, a claim is submitted to the third-party payer, the claim is either paid or it is not. This last step establishes a crucial feedback to the system. As long as claims are paid, psychiatrists continue to provide services in accordance with their best clinical judgment.

Clinicians are disturbed when claims review leads to the denial of treatment for a particular patient. This is of grave concern to the patient, and the long-term consequences are even more serious. Professionals would in most cases continue to provide the services they deem medically necessary and appropriate without regard for payment. Nevertheless, decisions about payment over a period of time would undoubtedly exert an influence. Psychiatric care might evolve in a sequence akin to the process of the survival of the fittest, or perhaps it should be called "the survival of the most reimbursable." It could become a monstrous application of the behavior modification technique. Presumably those facilities and those practitioners who favored the kind of treatment supported by third-party payers would flourish, while the rest would languish.

There is no question that those who pay for care have the right to make determinations about the care. They are accountable for the expenditures of tax dollars in the case of government programs, the premiums of subscribers in the case of private insurance, or the fringe benefits in the case of a union or employer. There is nothing inherently wrong with using funding to better organize the health care system. The danger is that decisions will be based on standards and criteria that do not reflect the best professional opinion of practicing psychiatrists—that the requirements of administrators and managers will outweigh the needs of patients.

Increasing regulation and the growing demand for accountability disturb the professional because they challenge the basic concept that members of the profession are capable of self-discipline. Have we failed to maintain adequate standards of professional responsibility? Do our systems overlap so much that responsibility must be shared? Have professionals lost the public trust?

Many clinicians, particularly physicians, are so accustomed to society's sanction they are convinced that complete autonomy is a prerogative automatically granted with the professional degree. This is simply not true. Practice of a profession is not a right, it is a privilege—a privilege that must be earned and repeatedly reaffirmed by responsible action and behavior.

EXPANDED APA ACTIVITIES

I will return to this matter of professional responsibility shortly. But first I want to highlight for you a few of the extensive activities APA has undertaken recently to increase our level of participation in decisions that affect our profession and the quality of care given to our patients.

Government Relations

The Joint Commission on Government Relations, composed of members from each of the seven geographic areas, coordinates the legislative network of district branch representatives. Mr. Jay Cutler, an attorney who served as Minority Counsel to the U.S. Senate Health Subcommittee and the Alcoholism and Narcotic Subcommittee and more recently as Minority Counsel and Staff Director of the Senate Labor and Public Welfare Committee, has been appointed Special Counsel and Director of the expanded Office of Government Relations. The annual Institutes on Governmental Operations (the sixth was held in March) have helped district branch legislative representatives to become more knowledgeable and effective in their presentations to government leaders. The Joint Commission has developed 10 key principles dealing with private practice, continuing education, malpractice, primary care, PSROs, nonphysicians in NHI, pluralism in the health care system, professional responsibility for medical education, input into regulations, and accountability. Legislative proposals are continuously monitored, contacts with key Congressional leaders made, and testimony provided as needed.

President Carter has shown a special interest in mental health by appointing a Presidential Commission on Mental Health under the honorary chairmanship of Mrs. Rosalyn Carter. This commission has been charged with preparing a final report by April 1978 that will, among other things, project needs for dealing with emotional stress over the next 25 years, find ways to more efficiently treat the underserved mentally ill, identify the types of research and educational systems needed, and estimate the cost of programs and how financing should be divided among fed-

eral, state, and local governments and the private sector. This 20-person commission has a strong consumer majority, but among its membership are 3 psychiatrists: Allan Beigel, M.D., Mildred Mitchell-Bateman, M.D., and George Tarjan, M.D. There are 2 additional physicians, one of whom is closely allied to child psychiatry; the other has had considerable experience with drug abuse problems. Advisory groups dealing with a number of special areas are being appointed. APA will work quite closely with this commission.

Commission on Public Affairs

To back up our governmental activities through a strong base of public support, the Joint Commission on Public Affairs has begun to set up mechanisms to enable the Association to become more active in informing the general public about the psychiatric profession, to monitor public attitudes toward psychiatry, and to cooperate on a continuing basis with the media and other institutions that influence the public's view of psychiatry.

Some of the means for achieving these goals are: publication in the *American Journal of Psychiatry* of guidelines for psychiatrists working with the communications media, establishment of a roster of press contacts, formation of a public affairs network with representation in each of the district branches, a public opinion survey, identification of friends and advocates of psychiatry, responding to attacks on psychiatry, informing major networks of the APA position on televised violence, and a planned symposium for science writers in the late fall of 1977.

Commission on Standards of Practice

The Commission on Standards of Practice and Third-Party Payment has represented APA on issues concerning government programs and private insurance carriers. In 1974, when serious cutbacks in the Blue Cross and Blue Shield federal employees program and in CHAMPUS were threatened, the commission spearheaded an effort that preserved the psychiatric benefits in these programs. A dialogue has been maintained with various third-party payers concerning standards and criteria for care, procedures for reporting claims, and the claims review process. A joint task force of APA and the federal employees Blue Cross and Blue Shield program is revising guidelines to reflect professional judgments of psychiatrists; peer review will be incorporated into the review process of claims for psychiatric care. The commission and the APA's Medical Director have also been authorized by the Board of Trustees to enter into a contract to help CHAMPUS set up a retrospective review system regarding claims presented to CHAMPUS for payment. APA will be asked to identify psychiatrists willing to participate in this system (with reimbursement at a level comparable to their regular hourly rate) and to provide an educational program for the psychiatrists participating.

Peer Review

Peer review activities have been advocated by APA as the most appropriate way to meet demands for accountability. The Peer Review Committee has prepared *Model Screening Criteria to Assist Professional Standards Review Organizations* for the AMA PSRO manual and has published the *Manual of Psychiatric Peer Review*. Richard Dorsey, M.D., an APA member, has consulted with about 40 district branches over the past year, supplying assistance in the development of their peer review efforts. In March of this year a National Conference on Peer Review brought together third-party representatives, consumers, technical experts, and psychiatric leaders to further refine and develop the APA peer review system. It is anticipated that the proceedings of this conference will be published by early fall.

Interprofessional Relations

Differences among the major mental health professions—psychiatry, psychology, social work, and nursing—are being discussed and sometimes heatedly debated. *Advance*, published by the Association for the Advancement of Psychology, has repeatedly criticized the officers of the Board and the Assembly of the American Psychiatric Association. In an article headlined "No Peace in Our Time" (3), *Advance* objected to what they labeled the "Gibson Syndrome." In testimony on national health insurance before the House Interstate and Foreign Commerce Committee I had said, "In my judgment, the diagnosis and determination that services are medically necessary should be made by a physician, a psychiatrist."

Also, the Association for the Advancement of Psychology, in a complaint filed with the Federal Trade Commission, alleged: "The Joint Commission on Accreditation of Hospitals standards are nothing less than artificial and predatory barriers . . . imposed with no medical, social, or economic justification. . . . [This] effectively and systematically prevents hospital patients from exercising their free choice of attending therapist. . . . Indisputedly, psychiatrists benefit from this arrangement." The Federal Trade Commission, which is still considering this complaint, was provided with a point-by-point refutation of the allegations that was prepared by APA's legal counsel.

Changes in traditional staffing patterns, greater emphasis on the team approach, and shifts in the level and degrees of responsibility are evolving. APA is not against change as long as it is rational and there are sufficient safeguards to maintain the safety and quality of treatment. An Ad Hoc Committee on Professional Relations has met for almost 3 years with representatives from psychology, social work, nursing, and the Mental Health Association to define more precisely the areas of practice appropriate for each of these professions. The presidents and executive directors of the two APAs have also met to improve communication between our two associations. The mental health pro-

fessions are confronted by difficult questions. In articulating our positions scientific study is preferable to power politics. Considered judgment, restraint, and statesmanship are needed; our professional associations have an opportunity to work together collaboratively and responsibly.

Confidentiality

Concerning problems affecting the confidentiality of the doctor-patient relationship, I think we can be proud of the leadership that APA has provided in recent years. In a series of steps beginning with the Conference on the Confidentiality of Health Records held in Key Biscayne in November 1974, APA was able to organize 17 national organizations into forming the new National Commission on Confidentiality of Health Records in Washington, D.C. The commission, chaired by APA Past-President Alfred M. Freedman, is an action-oriented organization dedicated to coping more effectively with the growing threat to the confidentiality of health records implicit in the growth of third-party payments, "need to know" agencies, and computer technology.

Role Conflicts

We may also be pleased that after several years of effort, APA was able to conduct the first major conference on problems of "role conflicts in psychiatry" in cooperation with the Hastings Institute of Society, Ethics, and the Life Sciences. While we must be concerned and alarmed about the abuses of psychiatry and psychiatrists in other countries, it behooves us also to cast a questioning eye at our own practices in the service of patients, on the one hand, and institutions, on the other, in the complex roles we play in the military, in universities, in hospitals, in prisons, and in other institutional frameworks. The report of the Hastings conference, which will be published within the next several months, will go far to raise our consciousness about the philosophical, ethical, and socioeconomic aspects of this problem area. Also, at the request of APA an open forum will be held during the World Congress of Psychiatry meetings in Hawaii to deal with the use of psychiatry to control political dissent.

Commission on Judicial Action

Through its Commission on Judicial Action, APA has proven remarkably effective in monitoring federal Courts of Appeal and Supreme Court proceedings and in submitting amicus curiae briefs in cases that promise great positive or negative impact on the treatment of the mentally ill and our future as a profession. The resources we can put into this important area are of course limited and the legal costs high. However, we anticipate increased activity following the appointment last January of Mr. Joel Klein, a member of a Washington, D.C., law firm, as counsel; he will coordinate the activities of the commission and represent APA in its role as amicus curiae in cases selected by the commission.

Continuing Medical Education; Certification and Recertification

Continuing education requirements were voted by the APA membership in 1974; now we have to see that the system is fully implemented in a manner designed to help our members meet state licensure requirements as well as APA standards. For example, we are changing the format of our annual meeting to enable members to earn more continuing education credits than ever before. For those who are unable to attend the annual meeting, the Psychiatric Knowledge and Skills Self-Assessment Program will serve a similar purpose. We have also been collaborating with the AMA in its continuing education activities.

A rational plan for insuring the continuing competence of psychiatrists through certification and recertification programs is making headway under our auspices. While we are mindful of the heterogeneity of our membership, we see these efforts as a symbol of our strong commitment to meeting our professional responsibility.

ADVOCATES OF THE MENTALLY ILL

In these extensive activities our Association rarely has explicitly designated authority. No longer does the public automatically accept our opinions and recommendations in areas of professional expertise. We must rely on educative efforts, carefully reasoned professional opinions, and effective presentations and leadership.

As we have become more active we have—through our national officers, the Assembly of District Branches, the district branches themselves, and our Medical Director and Central Office staff—engaged in a myriad of activities that go far beyond the scope of medical practice and scientific inquiry. Although the main concern of APA continues to be the scientific advancement of psychiatry, we must use this knowledge to serve as advocates for the mentally ill.

Some APA members interpret our actions as "selling out to the enemy" and repudiating what they believe to be our professional prerogatives. They have a point; there *is* a delicate balance. But we must recognize that controls and regulatory authorities have already been established. We do not have absolute authority over the manner in which we perform professional tasks. We do not have complete control over the setting or conditions under which we practice. Militant refusal to become involved may succeed in some instances, but these victories are shortlived. Control of health care is no longer the professional prerogative of the physician. Health care, much like a public utility, belongs to the public.

The involvement of APA is not a passive accommodation. It is active, with particular emphasis on advocacy of the needs of patients. APA does represent the interests of its members, but it does so as a profession,

not as a trade association. There need be no fundamental conflict. Patients and psychiatrists share a common goal—adequate health care for the mentally ill. Patients want care to be accessible, appropriate, and timely. Psychiatrists want adequate resources to provide such care and the opportunity to practice their profession under conditions that are in the best interests of the patient.

A NEW LEVEL OF ACCOUNTABILITY

In this day and age of accountability it is unrealistic to think that any profession can retain total control over professional tasks. We can achieve significant influence only by active participation with government, the consumer, third-party payers, and allied professions. We have known this for years, but we must now recognize that the current climate is more complex and more demanding. In the interest of quality patient care, responsible professional action is essential.

Of high priority is the introduction of peer review into the systems used for claims review and quality assurance. Claims review conducted by third-party payers has many disadvantages: 1) clinical judgments are susceptible to financial pressures, 2) third-party payers seldom have the clinicians capable of adequate review, 3) standards and criteria are established by the payers and not the providers, 4) the criteria are not open to public scrutiny, and 5) the findings of such reviews may be used by third-party payers to change the basic contract agreements under which treatment is provided. In short, reviews conducted by those outside the profession are designed to control costs, not to improve the health care system. They become a constraint imposed upon the professional.

On the other hand, if psychiatrists are made a part of the review system: 1) clinical judgments will reflect current psychiatric concepts; 2) psychiatrists with specialized competence will conduct meaningful clinical studies; 3) standards and criteria will be established, refined, and modified by the profession; 4) the standards and criteria will be open to public scrutiny; 5) and the findings will be used to improve psychiatric care as well as to control abuse and unnecessary expenditures. Claims review will no longer be a constraint; it will become a part of our professional quality assurance system—a resource contributing to the improvement of mental health care.

Peer review further illustrates the need for participation at both the national level and the local level. At the national level the Association, supported by the Office of Peer Review, has developed guidelines, influenced legislation and regulations, carried the message to members of Congress, worked with the major insurance carriers, and provided consultation to district branch peer review committees. But the actual review by peers must be carried out at the local level in accordance with standards and criteria developed for the area. Agreements with local carriers, the review of cases, and corrective actions must be done by district branch committees. The strength and the effectiveness of the American Psychiatric Association is based on the active participation of the total membership.

These are challenging times. A new level of accountability is expected from all professions. But this is not the problem. The danger is in allowing leadership to slip away from the mental health professions. This would be catastrophic, because the essence of professionalism is control over both the performance of professional tasks and the setting and conditions under which such tasks are performed. Individually and collectively we must demonstrate through responsible action that psychiatry is a profession worthy of the public trust.

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Response to the Presidential Address

BY JACK WEINBERG, M.D.

THE BRITISH PLAYWRIGHT Robert Bolt, in his play *A Man for All Seasons*, has a character called Common Man. After the final and peculiar rites of passage at the Tower of London devised for that Uncommon Man, Thomas More, who chose to be accountable to his conscience rather than to his monarch, this Common Man (who not surprisingly is the executioner) comes forward and ends the play with the following words,

> I'm breathing . . . are you breathing too? It's nice, isn't it? . . . It isn't difficult to keep alive, friends—just don't make trouble, or if you must make trouble, make the sort of trouble that's expected. Well, I don't need to tell you that. Good night. If we should ever bump into one another, recognize me. (1, p. 162)

The message is clear: March unquestioningly to the drum of authority even at the sacrifice of principles and loyalty to one's profession so that common man's axe may be stayed, so that one may be able to breathe, if not to live.

Dr. Gibson has today detailed for us the troubles, expected and unexpected—some of our making, most not—that we must face. In his characteristic cool and unflappable manner, and in keeping with the theme of this annual meeting and its much more civilized rites of passage, he has pointed out to us the forces in control of our destiny and our adaptive responses.

Many of the problems we encounter are not so much in the hands of our Association, its leadership and its staff, as in the hands of outside forces and bureaucracies whose machinery we are unable to budge. Also involved are those in our sister disciplines who attempt to vitiate our efforts to maintain our integrity as psychiatrists who bring to our therapeutic efforts the uniqueness of a medical education.

Our members expect our Association's leadership to help promote and maintain public trust in our profession and its accountability. Dr. Gibson has demonstrated admirable leadership in that direction in the face of criticism from within and without. Much has been achieved during his competent stewardship. However, it would be unseemly of him to resolve everything and leave no task undone for his successor. Accountability is an ever-continuing process. Accountable to the public and its agencies we are and we shall be, as our efforts in that direction indicate.

As the incoming President of our Association, may I, however, state that I am even more concerned about being accountable to our patients and our profession. I am certain that those are the sentiments of most of you. We find ourselves in the position of another character, in a tale told by Elie Wiesel,

> One of the Just Men came to Sodom, determined to save its inhabitants from sin and punishment. Night and day he walked the streets and markets preaching against greed and theft, falsehood and indifference. In the beginning people listened and smiled ironically. Then they stopped listening; he no longer even amused them. The killers went on killing, the wise kept silent, as if there were no Just Men in their midst.
>
> One day a child, moved by compassion for the unfortunate preacher, approached him with these words: "Poor Stranger, you shout, you expend yourself, body and soul; don't you see that it is hopeless?"
>
> "Yes, I see," answered the Just Man.
>
> "Then why do you go on?"
>
> "I'll tell you why. In the beginning I thought I could change men. Today I know I cannot. If I still shout today, if I still scream, it is to prevent man from ultimately changing me." (2, pp. 94–95)

Why is it, then, that I wish to shout, that I wish to be heard, that I wish to preclude the changing of us? It is simply because I want to warn against the triumph of expediency over purpose, of structure over function. Our purpose is clear and unvarying: it is to alleviate emotional suffering, even though we are fully cognizant that we are confronted by the human condition, with its unsurpassed inventiveness to mobilize pain. There is no substitute for the skillful, holistic diagnosis and the careful and meaningful treatment of the ill.

While I readily see the necessity for examining our structure and modifying it to promote more efficient function, I have been concerned that in pursuing structural change, function may be neglected. Our organizational functions, as I see them, are triple in nature: scientific, social, and guild concerns.

1. The scientific function of our Association is clearly stated in our Constitution. I see ourselves assiduously engaged in the realization of these objectives. Although our efforts seem to be overshadowed by other concerns, these are the primary and continuing priorities.
2. In regard to the social function, I am reminded of

Read at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2–6, 1977.

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the words of the great Rudolf Virchow (1821–1902), who observed that if medicine were ever to fulfill its great ends, it must enter into the larger political and social life of our time; it must indicate the barriers that obstruct the normal completion of the life cycle and remove them (3). No more eloquent statement needs to be made in any period in which medicine and psychiatry (as a branch of it) profess to be concerned with the health of its citizens. Everything that ever happens in the world in some way affects the emotional well-being of our people. Medicine, properly practiced, is not meant only to alleviate suffering; it should also vigorously pursue methods to prevent the occurrence of illness and preclude its recurrence. It is not only as citizens that we should be concerned with such issues as poverty, discrimination, war, and degradation of the human spirit. As conscientious physicians we should see in all of these ecological factors dangerous phenomena affecting the psyche, the soul—creating patients out of a healthy population. Illnesses are processes out of synchronization with oneself and one's environment. Health, after all, is the ability to control and command internal and external events that affect our life and affect behavior.

If we ourselves fail to recognize the validity of all this, certainly the world about us, including our social institutions, tends to ascribe these obligations to us. We are called upon to alleviate all social ills, not because we have been so vociferous regarding our omnipotence but rather because society at large believes that we may have solutions which transcend commonsense.

While we do not have all the answers, we must not avoid the obligation to continually search for solutions and actively participate in efforts to improve the human condition.

3. In regard to guild functions, I for one do not become disturbed when we are accused that APA has such functions. I believe that we should use every legitimate effort to oppose, educate, and convince those who, out of misinformation, mischief, or intent would cripple our discipline by restrictive legislation and regulatory measures. We must define ourselves and our areas of competence.

We are engaged in a struggle with other professionals in the mental health field, some of whom deny the value of a medical education and its uniqueness in the holistic approach to man's suffering. They would deny us experience with the stethoscope—a third ear, if you will, that allows us to hear the inner sounds of the human being. Or the ophthalmoscope, that has provided us with unique insights into the soul of man. Or empathic participation at numerous births, deaths, and sufferings of the flesh. All of these are not merely observational tools and experiential moments but are unconsciously incorporated into our very being; they form the image of the practicing psychiatrist. We must protect them from attack and dilution.

I firmly believe that we detract from our medical image when we adopt an alien vocabulary, a seemingly innocuous vocabulary but one that is inimical to our interests, as, for example, when we substitute "client" and "consumer" for the time-honored term "patient." There is nothing inappropriate in the term "patient," for its root derivation means to suffer, and who else indeed should be entitled to help in the health delivery system than sufferers? "Therapeutic contacts" become substitutes for doctor-patient relationships. The new jargon obviously emphasizes the business aspects of our profession, removes the human equation, leaves us open to the cruel accusation that we have become cold, calculating business people who have abandoned the time-honored aspects of humanism, of physicians who care.

The feeling that we are being reactive rather than proactive to issues and intimidating forces is pervasive in our Association. While we respond to situations and attempt to solve problems, I would like to see a long-range planning body established—a "think tank," if you will. In this way we can permit our best talent to formulate policy in a thoughtful, deliberative manner for years ahead rather than under the gun of an immediate crisis, although of course we will continue to act promptly in the light of past experience and in concert with our goals. I shall bend my efforts in the year ahead to bring such a body into being.

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Robert W. Gibson, M.D.
One Hundred and Fifth President, 1976–1977

BY ROBERT S. GARBER, M.D.

"INDIVIDUALLY AND COLLECTIVELY we must demonstrate through responsible action that psychiatry is a profession worthy of the public trust." These words state in a nutshell the philosophy of the man who has just completed his term as one hundred and fifth President of the American Psychiatric Association. But such a philosophy did not emerge full-blown when he assumed the badge of office in May 1976. Roots of his responsible, public-spirited attitude were manifest early in Robert W. Gibson's life and were even more discernible as he moved into the leadership of one of the most prestigious psychiatric hospitals in the nation and took a major role in community and professional activities, meanwhile becoming a successful husband and father and a friend and colleague of a wide circle of movers and shakers.

Getting to where he is today involved the expected hereditary and environmental factors—and also perhaps a little magic. Robert Gibson's father, Walter Brown Gibson, was a reporter on the *Philadelphia Ledger* and a free-lance writer of books on mystery, magic, and the occult. In 1931 he would turn out a mystery story whose hero was a debonair man-about-town given to solving heinous crimes in his spare time. "The Shadow" became one of the most popular series of magazine stories ever published and the basis of the enormously successful radio show many of us remember from our younger days. An excellent amateur magician, Mr. Gibson was a confidant of Houdini, Raymond, Thurston, and Blackstone, and coached young Bob in the tricks of the trade. Growing up with Lamont Cranston for a sibling and famed prestidigitators for family friends has to have had an impact on the hero of this tale.

Dr. Garber, a former APA President, is President and Chief Executive Officer, the Carrier Clinic Foundation, Belle Mead, N.J. 08502.

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It is a pleasure to report that Walter Gibson has revived and updated Lamont Cranston to tell us what evil lurks in the hearts of men in these perilous seventies. How reassuring to know that both Bob Gibson and Lamont Cranston are on the job now!

Mrs. Walter Gibson, the former Charlotte Wagner, had interrupted her plans to become a professional singer in order to marry and raise a family. A fine swimmer, she gave her son Bob her love of both music and athletics. Bob remembers vividly the encouragement she gave him in his efforts to excel at sports and the many hours she invested in transporting the various teams he joined to competitions. She also urged him on in his studies and was a strong backer of his hopes to be a physician. Still a major force in the Gibson story, she has recently emerged from a bout of bad health and is reported to be once more in fine fettle.

Bob's magical background comes through in the comments of the many people whose lives touched his in those growing-up years. The charisma was evident early on. One cohort cited another radio hero of those days, Jack Armstrong. "Right from the first, he was the all-American boy, making straight As, charming his contemporaries and elders, and excelling in football, wrestling and track." Being a guard, as he was in those days of one-platoon single-wing play, could have helped him acquire the strength he has shown in his career—every other play went over the guard in those days. Recognition came early: he was president of his high school class and of the student council and made the Pennsylvania State wrestling finals. A younger stepbrother recalls, "He was bigger than I was and could beat the tar out of me. But I respected Bob because he took the time to help me to be an athlete good enough to play with him and his friends."

His mother described him as a boy who never wasted any time. "He was a driver and spent his time on his books, reaching out in many lines as if seeking to find out what would interest him most."

The superintendent of his high school remembers clearly an incident when Bob was asked by a teacher why he had elected to take five major subjects instead of the required four. He answered, "Well, it's there and I might as well take advantage of it." That was one of the considerations that led the principal to declare

Bob Gibson one of the ten best students who ever graduated from Lansdowne High School. Bob was ahead of the recent surge of students interested in filmmaking, having become an accomplished filmmaker in the forties. He did the shooting, editing, and splicing of his own action films, the most adventurous of which was shot from the front seat of a toboggan racing down a slide at Lake Placid. His interest in filmmaking may have been cooled somewhat when he tried to shoot an oncoming snowball and got it full in the face.

COLLEGE AND MEDICAL SCHOOL

Bob went to Lafayette, serving as president of the freshman class and becoming Middle Atlantic States light heavyweight wrestling champion. The accelerated Navy V-12 course moved him on to Swarthmore and to service as a corpsman at the Philadelphia Naval Hospital. The young man showed his considerable ingenuity and organizational ability when he promoted a back-rubbing contest among the nurses. He and another corpsman served as judges of the various contestants' skills; the only way they could do that was to themselves become the "rubbees."

Girl-gathering activities stopped soon afterwards, when Bob gathered in a wife, marrying Gwendolyn Locke after his first year of medical school. Only one other of Bob's school classmates was destined to become a physician, and he was Bob's best friend, Gwen's date prior to Bob, and the one who introduced the future Dr. and Mrs. Gibson. After the introduction Bob made his interest in Gwen known to his friend and the competition was on. Bob notes that both men became psychiatrists and that it was Gwen's influence that was behind this. Gwen claims to have wondered for years whether that was a compliment or an insult!

When Bob and Gwen arrived at the University of Pennsylvania Medical School, married students were still exceedingly rare birds. He was the first married man to be president of Alpha Kappa Kappa, and he and Gwen were in great demand as chaperones for fraternity parties; the rules stated only that chaperones must be married couples, not that they had to be old folks. One wonders how inclined the young newlyweds were to break up the couples who retreated to dark corners of the fraternity house.

Bob's skills as magician and craftsman are remembered by his Penn classmates. Card-playing cronies often wondered if legerdemain was a factor in his extraordinary winning streaks, but one of his often beaten friends thinks it was just skill. He recalls that Bob could replay bridge hands days later, remembering each card and the exact order of the play.

Gibson resourcefulness benefited both the fraternity and the Gibsons when Bob and his stepbrother took on the job of remodeling the fraternity house kitchen. Neither of them knew one end of a hammer from the other, but the fraternity was tight on funds and so were the Gibsons. Operating at night so the building inspectors wouldn't get wind of the amateurs at work, the brothers did the job in exchange for free board. Bob and Gwen recall eating free at the house for two and a half years, since no one was quite sure when the debt was finally paid.

ENTRY INTO PSYCHIATRY

Because of his accelerated program Bob graduated three months ahead of the time his internship was to begin and spent that time on the staff of Trenton State Hospital. The writer, then assistant director at Trenton, had made him an offer he couldn't refuse: $250 a month, a furnished apartment, and a job for Gwen. It was a fateful time for the profession and a memorable period for the writer, who watched Bob's growing involvement in the plight of the psychiatric patient.

A former classmate recalls his own years on a biochemistry fellowship when he talked with Bob, by then doing his residency, about their plans for the future. "I remember Bob talking then about how people were basically good, but that their goodness had often been blocked from developing because of events which had made them into hostile or mixed-up people." Colleagues in his later career confirm that he has maintained that basic faith in the goodness of human nature. We can be pleased that this view of humankind rather than The Shadow's sinister opinion prevailed in his thinking.

While he was on duty at the Bethesda Naval Hospital, Bob was recruited for residency at Chestnut Lodge. The recruiter was then tapped himself for the National Institute of Mental Health. "I told myself that I should be pleased that I'd been a benefactor for Dexter Bullard even though I didn't get to work with Bob at Chestnut Lodge. We have collaborated over the years in a variety of other situations. He is a truly outstanding person and I've taken great pleasure in watching his steady growth to this present position of leadership and recognition."

CHESTNUT LODGE

The busy resident somehow managed to find time to write books and lyrics for the annual staff musicals at Chestnut Lodge, and his interest in music continues to this day.

A memorable patient of the Chestnut Lodge years was Miss H.L., a remarkably docile and untroublesome patient except for one unfortunate habit: she was given to stuffing towels into the toilets and flushing them. The results were major plumbing crises that necessitated digging up pipes all over the grounds to remove the blockages. It must have been something of a relief when she was transferred to Sheppard and Enoch Pratt Hospital. Years later, when Bob was directing major reconstruction of the aging Sheppard and Enoch Pratt plant and the grounds looked like a

World War I battlefield, the medical director of Chestnut Lodge paid a visit. Looking over the trenches, craters, and piles of rubble that disfigured the grounds, he commented, "I see you still have Miss H.L. with you."

During his time at Chestnut Lodge, Bob worked on an intensive patient study which was the basis for *Schizophrenia and the Need-Fear Dilemma*, a book of which he was a co-author. The work is a major contribution to our understanding of the psychological aspects of the illness in a time when advances in our knowledge of the biological problems involved tend to be getting most of our attention. Taking the broad view is characteristic of Bob Gibson.

SHEPPARD AND ENOCH PRATT

In 1960 Bob became director of clinical services at Sheppard and Enoch Pratt Hospital. Three years later, as the fourth medical director, he assumed responsibility for the complete administration of this private psychiatric hospital. (Two of his predecessors, Edward N. Brush, M.D., and Ross McC. Chapman, M.D., had served as Presidents of APA.) It was a critical time in the history of the institution, which had served the mentally ill for more than 70 years. To many medical people, the day of the private psychiatric hospital was over. "The Sheppard," once a country hospital eight miles from Baltimore, was being engulfed by Baltimore's spreading suburbs and by the rapidly growing town of Towson. There was a strong feeling that the hospital should sell off most of its 400 acres of now valuable land to housing developers and build a small hospital in one corner of the remaining property. The real estate sale would endow such a small hospital comfortably for years to come. However, the new medical director convinced the trustees to advance rather than retreat. The Gibson dream was of an expanded institution offering the best of inpatient care, research, community service, and the training of mental health professionals.

The original structures, dating from 1853, underwent major remodeling (1). New programs and procedures were initiated, and the hospital was on the march. Land was sold to the Greater Baltimore Medical Center, St. Joseph's General Hospital, and Towson State College. Gibson was responsible for the establishment of a committee that supervised the cooperative efforts of these institutions with Sheppard Pratt, all working on common problems and on programs of mutual support. The hospital provided psychiatric educational programs to the Greater Baltimore Medical Center and received aid from the center in maintaining the Sheppard Pratt crisis clinic. St. Joseph's helped in various community mental health programs and in the provision of prompt medical care. Sheppard Pratt provided psychiatric assistance to the students and faculty of Towson State College and clinical experience to Towson psychology students. A joint Towson-Sheppard training program for mental health workers now leads to a Bachelor of Science degree and to certification as a mental health technician.

The Gibson approach stressed the traditional role of the treatment hospital, providing psychotherapy based on the life experiences of the patients and on their need to integrate themselves into their communities. A team approach was initiated, and the numbers of psychiatric residents, social workers, and occupational therapists increased. Training for psychiatric nurses was carried out in cooperation with nearby community colleges.

Although himself a fully trained psychoanalyst, serving as training and supervising analyst of the Washington Psychoanalytic Institute, Bob has been open to all valid developments in psychiatry. Sheppard Pratt uses a wide variety of treatment modalities in treating both adult and juvenile patients.

The number of patients admitted doubled, as did the number of physicians in training. The burgeoning staff of social workers more than doubled their services to neighboring hospitals, colleges, and the community-at-large. New programs have also involved the hospital with correctional schools, clinics, police training schools, and community mental health centers.

The number of long-term patients steadily decreased, replaced by adolescents and children coming for treatment. Bob led the child guidance program in its expansion to a five-day service for children 5 to 12 years old who were too disturbed to attend school. An inpatient service was initiated for the same age group; the program took over part of a building that had once been filled with long-term adult patients. A high school and a junior high, which share the structure, have been accredited by the Maryland Department of Education.

Another structure, once used to house nursing students from distant areas, became available when the nursing trainees began coming from local colleges and so could commute from their homes. Again walls came down, pipes went in, and wiring was overhauled as another building was reshaped to accommodate more of the director's new programs. For several years it looked as if not Dr. Gibson but Miss H.L. was the busiest person at Sheppard Pratt!

His reorganization of Sheppard Pratt had much to do with his receiving the Edward A. Strecker Award, given by the Institute of the Pennsylvania Hospital each year to "a young psychiatrist who has contributed the most in the field of psychiatry." That also seems an appropriate description of the man whose progressive ideas, organizational ability, and strength of purpose were instrumental in Sheppard Pratt's receiving the APA Hospital and Community Psychiatry Gold Achievement Award in 1972 "for demonstrating that a private hospital can modify its traditional role and become a catalyst in the development of community services."

We come back to speculating about his early contact with magic and the mysterious: those who have worked with Bob Gibson at Sheppard Pratt wonder

out loud how it is that so many of his prophecies about things to come have materialized in exactly the ways he has predicted. The vice-president of the Sheppard and Enoch Pratt board of trustees describes Bob as a person who can keep more balls in the air and drop fewer than anyone he has ever encountered.

COMMUNITY AND PROFESSIONAL ACTIVITIES

Witness his involvements in the community and the profession that he manages to sustain in addition to his work at Sheppard Pratt. He was the first chairman of the APA Commission on Standards of Practice and Third Party Payment, was chairman of the Advisory Council to the Maryland Comprehensive Health Planning Agency, a consultant to the Maryland Medical Assistance Program, treasurer of the Maryland Health Maintenance Committee, and was recently appointed to the newly formed State Health Coordinating Council. He has been a frequent spokesman before committees of the U.S. Senate and House of Representatives, and the only psychiatrist to be made a Fellow of the Association of Mental Health Administrators. Somehow he has also managed to do clinical research; for example, he was principal investigator for an Office of Naval Research Project comparing the family background and early life experiences of manic-depressive and schizophrenic patients. He also was editor of the book marking Sheppard and Enoch Pratt's 75th anniversary (2). Francis J. Braceland, M.D., wrote about this book:

> The diamond jubilee of a famous mental hospital is in itself a noteworthy event. When at the same time it brings together Ives Hendrick, Larry Kubie, Wagner, Brody, Eisenberg, Martin, Sarah Tower, Harold Searles, and a host of brilliant discussants, and allows them free rein to express their observations, it then becomes a not-to-be-forgotten occasion. *Crosscurrents* records that event and recalls its brilliance. (2)

Dr. Gibson served as president of the National Association of Private Psychiatric Hospitals and was a Trustee-at-Large and Secretary of the American Psychiatric Association before being chosen our President. In his introduction of Bob at the 1976 Institute on Hospital and Community Psychiatry, APA Medical Director Melvin Sabshin, M.D., commented: "It is noteworthy in this age of accountability that Dr. Gibson has been elected to national office in the APA by popular vote in five contested elections." As Secretary he was an active member of the Executive Committee. A fellow member of that 1972 committee recalls, "What stands out in my mind is his very even temperament, which seems to be consistent no matter how much confusion there may be around him or even possibly stress within him. He always maintains his objectivity and at the same time that he is serious he can add a light, humorous touch to break the tension. Thinking over the many years that I have known him, the phrase 'he wears well' comes to my mind. . . . I have never seen him act in a petty fashion, nor has he seemed to be motivated by his own needs for power or whatever propels him; instead he has always seemed to be focusing on the issues at hand and never seems to lose sight of them."

The Group for the Advancement of Psychiatry has also benefited from his service—as a committee member, and committee chairman, and now president-elect. As chairman of the Ginsberg Fellowship Committee, he was described by a fellow member as "one who showed us the value of teamwork and the art of having fun while learning. He also managed to provide direction while encouraging individuality."

The president emeritus of Sheppard and Enoch Pratt's board of trustees comments: "From twelve fruitful years of association I can say that Robert W. Gibson is a driver who puts his dreams and plans into reality by careful objective actions, winning hearty support from his associates and ever expanding his own experience and outlook. One by one, many of his dreams and goals have become realities, and the end is not in sight."

Many of his associates cite his ability to delegate responsibility as one of his finer talents; that may explain how he manages to keep so many balls in the air. Moving the imagery from theater to sports, we can say that once the ball has been handed off by Quarterback Gibson, the receiver can count on the play caller's insistence on knowing where the play is at all times and whether the play is advancing, being blocked, or pushed back.

And always there is the acknowledgment of the man's great good humor. In the midst of an endless debate at an APA Board of Trustees meeting, part of the committee took a rest-room break, which Bob welcomed with the comment, "At least in here people seem to know what they're doing."

He seems to have an inexhaustible repertoire of songs, which he is not reluctant to share with amiable assemblages. He also writes poems of the Ogden Nash school. And none who were there will ever forget the costume party of the National Association of Private Psychiatric Hospitals, when the only totally unguessable guest was an African witch doctor in full robe, feathers, magic wand, and animal tail. No one figured out who the masked dancer was until Bob took off the mask and accounted for the authenticity of his garb, which had been a gift to Moses Sheppard from a Liberian chieftain. Shades of The Shadow!

THE PRIVATE SIDE

There is also a private Bob Gibson, husband of Gwen and father of Robert, Jr., Christopher, and Peggy. Determined not to deprive his family of his attention no matter what the professional pressures, Bob has taken on a few more jobs to juggle, such as troop physician for his sons' Boy Scout troop and chairman

of their camping committee. When other fathers came up with elaborate excuses for not accompanying their sons camping, Bob threatened to recruit mothers. He took a ribbing about his attempt to spend weekends in the woods with the women and his supposed disappointment when the other fathers were shamed into showing up. Bob and Gwen have produced three winners in Robert, Jr., a Baltimore probation officer working on his master's degree in psychology; Christopher, on duty with the Navy in Pearl Harbor; and Peggy, a Western Maryland freshman planning to be a physical therapist whose healing talents are now lavished on her thriving plants and Polish rabbits.

Gwen sees herself as one of those women raised to perform well in one sort of role, then moved by changes in the script to learn a whole new part. She has maintained her early commitment to being an effective wife, mother, and homemaker as she has worked out her own professional goals. She has become quite a juggler herself, keeping all the home front balls in the air while tossing in a B.A. from Towson College, a master's degree from Johns Hopkins, a job at the Baltimore City Psychiatric Day Care Center, activities for the Maryland Art Therapy Association, and the tasks of treasurer and program chairman of the American Art Therapy Association. No one who knows the Gibsons is without praise for the way Gwen has played helpmeet in Bob's professional and personal life—mother of a fine family and newborn professional in the changing "outside world."

Gwen's comments about Bob make him seem almost without flaw. Almost. He does have a failing, she reports. He goes into culture shock when they travel. When a Peruvian asked him in Spanish for directions to some point in Lima, he stammered, "I don't speak English." And when a passerby did the same in England, Bob replied in a British accent.

Despite this wife's eye view of Gibson the Traveler, an eminent Past President of this organization describes Bob in this way: "He looks like, acts like, talks like, and *is* the kind of a guy that you would like to have represent psychiatry anywhere." He has done just that as President of the American Psychiatric Association for 1976–1977. Your author stands overwhelmed by the catalogue of admiring adjectives he has accumulated in the interviews for this article: "serious, thoughtful, logical, decisive, articulate, clear-thinking, compassionate, considerate, professional, trust-evoking, strong-willed, flexible, amiable"; and from his mother: "driving and insatiably curious." The writer can only say in summation that it is an honor to have worked with Bob Gibson over the years and to have had him as our President this year. To quote Virgil in his description of a hero of his times, "*Quantum instar ipso est*": None but himself can be his parallel.

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Psychiatric Eclecticism: A Cognitive View

BY JOEL YAGER, M.D.

Pluralism is necessary in psychiatry to compensate for the errors and biases characteristic of the equipment we use to appraise clinical "reality"—our own perceptual-cognitive apparatus. Our attention to clinical situations is skewed: we notice "data" consistent with past assumptions and formulations, and consequently, those views are reinforced by our perceptions. The eclectic posture involves approaching each clinical situation from multiple theoretical perspectives and settling on a perspective that most closely agrees with the patient's needs and wishes without sacrificing the best information available to the psychiatrist. Such eclecticism defines the psychiatrist's role as that of a broad-based scholar who can apply what he knows to the clinical situation. The author discusses the implications for clinical practice and psychiatric education.

"ECLECTICISM" IN psychiatry has become fashionable in some circles and the subject for concern in others. The term itself has assumed different meanings in different contexts (1–3). It has been variously regarded as a psychiatric ideology consisting of more than just psychoanalytic psychiatry, an ideological interest in anything *except* psychoanalytic psychiatry, and an advocacy of an eclecticism of treatment procedures resting upon no identified single conceptual base. No existing theory adequately explains how all of the seemingly disparate practices employed in the service of "eclecticism" in the last sense might relate to one another. Simon (3) has pointed out the need for a useful theory of eclectic practice, "essentially a metatheory that would provide a rational basis for the application of other theories and types of practice." He concluded that general systems theory, which has been suggested as a theoretical basis for eclecticism because of its attention to all levels of organization from the molecular to the molar, has several limitations that render it inadequate to serve as such a theory.

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In this paper I propose that eclecticism can be profitably viewed from a different vantage point. My major thesis is this: eclecticism is made necessary not because of how "reality" is organized (the argument made from general systems theory), but rather because of how *we* organize reality; in other words, eclecticism is made necessary by the properties of our own perceptual-cognitive apparatus.

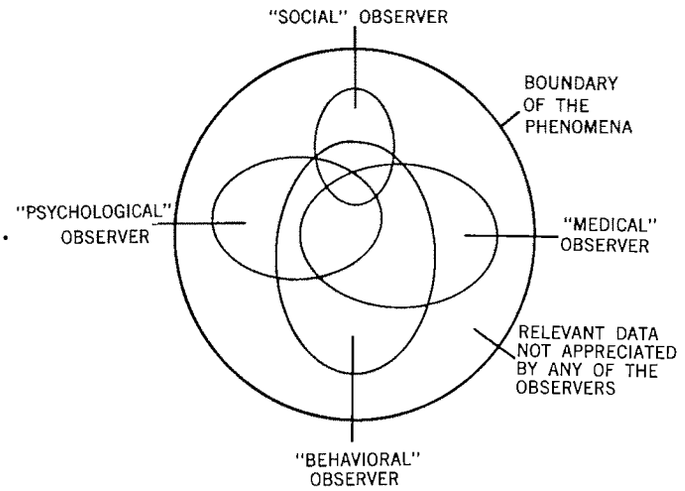
FRAMES OF REFERENCE AND THEORETICAL ORIENTATIONS

Lazare (4) has dramatically depicted the major problem facing the psychiatric clinician; i.e., human behavior is so complex that any one perspective is insufficient for a full appreciation of all there is to see. To illustrate, he presented a hypothetical case report and showed how observers oriented to the medical, psychological, behavioral, and social systems each focused on somewhat different data, appreciated a different reality, and drew different conclusions about etiology and implications for therapy. Yet the different perspectives are not necessarily mutually exclusive; in fact, they may complement each other in important ways. The clinical perspectives interact with the clinical situation as represented in the Venn diagram in figure 1 (taking into account that even the most thorough assessment may fail to recognize or elicit pertinent information).

The theoretical perspective performs two functions for the clinician: it organizes inquiry, observations, and understanding and simultaneously limits the field of vision. Both are necessary. Without a schema for searching, examining, and appreciating data, the clinician would be bewildered by incomprehensible chaos. Yet the same schema does the clinician the disservice of serving as a filter that, while selectively sharpening his focus on some areas, selectively excludes others.

Frank (5) has referred to the "assumptive world," the filters through which experience is processed and our conceptions of "reality" emerge. We psychiatrists, too, process "reality" through our own preconceptions to see things that fit with our usual ways of appreciating human behavior and pathology. Moreover, many of our "basic data" are themselves impossible to separate from context; what may appear as depressed mood in the context of an affective disorder may take the form of blunted affect in the context of schizophrenia.

FIGURE 1
Clinical Data Processed by Four Observers with Different Theoretical Orientations



Before we can know how reality is organized we need to examine the equipment used to measure "reality" and to learn its limitations and biases.

PERCEPTION

Psychiatrists who share common concepts frequently disagree about the presence of key clinical phenomena in specific situations. Seitz (6) found that senior analysts experience great difficulty in their attempts to agree upon the interpretation of the same material, "perhaps because various observers focus on different parts of the total picture." Other studies using analytic data, such as the work by Luborsky and associates (7), confirm the difficulty of reliable observation. Studies of nonpsychoanalytic psychiatric observation offer similar conclusions.

Cross-cultural studies reveal such differences in perception (8). Not only do American and British psychiatrists viewing filmed interviews diagnose patients differently, but they also selectively see different symptoms, usually those consistent with the diagnosis chosen. In general, American psychiatrists report a greater number of symptoms than do their British counterparts.

While clinicians using symptom-rating forms may agree about the interpretation of observable behavior (9), substantial difficulties remain. Even within the same institution, and given the same patient interview, clinicians sometimes disagree about phenomenological evidence of such conditions as delusions, thought disorder, and impairment of object relations (8, 10–13). Kendell (12) found that registrars' opinions about the relationship between "psychotic" and "neurotic" depression biased their ratings of depressed patients in spite of the fact that they used a standardized 41-item scale that required both historical and phenomenological information. For similar reasons, senior analysts' ratings may be even less reliable than those of house staff. Little (14) referred to this common phenomenon as "concept driven perception" and noted that it is not limited to the field of psychiatry. "Conspicuous interobserver disagreements have been demonstrated in the interpretation of x-rays, in endoscopy, in cardiac auscultation and in such an apparently straightforward observation as clubbing of the fingers" (14).

INFORMATION-PROCESSING CHARACTERISTICS OF THE PARTICIPANT-OBSERVER

As diagnosticians we use information that we are scarcely aware of, and we prefer to see and interview patients ourselves rather than trust the impressions and evaluations of others. Psychiatrists frequently form quick diagnostic impressions of patients—in two studies (13, 15), as early as 30 seconds to 2–3 minutes after the beginning of a filmed interview. Again, this phenomenon is not limited to psychiatry: neurological residents' tentative hypotheses are formed almost before the interview begins (16), and the same holds true for experienced internists (17). While many of the diagnoses made within the first 3 minutes of an interview involve "obvious cases," with good agreement between raters, with many others there is a major disagreement between raters.

We seem to operate as "pattern recognizers," checking our perceptions against our cognitive schemata for confirmation or refutation. We construct internal models of reality and shape our perceptions to agree with those models (18). Such internal models may allow us to recognize external events on the basis of a few "key" features and to extract signal from noise. However, the disadvantages of this internal model system—which may more or less accurately describe our perceptual-cognitive "equipment"—are several: no model is entirely accurate, and a system may be misled by its model; further, internal model systems are conservative and may be slow to change even when change might be most adaptive.

We appear to construct cognitive schemata by filling in the dots between scattered bits of evidence, using a sequence of our own design or a design borrowed from our friends and authorities. It is only when we can perceive a reality full of redundancies, where many bits of evidence converge in supporting our concepts, that we may begin to appreciate a relatively illusion-free reality (18).

COGNITIVE FACTORS IN THE CHOICE OF THEORETICAL ORIENTATION

We currently know little about the influences that determine why a given individual will select a specific theoretical orientation. A tendency toward ideological polarity is widespread throughout a variety of in-

tellectual domains, including mathematics, metaphysics, art, politics, and education, to name but a few (19).

Studies of psychiatrists' perceptions and thoughts reveal that factors which influence the perception of data and the diagnoses selected and adhered to include the views of senior, prestigious team members (10), the order in which information is presented to the examiner (13), the age of the psychiatrist (8), and the predominant modes of psychiatric thought to which he has been exposed. Personality differences are also important, and they may partly determine differences in the "style" of arriving at diagnoses (20), as well as the directions in which we each characteristically and systematically distort data (10, 21). Some research (22) suggests that clinical judgments are influenced by attitudinal variables within the therapist, such as tolerance, humaneness, and permissiveness, or, in contrast, disciplinarian, moralistic, and harsh qualities.

While the way in which psychiatrists think about clinical problems, and perhaps their theoretical orientations as well, may to some extent be related to differences in cognitive style, there has been little study of such possible relationships. The concept of "cognitive style" refers to several distinct qualities of thinking, such as modes of information handling (23); the rigidity, differentiation (24), and complexity of thought (25); the tendency to augment versus reduce the impact of incoming stimuli (26); and the degree of field independence versus field dependence (27). Field independent perception is the tendency to experience parts of a field as discrete from the organized background, while field-dependent perception is dominated by the overall organization of the field, with the parts perceived as fused. Field independence has been interpreted as indicating more psychologically differentiated and analytic thinking than field dependence. (Both styles are important, however, and a flexible combination of the two may be most adaptive.)

Two studies (28, 29) have examined the relationship between field independence/dependence and the characteristics of type A and B therapists, as originally described by Whitehorn and Betz (30). Both studies demonstrated a relationship between type B qualities and field independence. (Type B therapists were originally found to have vocational interests similar to those of printers and mathematics or physical science teachers, as measured by the Strong Vocational Inventory, while type A therapists' interests resembled those of lawyers. Type A psychotherapists were thought to be more effective with schizophrenic patients [30].) Although many questions remain about the type A-B therapist differentiation (31), and no conclusions can be drawn from these reports about cognitive styles and theoretical orientations, such research illustrates the potential for this approach in future studies. For example, it would be of interest to know whether field independence/dependence is correlated with a tendency toward atomistic versus holistic ways of viewing human behavior.

POLYFOCAL REALITY AND PSYCHIATRIC THINKING

Reliable and valid observations do exist in the field of psychiatry, but the empirical base upon which we operate is as yet meager. Moreover, in psychiatry it is not the individual "pure facts" that are exciting or useful. These facts, processed through our requirements for methodological purity and taken individually, are sterile, and they hardly fill our practical needs. Rather, it is the meaning that we assign to those facts and the theory that we use to order the independent "accurate" observations—the implications of these facts—that breathes life and generalizable clinical value into their existence. As a consequence, there are currently many ways of seeing reality—of connecting the dots. Each "complete system" actually does make a genuine contribution.

As a profession, how can we deal with this? Faced with a similar problem in comparative epistemology, the philosopher Steven Pepper (32) called for "post-rational eclecticism." In his evaluation of all the major theories of knowledge, he found each of the systems to be approximately of comparable adequacy to explain phenomena. Moreover, none is perfect, and each has its faults and limitations. (The systems that Pepper examined contain, loosely speaking, such theories as materialism, mechanism, pragmatism, platonic idealism, and "objective" idealism.) He concluded that each framework deserves attention in its own right and called for "rational clarity in theory and reasonable eclecticism in practice."

It seems that a similar state of affairs exists in the field of psychiatry and that for us, too, eclecticism is the most reasonable avenue.

THE PSYCHIATRIST'S TASK AND THE ECLECTIC OPERATION

Given that we can understand complex biopsychosocial phenomena from several perspectives simultaneously (at different levels of organization, and by focusing on different aspects of the same level), and given that at our current level of knowledge no one point of view in psychiatry is totally adequate, how can we conceptualize the psychiatrist's task? Broadly speaking, the psychiatrist's task is to effect the best possible solution for any clinical situation that presents itself, the strategy being decided upon only after many possibilities are considered.

Although this task definition seems deceptively simple, it clearly is not. The first step for the clinician in any situation is to decide what the problems *are* and where and how to intervene. How the problems are conceived, who defines them, the very words used in their definition—each of these elements helps shape the intervention. When confronted with a depressed person, if one thinks in terms of "primary affective disorder," "reaction to object loss," or "loss of posi-

tive reinforcers," the therapy chosen may follow from the words used in the original conceptualization. How, then, should one proceed?

The clinician performs what I term the "eclectic mental operation": he attempts to view a situation through each of several frames of reference—grids for the perception of "reality," if you will. These different perspectives are each applied to the specific situation, as if they were a series of different colored lenses and filters flashing one after another before the psychiatrist's eyes. The psychiatrist asks himself, "Concerning the psychodynamics of this situation, what should I be thinking about and asking? What about the biological aspects? What would a family therapist say? How do Szaszian and Laingian issues bear upon this situation? How would therapists of different orientations see the problem; what sorts of interventions would they recommend, and what sorts of results might they expect? What is the evidence for that?" The exercise is similar to what de Bono (33) has called "lateral thinking," i.e., approaching the available information from several points of entry and repatterning the information in an attempt to see alternative possibilities. Lazare (34) has suggested a number of hypotheses to be used by clinicians in initial evaluations.

The patient's perception of reality is another filter the psychiatrist must carefully consider. Patients' comprehension, assumptions, and expectations of their problems and of "appropriate" treatment frequently differ enormously from those of psychiatrists (35).

After viewing the situation from many vantage points, the psychiatrist can then define the problems for intervention and their relative priority. Aware that many truths can coexist, the eclectic psychiatrist will give primary attention to the "truths" that meet the following criteria:

1. Most closely coincide with the patient's definition of the problem, but not at the cost of setting aside the best judgment that the psychiatrist can make based on available information.

2. Most closely suggest a form of therapy that would be emotionally and philosophically consistent with the patient's beliefs and with his expectations of treatment. This is necessary because available evidence (36) suggests that the extent to which patients will return for subsequent visits, comply with medication prescriptions, and be satisfied with their care is related to how meaningful and acceptable the problem formulation and treatment are to them.

3. Based upon what the psychiatrist knows, offer the greatest promise for alleviating the defined problems as quickly as possible, while minimizing the potentially harmful biological, social, financial, and time-consuming side effects.

Within these "truths" there can be no such thing as a "bad" patient (although there can certainly be difficult and frustrating ones). The problem formulation and intervention mode most appropriate to each patient must be thought about and employed. Another implication is that treatment programs set up with too many preconceptions about what *all* patients require may frequently mismatch specific patients' needs.

THE PSYCHIATRIST'S ROLE: A DEFINITION BASED ON THE NEED FOR ECLECTICISM

If we take for granted certain essential qualities of the "good" psychiatrist, such as warmth, concern, intuitive awareness, sensitivity, and caution, the psychiatrist's role emerges as that of a broadly based scholar in the field of psychiatry and behavioral sciences who can apply this knowledge to clinical situations. Several features are implicit in this role definition, as follows.

1. There is a need for *broadly based* scholarship; one's knowledge cannot be restricted to one small theory or technique.

2. The psychiatrist needs to be a *scholar*—intimately familiar with current information in pertinent fields and aware of new developments. If he rejects one point of view in favor of another, the psychiatrist should be able to provide a critical, reasoned explanation, not one based simply on authority or subjective whim.

3. The psychiatrist needs a large repertoire of clinical skills and must be able to use these skills pragmatically.

4. When there is reason to believe that a patient would best be served by modes of therapy with which the psychiatrist is not familiar, he must be able to make appropriate referrals to other psychiatrists and to nonpsychiatric professionals.

While these pronouncements about the psychiatrist's task and role may seem utopian, they are offered as targets toward which professional development in psychiatry can move—directions only, since they can never be end points. Acceptance of this role definition carries with it implications for the objectives of residency programs (37) and for efforts in the continuing education of psychiatrists.

THE ASSESSMENT OF AN ECLECTIC APPROACH

When supporting a particular point of view in psychiatry, one is obligated to consider how that approach would measure up against others and whether it makes any difference to the patient. Bearing in mind the caveat that therapeutic approaches may be effective for reasons not at all related to their theoretical underpinnings, how could we study the relative value of an eclectic approach? Since eclecticism is not a specific treatment modality but a method for selecting the situationally optimal modalities from among all of those available in a given case, such a study would require attention not only to the outcome of treatment, in itself methodologically thorny, but to the process as well. "Doctor's choice" treatment situations might be com-

pared to specific treatments, where the reasoning that pertained to the doctor's choices was made explicit and could be examined for the degree to which eclectic reasoning (as described above) entered into the treatment plan. Experimental designs would, of necessity, be multifactorial, and such studies are difficult to do well.

With respect to the psychotherapies, for a wide variety of patients and problems there is as yet little compelling evidence that any one is incontestably superior to any other. Moreover, therapists with different orientations may have divergent opinions about the benefits of a particular therapy with a given patient (38).

In actual practice, the similarities between the various psychotherapies are considerable and the effect of these similarities may outweigh the differences. In addition, the very fact that a therapist *has* a system of therapeutic beliefs (that the patient may share) may itself be an important element in the effectiveness of the therapy by helping to evoke hope and combat demoralization in the patient (5). This situation creates the possibility of an increasing paradox: it is conceivable that a therapist who strongly believes in a narrow and perhaps scientifically unsupportable therapeutic system may be a *more effective* therapist for some patients than a therapist who is skeptical about that (or any other) method of psychological therapy. The process rather than the content may be the essential ingredient. In any event, for the approximately two-thirds of all patients who will derive some benefit from most types of psychotherapy, eclecticism may not make too much of a practical difference; at the least, any such differences may be especially difficult to demonstrate.

However, at least for some depressed (39) and schizophrenic (40) patients, recent studies have demonstrated the value of simultaneously employing a combination of treatment approaches (drug therapy together with some form of psychotherapy or environmental therapy) for achieving both symptom relief and improved social functioning. Each therapy is based on at least a different level of conceptualization, if not on categorically separate conceptions, about the pathogenesis of the patient's problems. Neither therapy is individually as effective as a combination of the two. Such rational applications of different therapies, each aimed at different aspects of a patient's problem matrix, are neither indiscriminate nor shotgun therapies. It is easy, and idealistic, to conceive of treatment programs that might combine elements of individual, family, and/or group methods, that employ elements of dynamic, behavioral, cognitive, social learning, Gestalt, drug, and/or a host of other therapies, with the treatment elements individually tailored after thoughtful appraisal of each facet of a patient's total situation. However, such eclectic treatment strategies have been criticized as being "shallow" and perhaps even counterproductive when compared with the use of only one form of therapy at a time, applied in depth.

Yet such objections do not fully appreciate the fact that empirical pragmatism—not theory-bound treatment—has been the cornerstone of advances in clinical medicine; useful theory not only grows from the insights it may provide, but it must build upon results. When facts about treatment effectiveness are few or conflicting, theory-based treatments may be clinically limiting, treating the theory and not the patient. A theoretical "shallowness" with an open-minded approach may be preferable to prematurely "deep" understanding.

Moreover, if the decision process outlined above is used to determine appropriate treatment for a patient, when the evidence indicates that one mode of treatment is best and that others used concurrently would add nothing or even detract from the value of the most effective treatment, then the best treatment should most certainly be used in pure form. Unfortunately, however, with regard to the psychotherapies there is reason to suspect that even with orthodox adherents to a theoretical model for treatment, their actual practices vary enough from one another and from the model that pure forms of such treatments may be difficult to identify.

It may be that the benefits of eclecticism can best be demonstrated through a review of the management of "difficult cases," i.e., those not responding to an initial treatment strategy. The psychiatrist's ability to rethink the problem, to "shift conceptual gears" and find new points of approach, should theoretically increase the likelihood of effective outcome. However, if the psychiatrist lacks either the cognitive flexibility to shift perspective or sufficient knowledge about alternative frames of reference or alternative treatment skills, a patient may either continue to be unnecessarily treated by an inappropriate method or may be prematurely consigned to the "treatment failure" category.

THE LIMITS OF ECLECTICISM

At best, an eclectic approach can hope to offer patients the most reasonable treatment approach for their particular problems at a particular time in psychiatry's history, but obviously there are no ready answers for many problems. However, professional education that encourages the learning of multiple frames of reference may temper the effects of the attention-skewing tendencies of our perceptual-cognitive apparatus.

In the last measure, our ability to be eclectic is limited by the skill with which we select from and the extent to which we keep up with and integrate the painfully overwhelming mass of existing and yet to be created biopsychosocial information that provides skeleton and flesh to the various frames of reference. At the same time, we are all to some extent bound by our habitual sets and attitudes, perhaps some more than others by virtue of differences in cognitive style. Since these factors may intimately affect the quality of

care that our patients receive, they deserve careful attention and further study.

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Auditory Evoked Potentials and Selective Attention in Formerly Hyperactive Adolescent Boys

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Although many of the prominent symptoms of minimal brain dysfunction (MBD) subside during pubescence, adolescents who had MBD during childhood may have persistent neuropsychological dysfunctions. The authors studied the auditory average cortical evoked potentials and behavioral responses of 9 formerly hyperactive adolescent boys and 9 matched control subjects who performed a selective attention task. Experimental subjects showed indications of impairment on both electrophysiological and behavioral measures of selective attention. Although the findings are consistent with a neurodevelopmental lag interpretation of MBD, they may also imply a persistent dysfunction related to the frontal association cortex.

THE TERM minimal brain dysfunction (MBD) refers to a constellation of behavioral and physiological aberrations during childhood that include soft neurological signs, hyperactivity, and attentional deficits (1). The diversity of symptoms involved in the MBD diagnosis has led to the view that this disorder may not represent a unified syndrome, but rather may include a number of separate etiological categories. In spite of the heterogeneity of the MBD symptoms and the lack of a unified etiology, the large amount of data presently being gathered on children diagnosed as having MBD provides useful characterizations of the psychological and physiological variables associated with particular symptoms, thus allowing for effective treatment and possible clarification of etiological factors.

There remains, however, a great deal of uncertainty with regard to the prognosis of the child with MBD. The concept of the disorder as a manifestation of neurodevelopmental lag during the childhood years has led to the inference that MBD consists of transitory dysfunctions that will be overcome with continued maturation. This concept is derived largely from findings that the diagnosis of MBD is often based on criteria that are abnormal only in terms of the child's age (2). These criteria include clinical and behavioral signs from neurological and electrophysiological evaluations, as well as psychological measures of attention, cognition, and impulse control. Further support is obtained from the observation that many of the presenting symptoms, particularly hyperactivity and attentional deficits, appear to subside at the beginning of adolescence (3, 4).

An increasing number of studies, however, report persisting neuropsychological dysfunctions in adolescents and young adults who had been diagnosed as having MBD (5–7). In a five-year follow-up study with 6- to 13-year-old boys, Weiss and associates (6) found substantial evidence for attentive and cognitive dysfunctions, restless and impulsive behavior, and poor social adjustment and academic failure. These findings support the view that MBD is a manifestation of a persistent, if not permanent, disorder (8).

A definitive picture of the fate of the MBD child is difficult to obtain. Research into any aspect of the syndrome must contend with the heterogeneity inherent in the classification of MBD. Several investigators have adopted research approaches aimed at reducing this heterogeneity by statistical methods (9) or by classification of subgroups (10, 11). In a systematic investigation by Douglas (10), a group of hyperactive boys was selected from a larger clinical population according to multiple medical, psychological, and behavioral criteria. We followed this approach to select for study a group of boys with hyperactivity and/or attentional deficits as major presenting symptoms.

The aim of the present investigation was to measure a number of neurological, electrophysiological, and behavioral variables in adolescent males who had been diagnosed in childhood as having MBD in order to: 1) assess the level of physiological and psychological functioning in this particular population compared to normal age-matched controls and 2) examine the adequacy of the neurodevelopmental lag concept in accounting for observations made during adolescence.

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The investigation focuses on measures that have proved successful in discriminating between MBD and normal groups in childhood, including evoked potentials to sensory stimuli (12–15), evoked potential and behavioral measures of attention (9, 11, 16, 17), and soft neurological signs (18, 19).

One possible indicator of neuronal processes is the auditory averaged evoked potential. Satterfield and associates (12) reported that a group of MBD children had longer latencies and smaller amplitudes than normal age-matched controls for some of the late components of this response. These results were considered to support the neurodevelopmental lag concept, in view of Schenkenberg's (20) findings that normal subjects exhibited decreasing latencies and increasing amplitudes for these components during the age span of 5 to 15 years. Measurements of auditory evoked potentials, moreover, seem to be appropriate for investigations of MBD since they have been related to attentive functions, which are among the outstanding disorders in MBD children. Of particular use in investigating electrophysiological correlates of attention is the experimental paradigm developed by Hillyard and associates (21) for the measurement in normal adults of evoked potentials to task-relevant auditory stimuli. They found substantially larger amplitudes of a late component of responses to tone pips to which subjects were instructed to attend than to nonattended tones. In the present investigation we used this procedure as well as the presentation of standard auditory clicks in testing adolescent boys who had been diagnosed during childhood as having MBD.

METHOD

Subjects

The experimental group consisted of nine adolescent boys who had been referred several years previously for evaluation and therapy to the Child Development Center of the Nassau County Medical Center. The initial intake procedure involved extensive physical and neurological examination, psychological testing, educational evaluation, and any additional testing needed in individual cases. On the basis of the information thus obtained the clinic staff decided on a diagnosis of MBD.

In addition to the MBD diagnosis the presence of hyperactivity was also used as a criterion for inclusion in the study. A child was considered hyperactive on the basis of reports from teachers or school psychologists, interviews with parents, and the evaluation of the examining physician. Subjects were selected if reports from at least two of these sources indicated hyperactivity and/or attentional deficits. Records of boys selected for the study indicated that they met the following additional criteria when the initial diagnosis was made: 1) presence of soft neurological signs, 2) no major neurological disorders, 3) no sensory deficits, 4) IQ of 80 or above, 5) attending school, 6) academic deficiencies, and 7) subsequent treatment with amphetamine or methylphenidate. Quantitative data on response to drug therapy were not available but the clinical records indicated beneficial effects for the majority of boys. At the time of the experiment the boys had been off psychoactive medication for from 2 months to 4 years.

The median age of the group was 14.04 years, with a range of 12.9 to 16.13 years. Nine boys who attended regular school at appropriate grade levels and had no history of minimal brain dysfunction or learning disability served as a control group; their median age was 14.07 years.

After each subject was selected his parents were invited by letter to participate in the project. The nature of the experimental procedure and the possible benefits of the research were explained to them in conversation and in a written consent form.

Procedure

The subject was seated in a comfortable chair in an electrically shielded and sound-attenuated chamber. Hearing thresholds for each ear at the relevant experimental frequencies were obtained.

The recording session consisted of presentations of binaural sequences of clicks or tone pips through stereo earphones. A series of clicks (0.1 milliseconds duration, 63 db intensity) was delivered at a rate of 1 every 2 seconds for 2 minutes. The tone pips were used in a selective attention task similar to that described by Hillyard and associates (21). Independent random sequences of 800 Hz pips to the right ear and 1500 Hz pips to the left ear were presented concurrently; each pip was 68 db above threshold and 50 milliseconds in duration, with interstimulus intervals randomized between 450 and 1500 milliseconds. Each series consisted of 185 pips and lasted 3.25 minutes.

Approximately 10% of the pips to each ear were of a higher frequency than the standard pips—840 Hz (right ear) and 1560 Hz (left ear). The "signal pips" were randomly presented every 2 to 20 stimuli.

The pip sequences were recorded on audiotape and played to subjects 6 times during the experimental session. During the first 4 sequences the subject was instructed to attend either to the left or the right channel, count the number of signal pips, and report it to the experimenter at the end of the sequence; during the last 2 pip presentations the subjects were instructed to press a hand-held button each time a signal pip occurred on the attend channel. To reduce carry-over effects the transition between attending to each ear was separated by a presentation of the clicks, for which subjects were instructed to ignore both channels. Two counterbalanced sequences of attending to the left or right ear were given, with four or five boys in each group receiving each sequence. A 2-minute rest period was given between each auditory sequence.

The recording session was preceded by an instructional period in which subjects were informed of the

task and played a sample section of tape until the experimenter was assured that the subject comprehended the task and could make the required discrimination. Subjects were informed that if they responded with the correct number of signal pips they would be rewarded with fifty cents per sequence. Instructions were also given for reducing bodily and ocular movements. All subjects were cooperative and seemed to comprehend the instructions.

Electrocortical Recording

EEG electrodes (Beckman) were attached to the vertex (C_Z) of the scalp, the right mastoid (reference), and the forehead (ground). Nonpolarizable silver-silver chloride electrodes were placed above and within the outer canthus of the left eye. All electrode resistances were below 8K ohms. Recordings of vertex EEGs and eye movements were obtained via a polygraph (Beckman, Model-R) (bandpass filters .053 Hz and 32 Hz) and stored on analog tape.

Off-line averages of vertex evoked potentials (AEP) to the clicks and tone pips were obtained with a PDP-12 computer using a five-channel averaging program. Separate AEPs were obtained for each series of 360 tone pips to each ear under the attend-left and attend-right conditions and for 171 click presentations. Averages were for tone and click stimuli for 400-millisecond and 500-millisecond epochs following stimulus onset, respectively. Possible contamination of the AEP data by eye movement and eye blink artifacts was assessed via an editing procedure by which trials during which such movements were indicated by EOG recordings were deleted from computation of the AEP. Since the edited AEPs for the three subjects with the greatest number of such artifacts did not differ appreciably from the unedited AEPs, unedited averages were used for subsequent evaluations.

Behavioral Measures

The last 2 tone pip sequences, to which subjects responded by pressing a hand-held button, yielded behavioral measures of selective attention. Button-press data and response time latencies were recorded by the experimenter. A response was scored as correct if it occurred within 1.0 second after pip presentation.

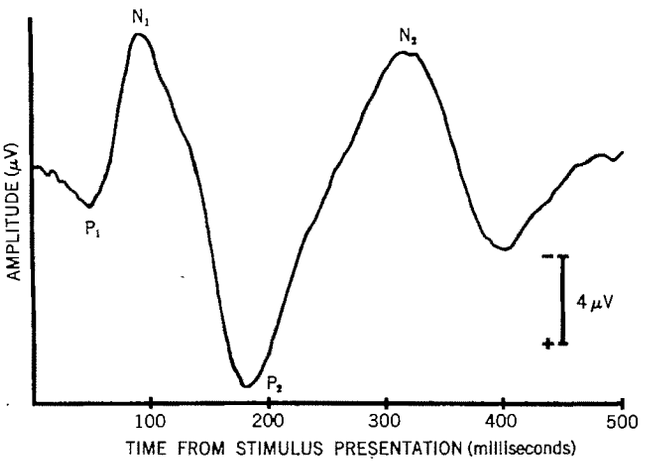
After the experimental session each subject was given a neurological examination to assess soft neurological signs. The examiner was not informed whether the individual was an experimental or control subject. Each subject's parents were also asked to complete a 73-item symptom questionnaire (22).

RESULTS

Evoked Potentials to Clicks

The auditory AEPs had waveshapes similar to those reported by other investigators (see figure 1). To facilitate comparison with results of relevant previous studies (12, 13) the latencies and the peak-to-peak amplitudes of the late components, designated N_1, P_2, and N_2, were analyzed. Values for these components are indicated in table 1. There were no significant differences on any of the measures between the experimental and control groups (two-tailed Mann-Whitney U test, $p>.10$).

FIGURE 1
Representative Average Evoked Potential to 171 Click Presentations*



*The waveform is typical of averages observed for both experimental and control subjects.

TABLE 1
Mean Latencies and Amplitudes of Averaged Evoked Potential Components for Experimental Group (N=9) and Control Group (N=9)

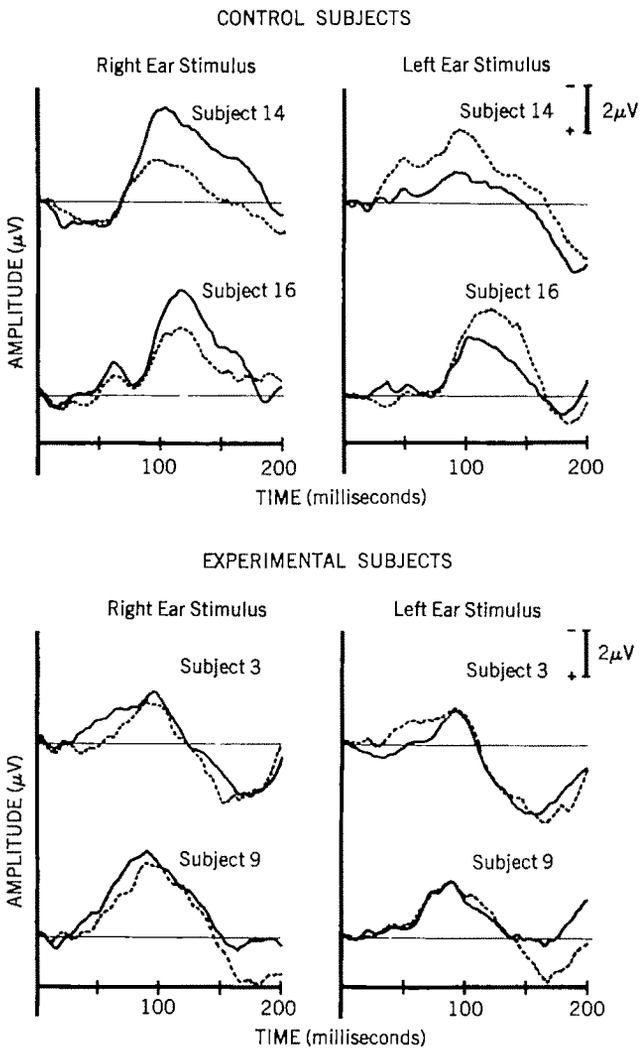
Component	Control Group	Experimental Group
Latency (milliseconds)		
N_1	88.5	86.1
P_2	170.3	176.8
N_2	318.6	315.9
Amplitude (μV)		
N_1–P_2	14.7	14.8
P_2–N_2	12.4	13.4

Evoked Potential to Tone Pips

Figure 2 presents representative AEPs to the tone pips for the attend and nonattend conditions from control and experimental subjects. The left and right columns are tracings from the left and right ear stimuli, respectively. The amplitudes of the N_1 peaks of the AEPs for control subjects during the attend condition were substantially greater than the N_1 peaks associated with the nonattend condition. This finding was consistent for tone pips to both ears. By contrast, the N_1 peaks of experimental subjects were not affected by the experimental conditions of attention.

The amplitudes of the N_1 and P_2 peaks of the AEP were measured baseline to peak, where baseline was considered to be the mean voltage over the initial 10 milliseconds of the AEP for each subject. Prior to statistical analysis the amplitude measurements for each

FIGURE 2
Representative Averaged Evoked Potentials Recorded from Vertex Electrodes in Two Experimental and Two Control Subjects*



*Averages are for 360 tone pips each to the right and left ears under conditions of attending to the right (solid lines) or left (dotted lines) signal tones.

subject for the attend and nonattend conditions were converted to a percentage of the attend condition to remove intersubject variability. In control subjects the mean of the N_1 amplitudes of the evoked potentials for the left ear stimuli was 26.0% smaller during the attend-right compared to the attend-left condition. For the right ear stimuli the mean of the N_1 component was 30.2% smaller during attend-left compared to the attend-right condition. Both of these differences were statistically significant by the Wilcoxen signed rank test ($p<.05$). For the experimental group, however, the corresponding differences in N_1 amplitudes were only 1.0% for the right ear and 12.8% for the left ear. Neither of these differences reached statistical significance (Wilcoxen test, $p>.05$). No significant differences in P_2 amplitudes between attend and nonattend conditions were found for either the experimental or control group. Thus the control group displayed a statistically significant electrophysiological sign (N_1 peak amplitude) of selective attention in both right and left ears; this sign was not observed in the experimental (MBD) group.

There were, however, considerable individual differences in N_1 amplitudes within each group. The average amplitude difference between the attend and nonattend conditions to the left and right ears ranged from 70% to −4% for control subjects and from 32% to −27% for experimental subjects. (Negative number indicates that nonattend amplitude was larger than attend amplitude.) The range for the experimental group reflected the fact that two of the nine experimental subjects responded with substantially higher N_1 amplitudes to the signal stimuli in the attend condition.

Behavioral Measures of Attention

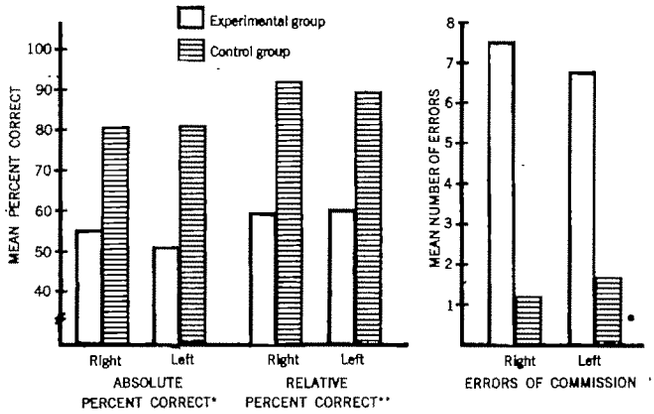
The subjects' reports of the number of signal pips for each of the 4 presentations of the tone pip sequence were converted to scores of the difference between the number of pips that were presented and those that were reported. Error scores were substantially and significantly greater (Mann-Whitney U test, $p<.001$) for the experimental than for the control group. These total error scores, however, provided no information about errors of omission or commission in subjects' responses to individual stimuli.

More informative data with regard to subjects' attentive functions were obtained from the procedure of pressing on the button in response to the signal pip. The results for this procedure, presented in figure 3, indicated significantly lower correct response scores in the experimental than the control group (Mann-Whitney U test, $p<.01$). The deficiency of the experimental subjects was even more pronounced in terms of commission errors, i.e., button presses during periods when signal pips were not presented. The median scores for the two groups were 4 and 15, respectively, with ranges of 0 to 5 for the control and 3 to 21 for the experimental subjects. The two experimental subjects whose scores fell within the range of the control group were the same subjects who obtained substantially higher N_1 amplitudes under the attend than the nonattend condition. The button response scores were correlated significantly with the percentage changes in the N_1 component amplitudes, with rank-order coefficients of .41 for absolute correct, .43 for relative correct, and −.45 for commission error scores ($N=18$, $\alpha=.05$).

Neurological Examination

The findings from a 54-item neurological examination were evaluated in 10 categories found useful for the assessment of soft neurological signs (18). These included measures for dominance, right-left coordination, fine motor coordination, overflow movements, tactile discriminations, and global clumsiness. The total incidence of such signs was only 8 for the experimental and 5 for the control subjects; no significant group differences were found (Mann-Whitney U test,

FIGURE 3
Performance Scores of Experimental (N=9) and Control (N=9) Groups on Push-Button Response to Signal Pips to the Right and Left Ears



*Absolute percent correct=ratio of number of correct responses to number of signal stimuli.
**Relative percent correct=ratio of correct responses to sum of correct and incorrect responses.

$p>.10$). The 8 signs among the experimental subjects was a significant decrease from the 21 soft neurological signs that had been indicated in their childhood records. A decrease in soft signs was found for every experimental subject.

The data obtained from the 73-item symptom questionnaire completed by subjects' parents were grouped into 24 categories for analysis. Results indicated a low incidence of symptoms for all subjects; experimental subjects showed a significantly higher incidence than control subjects on only 1 item, restlessness (Mann-Whitney U test, $p<.05$).

DISCUSSION

The data offer a number of insights into the adolescent fate of the child with MBD. The boys studied present behavioral and neurological pictures free of some of the traditional symptoms present in MBD children. Furthermore, the measures of the evoked response to auditory clicks appear in contrast to the reports of differences in amplitudes and latencies observed in young MBD children (8 years median age) compared to age-matched controls (12, 13). Our findings are in general agreement with the maturational lag view of the MBD syndrome and are consistent with the improvement in symptoms that can be inferred from the clinical records of the experimental subjects.

However, the electrophysiological and behavioral measures on the selective attention task point to important continuing deficits in the experimental subjects. The substantially higher error scores by these boys are in agreement with the persistence of attentional deficits that has been found in follow-up studies of hyperactive children during early adolescence (6, 11).

The electrophysiological measures for selective attention, when interpreted in light of the findings by Picton, Hillyard, and associates (23, 24), offer an insight into the possible neuronal system involved in the deficiency. In a detailed study of averaged auditory evoked potentials recorded from multiple scalp locations, these investigators found that the maximum amplitudes for the late components (P_1, N_1, P_2, N_2) were observed over the frontal and central regions and inferred that this represented widespread activation of frontal cortical neurons. In assessing the effects of attentive functions on AEP components, Picton and Hillyard (23) found no significant changes in components earlier than N_1 and concluded that processes of human auditory attention are mediated by a central mechanism involving changes in responsiveness in the frontal association cortex.

The findings by these investigators are in agreement with the results from our control subjects of increased N_1 amplitudes concomitant with tasks requiring more difficult selective attention. Our finding of the lack of this relationship in experimental subjects may imply persistent dysfunctions related to the frontal association cortex, thus providing additional support of the concept that frontal lobe disorders underlie the MBD syndrome (8). That the frontal lobes play a role in attentive functions has also been indicated by behavioral and electrophysiological findings obtained with brain-damaged animals and humans (25).

The interpretation of the present findings with regard to the concept of neurodevelopmental lag is complex, and several alternative hypotheses must be considered. The possibility that the persisting deficits in selective attention reflect permanent dysfunctions in the neuronal systems that mediate this higher-order function cannot be dismissed. This interpretation would imply that, although some of the dysfunctions subside during the maturational process, others, such as attentional disorders, persist throughout the lives of many individuals who were diagnosed as having MBD in childhood. This possible outcome has been obscured in previous clinical investigations because the diagnostic procedures used failed to push patients to the limits of higher-order functioning.

The present findings, however, may be explained in consonance with the neurodevelopmental lag hypothesis. Evidence has been reported in recent years indicating continuing development of cortical processes during the adolescent years. Thus normal developmental changes in measures of evoked potentials continue to age 17 years (20), and the amplitudes of the cortical contingent negative variation, a measure related to expectation of a meaningful stimulus, have been found to increase systematically between 6 and 15 years (26, 27). Since most of our subjects were in their early adolescent years, we cannot disregard the possibility that the behavioral and neurophysiological dysfunctions associated with the difficult selective attention task will be overcome with continuing maturation. This optimistic prognosis is supported by our

finding that two experimental boys, one of whom was the oldest in the group, were indistinguishable from the control population on every measure in the experiment, including those of selective attention. In view of their profound dysfunction in childhood and their unremarkable adolescence, one is compelled to consider maturational lag as an important underlying process in MBD.

These two interpretations of the findings are not necessarily mutually exclusive, nor are they the only ones with validity. In consideration of the unknown etiology of MBD and the considerable heterogeneity of patients assigned this diagnosis (even in our study group, which was carefully selected according to multiple criteria), it is entirely possible that some children will overcome their disorders during adolescence, whereas others may remain permanently dysfunctional on higher order tasks. It is also possible that some children will retain behavioral handicaps developed in response to negative reactions from persons with whom they interact to the profound aberrations in their developmental history. Such secondary reactions may persist throughout the person's life, marring his or her abilities.

Although inferences with respect to the heterogeneous population of children diagnosed as MBD are limited by our small sample, insight may be gained into the relatively homogeneous subgroup in this study. This subgroup was chosen according to criteria similar to those used in other investigations (10, 12). Our findings support the increasingly accepted view (e.g., 28) that many children diagnosed as having MBD in childhood are not out of the woods with the cessation of most of their symptoms in early adolescence. Continued research is needed on homogeneous subgroups of the MBD population in order to delineate the behavioral and physiological characteristics of these individuals in their adolescent period.

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Borderline Rage and the Therapist's Reponse

BY THEODORE NADELSON, M.D.

Borderline patients often engender feelings ranging from apathy to rage in therapists. The author views the borderline patient's rage as defensive against fear of total negation resulting from projective identification with hostile part objects. The therapist's withdrawal or anger parallels the patient's projection of affect. Countertransference offers the potential for understanding a patient's terror, but therapists may become fixed in identifications that distort their understanding. Clarity often comes slowly as the therapist directs attention to his/her own affective state in order to understand the patient's vulnerability.

THIS PAPER will focus on the therapist's response to the patient's aggression as the crucial issue in the long-term treatment of borderline conditions. With all borderline patients the therapist is always the object of projected bad or hostile introjects, which influence the relationship toward victim-victimizer reciprocity. The patient's anger suffuses the therapy, and the therapist must exercise considerable (and taxing) vigilance toward his personal reactions to this aggression. This vigilance has the potential to increase understanding of the patient's psychodynamics.

THE PATIENT'S AGGRESSION

Although there may be other more primitive or constitutional aspects of aggression in borderline character structure (1), I believe that an emphasis on the defensive function of the patient's hostility and on the manner in which the therapist receives, perceives, and reacts to it will provide a practical conceptual framework that may have operational value. My purpose is to offer a therapeutic alternative to apathy, withdrawal, or counterattack, which at times may seem natural, right, or "real."

Structural analysis (2–4) indicates that "highly specific defensive operations" underlie the aggressive behavior rather than the extreme aggressiveness, poor impulse control, or weak ego proposed by nonspecific approaches. It is often important to seek out the defensive aspect of the patient's aggression while reacting to attack; this mental operation helps to delay the therapist's response and may bring him closer to the essential issue of the patient's narcissistic vulnerability. Simply stated, the patient attacks in the interests of self-defense.

NARCISSISTIC VULNERABILITY

Early frustration by parental figures is probably an important developmental factor in borderline conditions. Patients feel they have been teased or toyed with with no regard for their emotions. Pent-up pregenital aggression tends to be projected onto parents, and early parental images are thus distorted in a paranoid way (1). In early parent-child interactions, parents may project their own needs onto their children excessively, never allowing them their individuality. Such feelings are also projected onto the therapist, who is seen as using the patient for his/her own ends. Often the therapist's response may support this conviction. As therapy proceeds, the projective distortion is corrected and the patient integrates or incorporates the therapist's concern for growth; this often leads to a focus on the individuation of the patient's own children.

My view of borderline and narcissistic patients emphasizes the narcissistic vulnerability that has arisen from their inability to integrate important early introjects, both bad and good (1). These patients have never arrived at a sense of self; they feel that they have "no right to a life" (5). The patient suffers from a continuing conviction that he/she is unwanted and unloved, unredeemable, damned, and guilty. This unsupportable feeling state must be dealt with continually, largely by splitting and projection of aggressive part objects. The threat that is posed to the borderline character derives from the negation imposed by aggressive introjects and from the defensive compromise itself, i.e., the splitting of the good from the hostile part objects. The splitting defense and the subsidiary defense of projection, however, prevent the borderline character from feeling whole; this also leads to a chronic sense of alienation in the interpersonal sphere. Such individuals are convinced that they can be eliminated or annihilated; one patient reflected that this would be done "casually or negligently." They feel they can be dispensed with like a used tissue.

The aggression that suffuses such patients' relation-

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ships with others is also an energetic expression or assertion of self-preservation against fearful threat. In this context, aggression is defense rather than drive, as Rochlin (6) pointed out. Thus I am speaking of two conceptually separable components of this aggression. One involves splitting and subsidiary mechanisms and defense by projection of the bad internalized part objects onto others. The other component is the related assertion of self against a conviction of worthlessness and eroded self-esteem, continually presented to the ego by the hostile introjects. This second component of aggression, the one I am emphasizing, is an ego response to the constant pressure of threatened annihilation. The patient's continued struggle with ego diffusion (1) and shifting feeling states results from the presence of unassimilable part objects.[1]

Such patients often state that when they feel angry, they experience less vulnerability and more of a sense of being "alive," although such feelings are transient. Projection is defensive against an internal sense of negation; by projecting their lack of self-regard onto others, these patients achieve a temporary surcease from the constant claim made by their hostile introjects. Projected anger is a necessary accompaniment to their disowning their own self-hate and the threat the world imposes on them.

COUNTERTRANSFERENCE

It is as if all patients with borderline conditions engender a countertransference anger that represents the emergence of the aggressive part of the patient from within the therapist (4, 7–9). These are usual, natural responses of the reasonably well-integrated therapist to attack, control, and nullification (10). Also, the therapist's wish to help is frustrated and there is anger at not understanding and not being understood; these are specifically the therapist's difficulties. All therapists have a potential susceptibility to such responses to borderline patients, although some may be more repetitively susceptible (1, 11–15).

The pivotal issue in treatment (given that the therapist's needs are more or less stable) is the understanding of aggression as it emerges between the two principals in the therapeutic interaction. The therapist must be willing to realize the presence of intense and angry feelings that are often masked by a sense of unrest, fatigue, or disinterest in the patient. He/she may often wish to let the issue go, to get through the hour, because of fatigue or the thought that the patient is too refractory on that particular day. Such reactions are signals that the therapist is feeling worthless and helpless, i.e., is resonating with the patient's feeling state. It is often necessary at such times for the therapist to examine his/her own feelings silently and to see how they may reflect the affective state of the patient. Countertransference is best recognized and used rather than disdained or ignored. A "totalistic approach," as suggested by Kernberg (1, 9), employs countertransference as a means of extending understanding and yields important information about nonverbal cues, particularly with borderline patients, when the therapist is aware of the potential use of his/her emotional responses as an indicator of the patient's feeling.

IDENTIFICATION AND COUNTERIDENTIFICATION

The patient projects worthlessness, greed, or hostility onto the therapist (4). This projection is a subsidiary form of "splitting," a process by which the ego is protected from conflict by separation of libidinal (good) and aggressive (bad) impulses. The need for projection, for such a low level of defense, reflects the patient's continuous and intense internal struggle against annihilation. Further, there is a related and conceptually separable lower level of defense, a more primitive form of projection—projective identification (16). This particular form of projection is common in borderline characters, who identify and "empathize fearfully" with the persons on whom they have projected their own aggressive internalized objects from the past (17). This is not absolutely different from forms of transference experienced with other kinds of patients, but the intensity and unshakability of the position often resembles the transference seen in psychotic patients (3). The therapist is experienced as an attacking, hostile person, and the patient identifies with the hostility by attempting a sadomasochistic struggle to control the projected impulses that cannot be controlled from within. The therapist often responds with apathy or loss of empathy, ranging from the wish to be distant to quiet thoughts of murder (7). When the therapist responds to the attack with rage, a seesaw battle often ensues.

The therapist's usual attitude during therapy makes him/her vulnerable to narcissistic injury. Attentive focus on the patient and the necessary identification, or "transient trial identifications" (18), often cloud objectivity and apperception of the therapist's own feeling states. Further, the goal of helping the patient makes one more susceptible to the patient's aggression and simultaneously more blind to one's own normal angry response. Counteridentification, a more permanent identification with the patient, occurs as a result of regression from such transient trial identifications. I view counteridentification as a mode of coping with the therapist's anger in response to the patient's assaults. It is a denial of the normal or human response to attack. The therapist needs to accept the inevitability of at least occasional regressions into counter-

[1]Fault is often found with the use of terms that seem to be derived from descriptions of digestive or physiological function. The justification for their use is that they are metaphors and are consonant with the patient's apperception. Borderline characters frequently are intensely self-preoccupied and see themselves as bad, or, defensively, as good only in contrast to others on whom they project their bad parts. As a phenomenologic experience the good and bad do not coalesce; they never perceive themselves as a unity with virtues as well as faults.

identification and understand that awareness of feelings can become an important therapeutic tool. Such awareness and vigilance regarding feelings of anger and apathy aid in maintaining empathy and in recognizing the transient nature of identification; the alternative may be fixity and stalemate.

THE "SECOND EFFORT"

The therapist must feel good about him/herself and separated from the patient in order to continue a helpful dialogue (19), while realizing that he/she is subject to emotional forces that are sometimes too difficult for immediate mastery; that he/she wishes to help as best he/she can, that the patient is defending against a sense of vulnerability rather than being an "aggressive, hostile person." The second effort is formed when one becomes more fully aware of the counteridentification and tendency to continue the reciprocal victim-victimizer interactions. Some therapists deny their anger (7); the second effort taxes the therapist's vigilance and preparedness to see him/herself as subject to some of the same forces that burden the patient. The therapist's "strain" (15), then, is to use those feelings in the interest of the reparative impulse rather than yielding to the tendency to narcissistic retreat (i.e., isolation, loss of empathy, and withdrawal during the therapeutic interaction). There is no way to achieve immunity against such feelings, but it helps to be aware of the necessity of recognizing one's anger in treating borderline patients.

In the course of a therapy hour, counteridentification often replaces empathic response to a patient without the therapist's awareness. When borderline patients accuse me of ineffectiveness, banality, or lack of understanding, I often experience discomfort accompanied by the conviction that the patient is quite correct. On one occasion, my thoughts ran as follows: "I feel that I have not helped the patient, I am repetitious, perhaps I help no one." There are times when I find myself wishing that I could stop treating such patients; I wish they would go away or die. On other occasions, I have thought that I was probably about to catch the flu and would be sick for a few (peaceful) days. It is helpful to subject these feeling states to self-examination. Usually, I discover the patient's sense of negation *within me*.

On one occasion, a patient was in the midst of one of her frequent and lacerating criticisms of my ability. I had "not helped her at all." She had on many previous occasions suggested that we terminate treatment or cut down on treatment time. I quietly suggested that we could discuss the possibility of fewer visits. She cried out in terror, "Don't shout at me—please."

When I suggested that we discuss interruption of her treatment, I felt that it might be best for the patient in that she could be relieved of her hostility toward me: I felt that she might perceive termination as freedom to leave a relationship in which she felt trapped and angry. At a preconscious level, I too felt trapped, defeated, and depressed. I was not totally aware of the reason for my discomfort until the patient ended her lacerating criticism and openly expressed the sense of helplessness, fear, and self-negation that she had projected onto me.

It is very important to understand not only the tendency toward introjection of the patient's negative feelings but the repetitive and fixed way in which the therapist may take these feelings on. It was this phenomenon that prevented me from appropriately clarifying the affect of the patient I have described. I reacted to her feeling state within myself. Later, when I saw this cycle and realized it had happened before, I could offer an appropriate clarification directed toward her underlying self-negation. I could only do this after I appreciated that I was placing back within her the self-negation and helplessness that she had thrust onto me. On subsequent occasions I have learned to point out her self-negation, which is reflected in her negation of me. I can be separate without trying to be rid of her.

The elusive but important issue presented by Malin and Grotstein (17) is that identification is always a part of the more primitive process of projective identification. For patients with borderline characters, projective identification represents an important means of achieving object relationships.[2] My initial agreement with my patient's projection (i.e., my indication that I might be of no help) was a form of projective counteridentification, an assimilation of her projections (4).

I wished also unconsciously to rid myself of the patient, to separate and distance, in order to avoid negative and/or sadistic feelings and to be free of the necessary and wearying self-analytic vigilance. At that, the "I-Thou" relationship ended, at least temporarily. Later, when I was free to scrutinize both of our affective oscillations and became aware of my own anger, I was able to help her examine her projection. Dialogue was then reestablished and our relationship had far less reciprocal negation. This episode also made me aware of my own general impulses and sensitivity to the stress such patients impose on me. A new, positive phase of treatment with a stronger sense of alliance then began. Attention was directed toward the patient's feelings of valuelessness and her anticipation of ridicule, which resulted in cycles of rejection of others and a terror of loneliness that forced her into subservient positions.

With other borderline patients, the therapist can be-

[2]Projective identification is not limited to borderline conditions. It can be seen generally as a potent force in human life. Indeed, altruism and love may have their beginnings in a form of projective identification in which good part objects are projected onto or into another or others. Identification in such cases is with a cathected object that reflects one's own goodness, positive feeling, or ego ideal. Relatedness is achieved in this fashion and is accompanied by a feeling of increased worth. Projective identification with hostile part objects (projected onto another person) may also be seen as an attempt to achieve relatedness or engagement. However, in such cases the identification is hostile, recapitulating the sense of worthlessness that has its origins in earliest relationships.

come "stuck" in what appears to be a totally supportive role. The patient does not change and never gives any sign of hopefulness about the future. There may be little overt anger toward the therapist. Yet in retrospect (after much time—longer, usually, than the therapist wants to realize) the cycle of projective identification and introjection of the patient's negative feelings can be seen in discrete interactions. Often this cycle colors the entire therapy. The therapist is both the victim and victimizer, dragged along in a sense of helplessness and frustration in a situation that mimics aspects of the patient's own life. The therapist also punishes the patient by keeping his/her distance and by avoiding the center of hopelessness and vulnerability. Only by appreciating one's own discontent and its importance can one stop colluding with the punitive forces within the patient; then, helpful treatment can begin.

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Collaboration Between Ohio Psychiatrists and the Legislature to Update Commitment Laws

BY VICTOR M. VICTOROFF, M.D.

The author describes how the Ohio Psychiatric Association (OPA) worked with the state legislature to ensure that the revision of Ohio's laws regarding involuntary commitment of the mentally ill reflected what psychiatrists felt to be in the best interests of patients. The OPA was successful in changing the definition of "persons subject to hospitalization by court order," in abolishing suggested 90-day and 180-day limits on the hospitalization of certain kinds of patients, in supporting the participation of psychologists in the admission process after the presence of a medical examiner had been affirmed, and in protecting the confidentiality of private patients' records.

IN THE SPRING of 1975 the Ohio legislature undertook a major revision of the statutes regarding the mentally ill; the revision would accent the need to protect the civil rights of involuntary patients.

Early in March 1975 the leadership of the Ohio Psychiatric Association (OPA) learned that a bill revising the statutes had been assigned to a subcommittee of the Judiciary Committee of the Ohio House of Representatives. Psychiatrists had long been pressing for extended guarantees for patients' rights, but we were concerned that in the enthusiasm to establish protection of civil rights, patients' rights might be sacrificed. Our apprehension was justified. On examining the bill (House Bill 244), we found the following problems:

1. The definition of "persons subject to hospitalization by court order" included only those who were overtly suicidal or homicidal.

2. Allegedly suicidal patients would be discharged after 90 days' hospitalization on the theory that "if their desire to kill themselves continued that long it was either a rational choice and not a product of mental illness, or if a continuing illness, untreatable."

3. Mentally ill persons regarded as nondangerous would be discharged at the end of 180 days "because if no treatment is effective in half a year it is probable it will never be and it is wrong to lock a person up indeterminately . . . if there is no foreseeable benefit and if the person is harmless."

4. Trial by jury would be available on request of the respondent. (Throughout the bill the patient was identified as the respondent.)

5. Psychologists (in tandem with nonpsychiatric physicians) would be authorized to certify regarding mental status in probable cause and full court hearings. There were also a few lines that could be interpreted to allow psychologists to admit, treat, and discharge patients in hospitals.

6. A legal rights service independent of the state's General Assembly, the Department of Mental Health and Mental Retardation, and the Office of the Attorney General would be created; this service would have unprecedented power. It would monitor the fate of all people affected by the statutes. Appointed by the Chief Justice of the Supreme Court of Ohio, the administrator of this legal rights service and his or her staff would be responsible to no one; all they would have to do would be to provide annual reports. All records pertaining to respondents, including those in computerized data banks, were to be available to this legal rights service on demand.

7. The language of the statute could be interpreted to include the availability to the service of charts of patients in private offices. The rights of privacy and privileged communication would thus be suspended for the new advocacy service.

8. The most stringent criterion for legal evidence, "beyond a reasonable doubt," would apply to testimony by witnesses in determining whether a respondent were subject to hospitalization under the statutory definitions. Throughout the bill there was an accent on the least restrictive alternative of care.

RESOURCES OF THE OPA

The resources and experience of the OPA were tested by the proposed bill. We had already employed as executive secretary an attorney with experience in the political process. He has worked on a retainer for us,

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The author would like to thank Ohio Rep. Harry J. Lehman of Shaker Heights for his sage and dispassionate contribution to this paper; Prof. Michael Kindred of Ohio State University, an honorable opponent in these transactions; and, especially, Rep. Edward J. Feighan of Cleveland, who won our respect as he championed our minority cause before his colleagues on the Judiciary Committee of the Ohio House of Representatives.

doing administrative chores, keeping records, and setting up meetings as well as monitoring the legislature.

The OPA, whose membership numbers almost 500, actively pursues relationships with other mental health organizations. We have called into existence the Ohio Alliance for Mental Health when provocative issues have surfaced. This forum brings together representatives from both professional and citizen mental health organizations. The OPA offers the alliance a temporary chairperson to make arrangements for meetings, establish an agenda, keep constructive testimony flowing, and maintain communication flow among participants.

The OPA also encourages its branch associations to establish their own local legislative committees. We invite the chairperson of the Ohio State Medical Association's Mental Health Committee to attend OPA council meetings, and our president has been reciprocally invited to participate in committee meetings of the Ohio State Medical Association (OSMA). The director of the Ohio Department of Mental Health and Mental Retardation is regularly invited to take part in OPA meetings.

About once a year OPA holds a working meeting with members of the legislature; these meetings range from cocktail parties to two-day retreats. Our members are urged to volunteer for a project called "lunch with a legislator"; each member involved in this program is expected to be a liaison agent for a particular legislator and to keep the legislator informed about issues that concern OPA.

REACTION TO HOUSE BILL 244

When House Bill 244 was presented to the judiciary subcommittee OPA mobilized its resources. Our legislative committee reached out for our natural allies, OSMA's Committee on Mental Health, the Ohio Hospital Association, and the Probate Judges Association. We also attempted to organize the private psychiatric hospitals into a separate legislative action group but ran thin on manpower and time and never consummated the project.

At our legislative representative's summons we made ourselves available on short notice to attend hearings and present our views. We met to plan testimony, devise amendments, and arrive at consensus. On at least one occasion psychiatrists from all parts of the state convened to confront what seemed to be a crisis only to be told that the legislative subcommittee could not meet because it did not have a quorum.

Much of the proposed bill was sound. However, there was so much wrong with it that we had to decide under what conditions we would seek to defeat it, what we could compromise on, and what we could bargain away. The irreducible "no-surrender" issues were the following:

1. The definition of "mentally ill, subject to hospitalization" should include people incapable of caring for themselves who, by reason of mental illness, cannot function in society and, because of their illness, may not realize or cooperate in their need for hospitalization.

2. The statutory limits on the duration of hospitalization should be removed.

3. Psychologists should not be given responsibility for the determination of mental illness without reference to a physician; psychologists should not be allowed to practice in hospitals independent of physicians.

4. The concept of an unsupervised legal rights service answerable to no one and with access to any medical record should be repudiated and resisted.

•We were given two weeks to iron out our differences with the psychologists and advised to come back to the subcommittee with accord. After a futile exchange of correspondence with the psychologists we returned to the committee deadlocked.

An important reason for the intransigence of the psychologists was that they had previously managed to add to Senate Bill 185 the term "licensed clinical psychologist and licensed physician" to the word "psychiatrists" in the definition of those qualified to decide on the mental status of persons alleged to be criminally insane. This bill had been passed unanimously in the legislature without our knowledge. The break in vigilance that allowed the statutory establishment of the equation that one psychiatrist is equal to a clinical psychologist plus a physician was to prove costly in the debate on House Bill 244.

The legislators were not impressed with our testimony that medical students receive an average of 400 hours of training in psychiatry and that only a physician can make a diagnosis of "no medical illness." Nor were they impressed with our concern that psychologists may advise the court wrongly with respect to the care of the patient's needs and thus may cause precious time to be lost determining proper disposition of the patient's case and that the bill would substitute fewer than 1,000 psychologists of uncertain competency for 14,000 physicians who have discharged their responsibility for many years.

COLLABORATION

On May 5, 1975, at a crucial time in the proceedings, the chairman of the subcommittee broke his knee playing baseball. As a result we won almost a month of constructive time. Because the process by which legislative committees operate requires that amendments be proffered by a member who understands the issues and can respond to questions by fellow members we sought out a bright, articulate committee member to help us.

The committee member we sought out advised us to prepare amendments in specific accordance with approved custom, something we had not been previously informed was necessary. When the legislator was con-

vinced of the merit of our position, he sponsored our cause in subsequent meetings of the committee.

As a necessary element in the education of this committee member we invited him to meet two patients on the psychiatric ward of a suburban hospital. Both patients had given their written consent to this visit. One of the patients had been admitted to the hospital after she had hurled a bottle through a picture window in order to break a curse put on her house by a jealous neighbor. The other was a manic-depressive patient who had stopped taking lithium and destroyed a considerable amount of her household furniture before the police took her to the hospital. Both patients admitted that they had been hospitalized against their will but that they belonged in the hospital, trusted their doctor, and expected to be well soon. We pointed out that if the proposed bill had been passed in its unamended form neither patient could have been hospitalized.

By the time the bill moved to the Judiciary Committee hearings we had developed rapport with about half of the members and felt we had a fighting chance. With the aid of powerful testimony from Dr. Moritz, Director of the Ohio Department of Mental Health and Mental Retardation, who said that he would ask the governor to veto the bill if the definition section was not changed, and on hearing our protagonist member's description of his experience at the hospital, the Judiciary Committee accepted our amendment to the definition section of the bill.

Our most earnest effort was then directed toward convincing the committee not to authorize the involvement of the proposed legal rights service with voluntary hospitalized patients. We stressed that privileged communication is not a social indulgence or a casual privilege that merely facilitates commerce between the health professional and his or her patient; it is a sine qua non for the therapeutic process.

Members of the legal aid society unexpectedly admitted that they were uncomfortable about the matter of confidentiality and suggested that amendments be made to allow voluntary patients in private hospitals to decline the services of the legal rights service on their own initiative. We insisted that patients entering a hospital should not have to reassert their constitutional right to privacy.

The bill was reported out of the Judiciary Committee and voted on by the House of Representatives on February 17, 1976. Our critical amendment permitting access to patients' records in privately operated mental hospitals only on the request of the patient was recommended at the last moment. The House approved the bill by voice vote.

DISCUSSION

Considering the four substantive issues on which we were unwilling to compromise, what was the outcome? On the expansion of the definition of mental illness, we got the wording changed. We achieved the abolishment of the 90-day limit for hospitalization of suicidal patients and the 180-day limit for chronically ill "harmless" patients. We gained a weighted decision on the issue of psychologists in that psychologists would not be authorized to practice in hospitals independent of medical supervision, but we were forced to compromise the position of nonpsychiatric physicians, who would now be required to call on a psychologist to collaborate in the examination of patients in probable cause and formal court hearings when a psychiatrist was not available.

We won a split decision in regard to the legal rights service, salvaging the concept that care in private hospitals is not only different from but better than care in public hospitals and maintaining the right to privacy for voluntary patients in private hospitals. The criterion of "beyond a reasonable doubt" was changed to "clear and convincing evidence," and a host of lesser amendments changed this bill to the point that we could at least tolerate the new legislation.

The bill was referred to the Judiciary Committee of the Ohio Senate. Social workers and guidance counselors then demanded the same privileges that the clinical psychologists had obtained. Civil libertarians tried to add an amendment that would require voluntary patients in private hospitals to obtain court approval for surgery, ECT, and other therapies. We debated these changes, and the Senate rejected the modifications. On April 29, 1976, the day before the legislative session ended, the bill was passed by a vote of 32 to 1.

RECOMMENDATIONS

We are convinced by our experience that legislators and the medical profession are now joined in social alliance. Consumer demands, federal money, and judicial decisions require accountability, rules, limits, and standards to be written into the laws that inevitably affect the way physicians practice.

Assuming that an APA district branch accepts the realities of the legislative process, we recommend certain perspectives and actions. First is the appreciation of the importance of time. The psychiatrist must be ready to present his or her case at the convenience of the legislators. There are no accommodations or special courtesies given to medical professionals.

The psychiatric association must be represented at every legislative meeting regarding any relevant bill by the physical presence of knowledgeable members authorized to speak for the association. Delegated legislative representatives should be carefully chosen and meticulously briefed. They must then be given discretion, within stated boundaries, to deal with events as they see fit. The legislature will not wait for the delegates to call for instructions nor will it defer a decision until the organization's executive council meets.

An attorney familiar with medical matters, parliamentary procedure, and the legislative process should be hired on a retainer to watch for legislation affecting

psychiatry, to cultivate friends among the legislators, to be ubiquitous in the corridors of the state capitol, and to advise when to press for passage or to kill various bills. The members of the association should be kept informed about this attorney's work; it will be easier to obtain their acquiescence to the inevitable increase in dues when they realize the costs of representation and the penalties of not having it.

State legislatures vary. District branch officers must determine where the power rests to know how and where to tap into the system. In New York, for example, it rests with the Speaker of the House. In other states, such as Ohio, the power is spread among committee chairmen, who determine agenda, priorities, assignment of bills, and the timing of presentations to the committee.

Certain items of legislation favor working through the executive branch, as we did with the director of the Ohio Department of Mental Health and Mental Retardation and his connection with the governor. The legislator who identifies himself or herself with the psychiatric association should rely on the judgment and views of the association's leadership but will not solicit them. The legislature is a market in which many proposals compete. There is continual competition among issues to establish priorities. Consider that in most states only 1 bill in 10 presented to the legislature in a given session will pass.

Most legislators work in this capacity part-time; they are usually ordinary citizens, particularly in the small states. They reflect a cross-section of the populace. They do not have time to research the issues presented to them; they have to depend on background information, their instincts, and their education.

The political process has to be considered. In Ohio we have taken the attitude that mental health must be nonpartisan; this is a good rule, but it may be necessary at times to depart from it.

Continuing education of key people in the legislature should go on even when the assembly is not in session. Legislators should be invited to meetings of the association as guest speakers.

In dealing with the legislature it is highly desirable to present an "agreed" bill. Legislators want to be free of difficult choices, especially controversial philosophical decisions. It is a relief to them if certain bills and issues loaded with explosive potential slide smoothly through the legislative process because contentious parties have already agreed to the provisions.

Establishing the doctrine of making bridges between the APA district branch and all other mental health organizations within the state and soliciting financial support from our membership, as we have done in Ohio, are adaptational devices for the survival of psychiatry and the practice of medicine in years to come. The process is unending. Immediately after completing work on one bill and one session the leadership must prepare for another.

The cycle can well be identified by a quotation from Edgar Lee Masters' *Spoon River Anthology:*

> Ten thousand times I've done my best,
> And all's to do again.

The Prevention of Tardive Dyskinesia

BY GEORGE E. CRANE, M.D.

The publication of numerous articles on tardive dyskinesia has had little effect on the excessive use of neuroleptic drugs in psychiatric populations. If current prescribing patterns are not drastically changed, larger numbers of patients will be afflicted by conspicuous, sometimes disabling neurological side effects. The risk of permanent neurological disorder can be minimized by monitoring side effects and systematically reducing drug doses in hospitals, outpatient centers, and private practice. However, one should not underestimate the difficulty in implementing such a program in facilities that rely heavily on chemotherapy.

DURING THE last decade numerous articles have been published on tardive dyskinesia. In 1972 and 1973 the package inserts of all neuroleptic drugs were updated to include information on this disorder, and in early 1973 an editorial entitled "Neurological Syndromes Associated with Antipsychotic Drug Use" was published in the *Archives of General Psychiatry* (1) and other reputable medical journals. More and more authors are cautioning physicians to use greater discretion in their administration of neuroleptic drugs to their patients (2–4). Certainly there has been no shortage of clinical information on tardive dyskinesia.

In order to determine whether education would affect the prescribing patterns of physicians, I delivered to the staff of a well-known hospital a lecture in which I emphasized the necessity of reducing the doses of neuroleptic drugs for all patients. When I compared the amount of medication prescribed in 8 representative wards shortly before this talk and 6 months later, I found that the average daily dose had increased from 250 to 252 mg of chlorpromazine or equivalent doses of other neuroleptic drugs. This is a clear indication that the impact of my educational efforts on the prescribing habits of the physician has been nil. The results of this survey cannot be generalized, of course. Yet if an oral communication accompanied by a discussion of a topic of mutual interest is ineffective, I doubt very much that the medical community can be influenced by the more impersonal communications offered by the scientific literature.

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EFFECTS OF CURRENT PRESCRIBING PATTERNS ON PATIENTS

Large doses of phenothiazines (2000 mg of chlorpromazine or 80 mg of trifluoperazine) are more likely to produce or aggravate tardive dyskinesia in patients aged 40–55 than are the conventional doses (5). But in older persons the risk of persistent motor abnormalities rises sharply even when relatively small doses (100 or 200 mg of chlorpromazine a day or equivalent doses of other neuroleptics) are taken for over 6 months (6).

It is well known that tardive dyskinesia is the result of the cumulative effects of neuroleptics. The following study provides additional evidence. In 1970 I surveyed the incidence of drug-induced neurological disorders among 900 chronic schizophrenic inpatients (6). Four and one-half years later 221 patients from the original sample were selected for another project, which gave me an opportunity to assess their neurological status and compare it with my observations from the first survey. (The patients' average daily doses of neuroleptics had not changed significantly.)

Table 1 shows that the total number of patients afflicted with tardive dyskinesia, particularly of a severe

type, increased substantially during this period. Since this was not a controlled study the possibility of artifacts cannot be ruled out. For instance, the greater prevalence of tardive dyskinesia at the time of the second survey may simply reflect a greater sensitivity of the methods used to record the severity of the motor disorder. It does not seem likely, however, that severe dyskinesia was overlooked in 11 patients who were found to be symptom free in 1970 and severely affected 4½ years later (see table 1). Thus the number of neurological disorders can only increase if current practices of antipsychotic drug use are allowed to continue.

The large doses of neuroleptic drugs that are now prescribed by most psychiatrists are not only harmful but also unnecessary. In order to prove this I instituted a program of drug reduction in 4 wards housing approximately 160 patients. They were under my supervision for periods of up to 3 years. At the conclusion of this study it was clear that the original dose levels could be substantially reduced for most patients without affecting the general climate of these wards. This study has been reported in detail elsewhere (7).

The next step was a cooperative effort with 3 staff physicians to see whether these results could be replicated in 4 wards over which I had no direct control. The degree of my participation in this project varied. One of the physicians was urged to reduce medication without discussing the merits of this procedure in individual cases. There was much closer cooperation with the other 2 physicians in that we jointly evaluated each patient's behavior in the process of adjusting dose levels. There was an overall decrease in dose levels, but the best results were obtained in an open ward where my participation in the project was the greatest. In general, regardless of the degree of my cooperation, attempts to lower doses were unsatisfactory in wards with a large number of disturbed patients. Most patients continued to be disturbed despite the high doses prescribed to them. Thus the amount of medication prescribed reflected the severity of behavioral aberrations rather than the effectiveness of the treatment.

In conclusion, neuroleptic drugs are not the answer to the problems created by patients with serious psychiatric disorders. A program of drug reduction will not be completely successful unless there are alternative ways of managing such patients.

PREVENTING TARDIVE DYSKINESIA

A maintenance dose of 400 mg of chlorpromazine a day or equivalent doses of other neuroleptics seems to be safe and probably quite effective for most patients. However, for persons over 55, doses should not exceed 150 mg a day. For geriatric patients, doses should not be higher than 75 mg a day (8).

The monitoring of side effects is another essential part of a preventive program. Physicians and nurses

TABLE 1
Number of Chronic Schizophrenic Patients with Tardive Dyskinesia and Parkinsonism in Two Surveys

Side Effect	Group 1 (N=221) 1970	Group 1 (N=221) 1974	Group 2 in 1970* (N=25)
Tardive dyskinesia			
Severe	7	25	5
Moderate	19	36	2
Minimal	19	30	2
Parkinsonism	26	17	4
Parkinsonism and tardive dyskinesia	3	6	1
None	147	107	11

*Group 2 consists of the group 1 patients who had severe tardive dyskinesia in 1974. (Breakdown is by the ratings of tardive dyskinesia at the time of the first survey in 1970.)

can easily recognize drug-induced neurological disorders after a relatively brief period of training. The main problem is that side effects are not adequately recorded. Hence the physician cannot follow the progress of an emerging neurological disorder, nor is he in a position to initiate corrective measures.

The physician should bear in mind that the patient he sees for the first time may have a long history of drug therapy by other practitioners in private offices, clinics, or hospitals. Therefore, it cannot be emphasized sufficiently that the physician should perform a routine neurological examination before prescribing a neuroleptic drug and that he should also record all findings, both positive and negative. The physician should also try to obtain as much information as possible about previous medication. If the patient is already afflicted with a neurological disorder, the importance of gathering these data is obvious from a medical and legal point of view. But even though new patients are symptom free despite intensive antipsychotic drug therapy, their susceptibility to side effects is much greater than that of patients who have never received such drugs. Hence, a more cautious use of neuroleptics is in order in such cases. The form presented in appendix 1 is suggested for the purpose of recording relevant information at intake. On subsequent neurological examinations proper entries may be made in the patient's record whenever drug prescriptions are renewed.

The recommended procedures will permit prompt detection of neurological impairment and immediate corrective action in order to prevent early dyskinesia from becoming permanent. It is doubtful that these measures will completely eliminate tardive dyskinesia, but they may result in an acceptably low incidence of this disorder in the future.

CONCLUSIONS

It is quite obvious that the publication of articles on tardive dyskinesia and the pleas of a few concerned

investigators to use neuroleptic drugs with greater discretion have had little impact on the prescribing practices of physicians. Efforts to improve the quality of patient care by peer review committees, medical associations, and other organizations have been equally ineffective, which is a clear indication that major medical problems cannot be solved by bureaucratic procedures.

The utility of a program of drug reduction in conjunction with an effective system of monitoring adverse reactions cannot be disputed. Nevertheless, one should not underestimate the obstacles that must be overcome when certain practices are based on faith rather than on medical considerations.

Only a more rational approach to psychopharmacological drug use will avoid further regulatory action by federal, state, and local agencies. In addition, a more responsible attitude on the part of the medical profession will prevent harassment by civil liberties groups and will undoubtedly reduce the risk of exorbitant malpractice insurance premiums and the constant threat of lawsuits by the legal profession.

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APPENDIX 1
Drug History and Neurological Status at Intake

Name_____ Age_____

1. Previous treatment with neuroleptics _____
2. Informants _____
3. History of neurological disorder _____
4. Complaints (subjective, by relatives, or by other observers) _____
5. Postural disorder (e.g., scoliosis, torsion of the neck or trunk) _____
6. Parkinsonism (bradykinesia, rigidity, loss of associated movements) _____
7. Tremor (of tongue, hands, or legs) _____
8. Tardive dyskinesia (type, location, severity) _____
9. Hypotonia _____
10. Other (chorea, dystonia, tics) _____
11. Comments and treatment plan _____

Clinical Research Techniques in Tardive Dyskinesia

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The authors have developed standardized assessment and control techniques designed to provide objective measures of tardive dyskinesia in patients progressing through an experimental treatment program. Standardized videotaping is carried out and blind ratings on the Abnormal Involuntary Movement Scale are made by a team of psychiatrists and neurologists. Other assessments include several measurements of vocal function and a quantitative acceleration profile standardized on normal subjects. Such measures allow quantifiable determination of response to drug treatment and may provide clues to the etiology and definition of the dyskinetic syndrome.

MEDICAL OPINION concerning the precise pharmacological mechanisms involved in the etiology of tardive dyskinesia remains divided, although the implication of altered neurotransmitter substances is rather firmly established. Our own studies and clinical observations tend to confirm the widening belief that what physicians call tardive dyskinesia may be a group of symptomatically similar syndromes, all of which occur as side effects of antipsychotic drugs but which have varying specific etiology, depending on 1) which neuroleptic or combination has been used, 2) the duration of prescription, and/or 3) a poorly defined idiosyncratic predisposition in the individual. Additional information concerning biochemical, neurophysiological, and clinical substrates of the condition is required before we can arrive at a complete explanation of tardive dyskinesia.

A considerable amount of clinical research has been conducted during the last 10 years on the pharmacology and treatment of tardive dyskinesia. Investigators have administered additional neuroleptics, deanol, lithium, amantadine, L-dopa, methylphenidate, physostigmine, and other agents known to affect pertinent neurotransmitter substances; they then attempt to monitor any ensuing clinical remission or increase in dyskinetic symptoms (1–12). The methods of assessing and quantifying pre- and postdrug abnormal movements have included blind and informed ratings of videotapes, direct interviews with patients before and during the experimental trial, a variety of rating scales, and electrophysiological techniques (3, 13–15). The variety of affected areas, the unpatterned nature of the movements, and the tendency of patients to self-consciously limit their dyskinetic movements during ratings present considerable problems in obtaining measurements of symptoms and interfere with accurate assessment of the efficacy of compounds administered in clinical trials for relief of the syndrome.

We have attempted to develop in our laboratory a series of assessment and control techniques for tardive dyskinesia that are intended to provide objective measures of the condition as patients progress through our experimental treatment program. A series of measures rather than a single rating instrument is used as a means of cross-validation within the study. These methods include blind rating of videotapes using the Abnormal Involuntary Movement Scale (AIMS), placebo control, assessment of neuromuscular function as reflected in speech, global assessment by ward staff, and the quantitative acceleration profile, an electronic testing procedure based on the principles of the accelerometer.

VIDEOTAPING

Patients are videotaped in a standard procedure before receiving study compounds, once a week during drug administration, and once a month after discontinuation of the drug. Patients previously diagnosed as suffering from tardive dyskinesia are escorted to a studio within the hospital and seated on a stage. The camera operator follows the order of the AIMS, focusing for one minute each on the patient's face, lips, jaw, tongue, upper and lower extremities, trunk, and global

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appearance. Patients are also asked to walk across the stage several times for assessment of gait. The videotapes are coded by a research assistant and reviewed by a team of psychiatrists and neurologists at the end of the study. Each segment is given blind ratings on the AIMS for degree of dyskinetic symptomatology. Numerical ratings from 0 (no abnormal movement) to 5 (severe abnormal movement) are analyzed by the Wilcoxin matched-pairs signed-rank test. Interrater reliability is assessed using the Kendall coefficient of concordance.

PLACEBO CONTROL

Although it is unlikely that tardive dyskinesia has a significant psychosomatic component, it is well known that patients can consciously limit their abnormal movements for brief periods of time, particularly when they feel they are being scrutinized. This seriously hinders accurate assessment of medication-related improvement. We use placebo for the first 2 weeks to control our trials of active medications in the treatment of dyskinesia. Patients are videotaped and subjected to the rating procedures used with the active drug, and tests conducted on placebo are rated blind.

SPEECH ASSESSMENT

Assessment of vocal function in dyskinetic patients is based on the premise that individuals afflicted with neuromuscular disturbance will be affected in the very subtle and precise areas involved in speech articulation. It is well known that patients with tardive dyskinesia display prominent bucco-facio-lingual hyperkinesis and discoordination, and it has been postulated that these disturbances are reflected in altered articulative abilities among dyskinetics. It has also been widely recognized that patients who have maintained dyskinetic symptoms for a prolonged period of time develop marked hypertrophy of the tongue, which may reduce the critical ratio between orality and nasality in speech by impeding the passage of air through the oral cavity. It may also be hypothesized that the dysarthria associated with dyskinesia may affect oral-pharyngeal closure, with consequent diminution of speech orality and increased nasality components. The speech tests are designed to provide quantified assessment of the vocal impairment associated with tardive dyskinesia and to reflect changes in impairment over time.

The following speech assessment tests are administered at baseline and repeated at fixed intervals during the drug trial: 1) the Templin-Darley Diagnostic Test of Articulation (16), 2) oral structural and muscular examination, 3) standard reading passage, 4) conversational speech sample, 5) intensity range during conversational speech, 6) softest intensity on sustained vowel, and 7) loudest intensity on sustained vowel.

The Templin-Darley test requires the subject to produce 141 phonemes or blends in the context of a single word. Stimuli are presented visually, either by picture or printed word.

The oral structural and muscular examination evaluates two major areas: nonspeaking motor control and motor control for speech. Breathing, laryngeal activity, tongue mobility, facial muscles, muscles of mastication, palatopharyngeal muscles, diadochokinetic rate for sounds, sound combinations, and articulators are assessed.

The standard reading, the 63-word "Zoo Passage," contains all English sounds except nasals and was designed by Dr. Samuel Fletcher in 1972 (17). Conversational speech is elicited by encouraging the subject to discuss his impression of the Houston Veterans Administration Hospital and of the city of Houston compared with his home town.

A group of six speech pathologists listens to four or five samples of the speech of each patient. Each sample consists of 30 seconds of reading and conversational speech and is presented three or four times in combination with each of the other samples of the patient's speech. Listeners are asked to determine whether they hear normal or deviant speech or voice and to characterize any deviations noted. Imprecision of articulation and breathy voice quality have appeared in our early tests to characterize the speech of patients with dyskinesias.

The intensity tests are conducted using the Tonar-II vocal assessment device developed by Fletcher (17). The Tonar system consists of a sound-separation microphone assembly used to isolate the oral speech component from the nasal, a control and display console, and a reinforcement panel assembly. The microphone assembly is a two-chamber acoustically isolated device with a ceramic microphone used as the receptor in each chamber. The signals from each microphone are amplified within the assembly prior to transmission to the control console. The control and display console consists of a dual-channel analyzer system, a nasality ratio computer, and analog and digital displays for presentation of signal and frequency information.

The primary function of the Tonar is to measure nasality. Nasality in the instrumental sense is the ratio of nasal signal to the sum of the nasal and oral signals, expressed as a percentage. The scale of nasality is from 0 to 100%, and the normal range is 5%–20% if the English nasal consonants are eliminated from the test passage.

A hypernasal condition is said to exist if nasality exceeds 20%, and nasality less than 5% is defined as hyponasal or denasal. Sound and pitch level measurements are included in the system. It is postulated that changes in vocal function and coordination will accompany the onset of drug-related neurological disorders and provide early indicants of impending gross neuromuscular dysfunction or reflect improvement in the dyskinetic condition following treatment within the study protocol.

ACCELERATION PROFILE

The quantitative acceleration profile, which is done with triaxial vector accelerometry, provides a graphic display of the motion characteristics of a particular anatomical site and may be used to observe involuntary motor activity during a resting state or to study movement quality while a subject performs a specific range-of-motion exercise. Motion is detected by a miniature triaxial accelerometer attached to the site being studied. For purposes of monitoring drug-induced parkinsonism and dyskinesia, these areas include wrist, ankles, and chin. Electronic circuitry combines the three outputs in a vector fashion to obtain an absolute magnitude value of acceleration and its first derivative with respect to time. A graphic display of acceleration and acceleration change is provided by a strip chart recorder, and these measures are electronically integrated over a predetermined time interval to obtain values for total change of velocity and total change of acceleration. The latter measures are displayed digitally at the end of a specific observation.

This technique has been evaluated in a group of normal subjects, and baseline data now exist with respect to the various measures and derivations and the expected ranges of normal variation. It is hypothesized that characteristic differences will be apparent in the motion profiles of patients developing drug-induced movement disorders and that these findings will be useful in diagnosing emerging symptoms and monitoring treatment-related changes. Each subject is evaluated in the resting condition and during active exercise.

The active protocol involves abduction/adduction of the left and right upper extremities in a range-of-motion task. The subject is seated in a straight-backed chair placed against a backboard. The subject's midline is centered on a floor-to-ceiling reference line and a set of targets (4-inch-diameter foam rubber spheres) is arranged in space to delineate the designed path for the range-of-motion exercise. All target positions are measured with respect to the reference line and the floor to permit the same test conditions to be used on subsequent visits. The lower target is placed so that the subject is able to touch it with his fingers when his arms are relaxed and falling at his sides, and the upper target is situated above the first so that full abduction of the arm at the shoulder is required to touch it. All subjects are paced at 0.3 repetitions per second by alternately illuminating the two targets. Each test sequence lasts 30 seconds and each arm is tested three times, with short intervening rest periods.

The characteristics of each arm are quantified by use of the following five indices:

1. The simple periodic motion equivalency factor (SPMEF), a direct estimate of the smoothness of motion, is a comparison of the observed ratio of total change of acceleration and total change of velocity with the theoretical ratio, which is calculated from the target dimensions and the pacing rate, assuming that the motion is sinusoidal (i.e., perfectly smooth). Values above 1 indicate the presence of frequency components higher than the fundamental frequency (i.e., the pacing rate); the higher the SPMEF value, the less smooth the motion.

2. The theoretical peak equivalent (TPE) compares the observed average peak acceleration value during each motion cycle with a theoretical sinusoidal model equivalent. TPE values above 1 (the lower limit) provide a measure of the excess acceleration the subject uses in moving quickly from one target to another and is thus an index of the "effort" expended.

3. The theoretical integration of peak acceleration ratio (TIPAR) is a comparison of the observed total change of velocity and the theoretical value that would be expected if the value obtained for average peak acceleration were based on sinusoidal activity. Since a value of 1 would indicate the presence of sinusoidal behavior, deviations from 1 provide an index of actual wave shapes. Because values above 1 are associated with wave forms of less area than their sinusoidal equivalents, the TIPAR quantifies the relative "steepness" of peaks in the acceleration-versus-time tracing.

4. Total change of velocity is simply the total area delineated by the tracing of acceleration versus time. As the name implies, it quantifies velocity characteristics over the time period of the range-of-motion task.

5. Total change of acceleration is the integration of the change-of-acceleration-versus-time tracing. A measure of the irregularity of motion is obtained by quantifying the total change of acceleration and comparing it to the total change of velocity.

The passive portion of the examination involves quantification of spontaneous movements detected at 1) the left and right hands, by an accelerometer attached on the dorsal aspect over the knuckles, 2) the left and right legs, by an accelerometer attached on the lateral aspect of the leg just above the ankle, and 3) the chin, by an accelerometer attached in the midline. During the evaluation of the hands, the subject sits with his arms relaxed and his hands resting in his lap. During evaluation of the legs and chin, the subject engages in a simple task in which he is asked to maintain the points of two pencils in close apposition while the hands are resting in the lap. This maneuver is intended to minimize voluntary suppression of motion in the anatomical site under study. During examination of the legs, subjects sit on an examining table with their legs hanging free.

Quantification of the resting characteristics is based on examination of the tracings of acceleration versus time obtained during fixed 2-minute observation periods. Each spontaneous, involuntary muscular contraction that exceeds a preset amplitude above the inherent noise level of the system is recorded. The results are displayed as histograms, showing the number of movements falling into each of several peak amplitude categories. The results thus provide a measure of the total number of involuntary events occurring in a fixed interval of time as well as a measure of the absolute amplitude of each involuntary event.

ETIOLOGY

Questions involving the biochemical etiology of tardive dyskinesia have been approached through postmortem examination of brain tissue (18, 19) and comparison of dyskinetic features to those of other movement disorders such as Huntington's disease and drug-induced parkinsonism. We have examined the etiological mechanisms in tardive dyskinesia in the context of clinical trials of compounds hypothesized to affect the implicated neurotransmitter systems. By monitoring our patients' responses to the various compounds, we can attempt to interpolate the nature of the biochemical abnormality that appears clinically as tardive dyskinesia. We have administered to several groups of patients the cholinergic substances physostigmine (8) and deanol (3), as well as methylphenidate (7), which stimulates central dopamine and adrenergic activity, and cyproheptadine, which is known to block the activity of serotonin in brain. Responses to each of these compounds and to compounds administered by other investigators have been so varied that we believe more than one neurotransmitter system may be affected and that the neurological event presenting the clinical symptoms recognized as tardive dyskinesia may in fact be any one of a series of neurotransmitter irregularities associated with specific offending agents, dosage schedules, duration of prescription, or poorly defined idiosyncratic factors.

Further elucidation of the roles of acetylcholine, dopamine (20), serotonin (21), and GABA (22) is required for clearer definition of the dyskinetic syndrome, as are epidemiological data comparing the complete medication histories of dyskinetic patients with a matched equivalent sample of patients treated with neuroleptics who have not developed dyskinesia. The differential diagnosis of tardive dyskinesia from Huntington's disease and other hyperkinetic disorders remains difficult, particularly in elderly patients. Development of efficient clinical methods of routinely assaying levels of biogenic amines and their metabolites in cerebrospinal fluid would be useful in making these distinctions and in determining whether one or several neurotransmitter systems are affected in drug-induced dyskinesia. The use of noninvasive computerized axial tomography has been suggested as a diagnostic technique (15) to distinguish individuals with structural brain changes, who would presumably derive little benefit from drug therapy, from individuals who are anatomically intact but suffer alteration of neuronal systems and who would be most likely to obtain relief from drug therapy. This and other novel research techniques represent significant advances in the definition and treatment of tardive dyskinesia and have already provided us with a limited degree of success in ameliorating this otherwise chronic and irreversible disorder.

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Effects of Dopamine Agonists in Tardive Dyskinesia

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The authors used a combined behavioral and neuroendocrinological strategy to investigate the relevance of abnormalities in the brain dopaminergic systems to the pathophysiology of tardive dyskinesia by assessing the effects of apomorphine, a directly acting dopamine agonist, and d-amphetamine, an indirectly acting dopamine agonist, in patients with tardive dyskinesia. Administration of I.V. d-amphetamine increased dyskinetic movements in most patients with tardive dyskinesia, a finding consistent with the dopaminergic hypothesis. Contrary to predictions based on animal models, apomorphine did not increase dyskinetic movements in these patients but instead substantially reduced dyskinesia in some patients. Patients with tardive dyskinesia did not have a greater drop in serum prolactin or a greater rise in serum growth hormone after apomorphine than normal or chronic schizophrenic subjects without tardive dyskinesia.

SOME AUTHORS (1) have suggested that one of the pharmacological abnormalities involved in the pathophysiology of tardive dyskinesia may be a functional supersensitivity of postsynaptic neurons in the nigrostriatal dopamine system induced by chronic neuroleptic administration. The main evidence supporting this suggestion comes from studies in rats, mice, and guinea pigs (2–9). Compared with saline controls, animals withdrawn from chronic treatment with neuroleptic drugs such as haloperidol, chlorpromazine, or clozapine show increased gnawing, stereotyped behavior, circling behavior, or locomotor activity when they are administered directly acting dopamine receptor agonists such as apomorphine or indirectly acting dopamine agonists such as amphetamine; these behavioral responses have been interpreted as evidence for supersensitivity of postsynaptic dopamine receptors.

Recent research has also presented some biochemical evidence consistent with supersensitivity in these animal models after termination of neuroleptics. Experiments by our group and other researchers demonstrated that rats show a greater decrease in indices of dopamine turnover after administration of apomorphine (8, 9), and Snyder and associates (10) also recently reported that dopamine receptors of these rats show increased dopamine binding.

Is the postsynaptic supersensitivity model relevant to the human clinical syndrome of tardive dyskinesia? Some indirect evidence from psychopharmacological studies in patients with tardive dyskinesia is consistent with the postsynaptic supersensitivity hypothesis. When neuroleptic drugs (such as haloperidol) that block postsynaptic dopamine receptors (11) are administered to patients with symptoms of tardive dyskinesia, the symptoms are at least temporarily reduced (12). Other drugs (such as reserpine) that deplete biogenic amines also reduce symptoms of tardive dyskinesia (13). Chronic administration of L-dopa increases dopamine in presynaptic neurons and also increases tardive dyskinesia symptoms (14). However, both L-dopa and reserpine act primarily through their effects on the presynaptic neuron; therefore, their behavioral effects are not directly relevant to tests of a postsynaptic supersensitivity theory of tardive dyskinesia.

We investigated dopaminergic mechanisms in tardive dyskinesia using a combined behavioral and biochemical approach. The effects of the two provocative test drugs that have been used most frequently in the animal studies—apomorphine, a directly acting dopamine agonist, and *d*-amphetamine, an indirectly acting dopamine agonist—were evaluated in patients with tardive dyskinesia. The behavioral strategy included quantitative ratings of changes in symptom intensity, and the biochemical evaluation involved a neuroendocrine strategy in which the effects of apomorphine on two hormones, prolactin and growth hormone, were assessed in patients with tardive dyskinesia and appropriate control subjects. Previous research has shown that dopaminergic influences have an important role in modulating the release of prolactin and growth hormone (15, 16); dopamine agonists decrease prolactin

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When this work was begun, the authors were with the Illinois State Psychiatric Institute, where Dr. Smith and Dr. Tamminga were Clinical Research Associates, Dr. Haraszti was Ward Chief, 5 W Psychopharmacology Research Ward, Dr. Pandey is Associate Director, Biological Research Laboratory, and Dr. Davis is Director of Research. Dr. Davis is also Professor of Psychiatry and Dr. Tamminga is Assistant Professor of Psychiatry, University of Chicago, Pritzker School of Medicine, Chicago, Ill. Dr. Smith is now Chief, Section of Behavioral Neurochemistry, Texas Research Institute of Mental Sciences, 1300 Moursund, Texas Medical Center, Houston, Tex. 77030, and Dr. Haraszti is in private practice in Chicago. Address reprint requests to Dr. Smith.

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and increase growth hormone (17, 18). If the dopamine agonist tests on animals chronically given neuroleptics, which have been used to demonstrate supersensitivity, can also be applied to analogous tests in patients with the clinical syndrome of tardive dyskinesia, a postsynaptic supersensitivity hypothesis would predict that both apomorphine and *d*-amphetamine should increase symptoms of tardive dyskinesia and that patients with tardive dyskinesia should show a greater fall in prolactin and a greater rise in growth hormone after apomorphine than appropriate control subjects.

METHOD

Patients with tardive dyskinesia were hospitalized on the research wards of the Illinois State Psychiatric Institute and gave informed consent to participate in evaluations and treatment studies of their disorder. Some characteristics of the patient population have been summarized in another article in this section by our research group (19).

Tardive dyskinesia symptoms were evaluated with the Smith Tardive Dyskinesia Scale (20), an observational scale in which many body parts (such as mouth, tongue, cheek, eyes, arms, fingers, waist, legs, feet, etc.) are evaluated for both dyskinetic symptoms (i.e., irregular jerking movements) and extrapyramidal symptoms (e.g., regular tremors, masked face, shuffling gait, etc.) on a 0–4 intensity scale, during a 5–10 minute structured observational rating period. One of us (R.C.S.) developed this scale with the advice of Dr. George Crane. The primary measures used in the statistical analysis were two summary scores derived from sums of ratings on specified body parts: 1) major tardive dyskinesia symptoms, which reflect primarily buccal-lingual symptoms, and 2) total tardive dyskinesia symptoms, the sum of all tardive dyskinesia items. Tardive dyskinesia ratings were done by the principal investigators (R.C.S. and C.T.) and by nurses and staff of the research ward who had achieved a fairly high degree of interrater reliability (r=.80–.95 on total tardive dyskinesia score). Tardive dyskinesia ratings were done routinely several times a week directly on the ward (observational ratings) and also at specific times after acute drug administration. Frequent videotapes (T.V.) of the patient's movements were also made, and these were scored together at a later date. The observational ratings were usually a consensus of two trained raters.

We used double-blind, placebo-controlled procedures to evaluate the behavioral effects of the drugs. All patients had been taken off neuroleptics and tranquilizers for at least 1 week, and most had been off drugs for 3–6 weeks. In the evaluation of the effects of apomorphine, each patient received varying doses of apomorphine (0.75–6.0 mg) or saline placebo subcutaneously at about 9 a.m. (the patients did not eat breakfast the morning of drug administration). Some patients did not complete the full series of doses because they developed side effects (nausea or vomiting) at one of the lower doses. Observational ratings were done 20–30 minutes after medication. In the evaluation of amphetamine's effect, either oral or I.V. procedures were used. In the oral drug protocol, the patients received an elixir of 20 mg of *d*-amphetamine sulfate or placebo solution on two different days, and observational and T.V. ratings were done 30–120 minutes after medication. In the I.V. protocol, 15–20 mg of *d*-amphetamine sulfate or saline placebo was administered intravenously through a scalp vein over a 10- to 15-minute period; blood pressure and psychological responses of the patients were used to help determine rate of administration and whether the drug should be stopped before the full dose had been administered. One patient received additional trials with higher doses of I.V. amphetamine. Observational tardive dyskinesia ratings were made before medication at 5- to 10-minute intervals for 45 minutes during the postdrug period, and again at 1 ½–2 hours after medication.

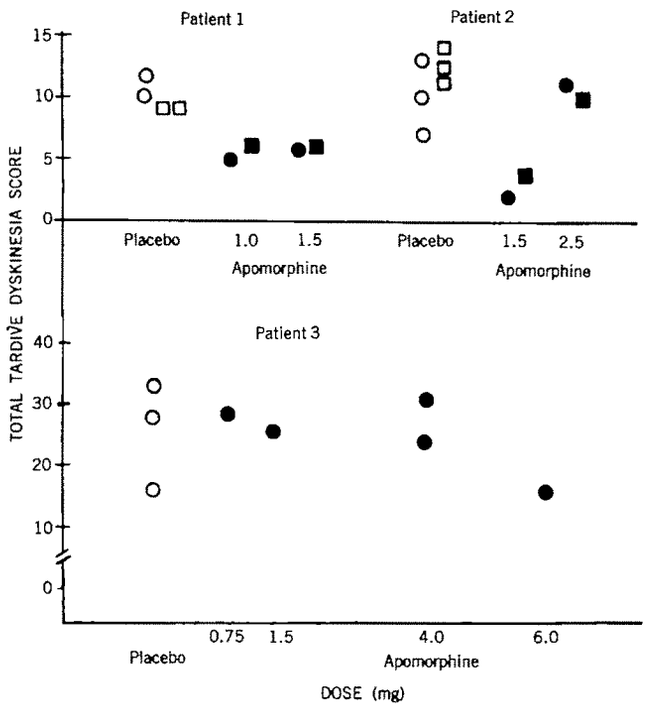
In the neuroendocrine tests, the patients had not eaten breakfast and were lying in bed with an I.V. catheter inserted in an arm vein. After they had rested for 30–45 minutes, several 5-cc blood samples were drawn and placed in heparinized tubes, and baseline tardive dyskinesia symptoms were scored. The patients then received 0.75 mg of apomorphine subcutaneously, additional tardive dyskinesia ratings were done, and blood samples were drawn over a 3-hour postdrug period. Blood samples were kept on ice and separated into plasma and red cells. Plasma was assayed for growth hormone and prolactin with double antibody radioimmunoassay techniques previously described by Pandey and associates (21) for growth hormone and with National Institute of Arthritis, Metabolism, and Digestive Diseases kits for prolactin.

RESULTS

Behavioral

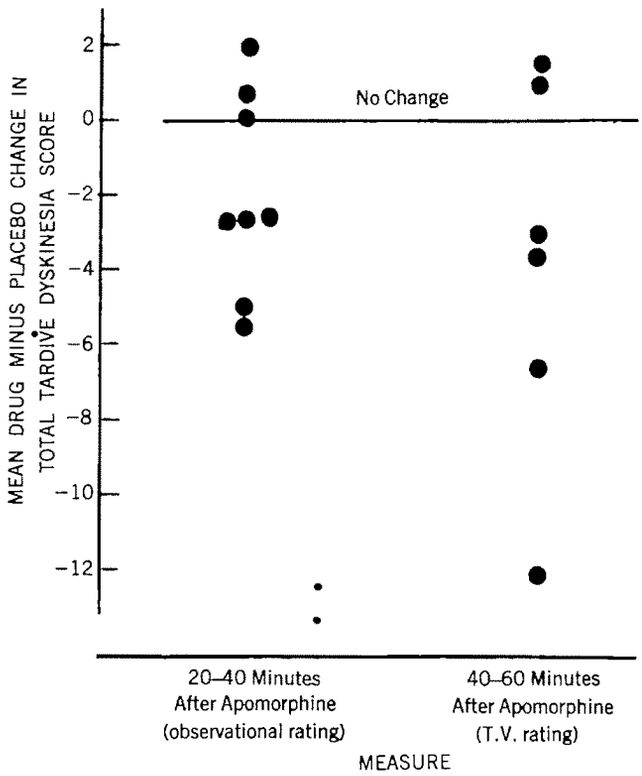
Contrary to our initial expectation, apomorphine did not increase symptoms of tardive dyskinesia. In some patients the drug produced a mild to marked decrease in symptom scores, and in other patients there was little or no change in symptoms. Figure 1 presents more detailed results in a few selected patients. Since there was no simple dose-response relationship for apomorphine effects in the dose range we tested, we computed mean differences between all apomorphine and placebo scores; these drug-placebo differences for 3 patients are presented in figure 2. Most patients experienced emetic effects of apomorphine (nausea and vomiting) at some drug dose, although the dose patients tolerated without side effects varied from 1.5 to 6.0 mg. However, there was no consistent relationship between the effects of apomorphine on emesis and movement symptoms.

FIGURE 1
Effects of Several Doses of Apomorphine on Symptoms of Tardive Dyskinesia*



*Each point represents total tardive dyskinesia score from single ratings performed either at 20–40 minutes or 40–60 minutes after apomorphine. Two or 3 different placebo days (○□) were interspersed between active apomorphine days (○■). ●○ represents observational ratings; □■, ratings from TV tape.

FIGURE 2
Effects of Apomorphine Versus Placebo on Symptoms of Tardive Dyskinesia*



*Each point represents difference of mean scores on 2–5 active apomorphine days minus mean score on 2–5 placebo days. Apomorphine and placebo days alternated in double-blind design.

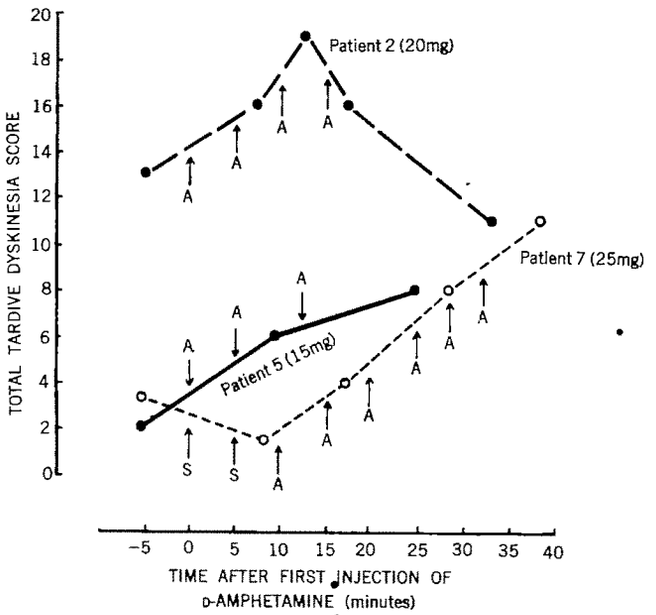
In contrast to the effects of apomorphine, *d*-amphetamine produced an increase in tardive dyskinesia symptoms in most patients compared with their predrug baseline (see figure 3), although a few patients showed only minimal changes. Compared with their predrug baseline on the same day, 5 of 6 patients showed clear increases in their tardive dyskinesia symptoms after 15–25 mg of I.V. *d*-amphetamine; patient 4 showed a small increase after 40 mg of I.V. *d*-amphetamine. The patients who showed the smallest increase in tardive dyskinesia symptoms after I.V. *d*-amphetamine also had only small or no increases in blood pressure after the drug (e.g., patient 4 had about a 10-mm rise after the 40-mg dose but returned to baseline within a few minutes). However, several of the patients who showed more pronounced increases in dyskinetic symptoms also had only small blood pressure increases. Oral amphetamine also tended to produce increases in tardive dyskinesia symptoms, but the effects of the oral drug were less strong and consistent than the increases with the I.V. drug (figure 4).

It is of interest to note that patients 5 and 6 were reported to have a history of tardive dyskinesia but showed only very questionable subclinical symptoms while in the hospital before the amphetamine I.V. test (total tardive dyskinesia scores of 0–2 on different days). On the basis of the extent of their current symptoms, serious questions were raised among the staff about the accuracy of the history of tardive dyskinesia. In both the patients, however, I.V. amphetamine intensified dyskinetic symptoms in the body areas where they were already present at marginal levels and also brought out additional symptoms in other body areas where there had been no indication of even a mild dyskinetic movement abnormality. The increase in dyskinetic symptoms remitted to baseline levels within 1 ½–3 hours after medication.

Neuroendocrine

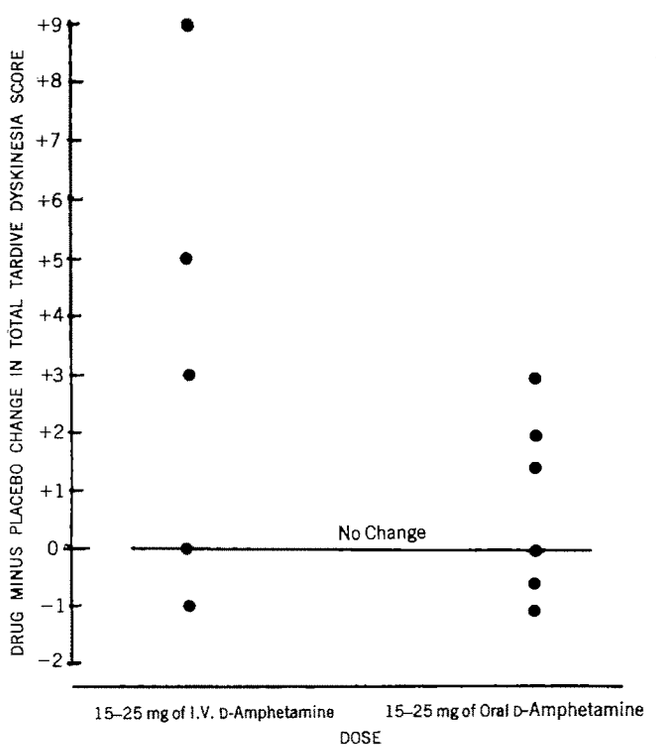
The results of the neuroendocrine tests with apomorphine also were not those anticipated on the basis of the simple supersensitivity hypothesis. As shown in figure 5, patients with tardive dyskinesia did not show a greater drop in serum prolactin after apomorphine than either of the two comparison groups—normal or chronic schizophrenic subjects without tardive dyskinesia. Similarly, patients with tardive dyskinesia did not show a greater increase in plasma growth hormone after apomorphine than either of these two comparison groups (figure 6). In fact, for both hormones there was a (nonsignificant) trend for patients with tardive dyskinesia to have slightly lower neuroendocrine responses

FIGURE 3
Effects of I.V. *d*-Amphetamine on Symptoms of Tardive Dyskinesia in 3 Representative Patients*



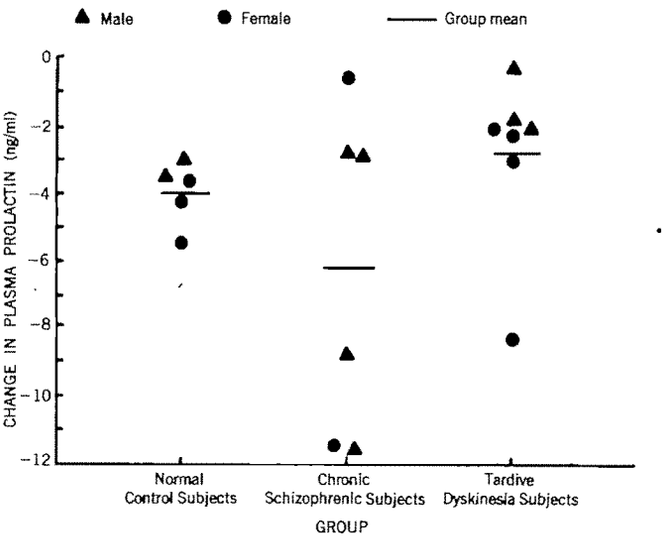
*Five or 10 mg *d*-amphetamine (A) or Saline (S) was administered at each point indicated by ↑. In the full sample, 7 of 8 patients showed increased tardive dyskinesia scores with the 15–20 mg I.V. amphetamine dose, and 1 patient showed a small increase only after a 40-mg dose.

FIGURE 4
Comparison of Effects of I.V. or Oral *d*-Amphetamine Versus Placebo on Symptoms of Tardive Dyskinesia*



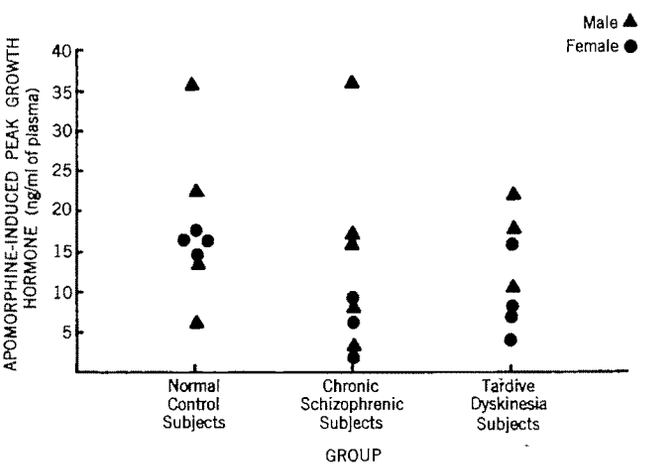
*Each point represents difference between maximum postdrug total dyskinesia score on *d*-amphetamine day minus maximum postdrug score on placebo day.

FIGURE 5
Effects of Apomorphine on Plasma Prolactin in Subjects with Tardive Dyskinesia, Chronic Schizophrenic, and Normal Subjects*



*Each point represents difference score between greatest drop in prolactin after apomorphine minus predrug baseline.

FIGURE 6
Effects of Apomorphine on Plasma Growth Hormone in Subjects with Tardive Dyskinesia, Chronic Schizophrenic, and Normal Subjects*



*Each point represents greatest rise in plasma growth hormone after apomorphine. Predrug baseline growth hormone levels were all below 1 ng/ml.

to apomorphine than subjects in both of the comparison groups.

DISCUSSION

The behavioral effects of I.V. *d*-amphetamine, which increased symptoms of tardive dyskinesia, are consistent with the interpretation that abnormalities in the brain catecholamine systems are involved in the

pathophysiology of tardive dyskinesia and also have practical clinical implications. However, the effects of apomorphine in patients with tardive dyskinesia did not seem to parallel the behavioral effects that apomorphine has been reported to have in the animal models that have been proposed as possible analogues of this human clinical syndrome. In rats withdrawn from chronic administration of neuroleptic drugs, the administration of apomorphine produces greater stereotyped movements and a greater decrease in dopamine turnover than in appropriate saline controls (2–10); these pharmacological effects have been interpreted as evidence for postsynaptic dopamine receptor supersensitivity. In human patients with tardive dyskinesia, on the other hand, apomorphine tended to reduce dyskinetic movements. The behavioral effects of apomorphine in tardive dyskinesia that we have reported in this paper are similar to the effects of this drug in two other movement disorders, Huntington's chorea (22) and dyskinesia induced by chronic administration of L-dopa in patients with Parkinson's disease (23). Some of the pathophysiology of these two diseases has also been hypothesized to involve supersensitivity of postsynaptic dopamine receptors. Several factors may account for these apparently paradoxical behavioral effects of apomorphine.

1. Recent experimental findings indicate that apomorphine also has important effects on presynaptic dopamine neurons or receptors, decreasing dopamine synthesis, as well as the stimulation of postsynaptic receptors (24–26). These effects make an unambiguous interpretation of our results regarding the postsynaptic dopamine receptor supersensitivity hypothesis in tardive dyskinesia more difficult. The unexpected effects of apomorphine in patients with tardive dyskinesia may, in fact, result from the predominance of presynaptic rather than postsynaptic effects of the drug in the dose range that we used. Consistent with this alternative suggestion, we have recently reported that, at similar doses, apomorphine either reduced or produced no change in psychotic symptoms in some actively ill chronic schizophrenic subjects rather than exacerbating psychotic symptoms, which would have been predicted from a dopamine receptor supersensitivity hypothesis of schizophrenia (27). If a drug could be found that exerted its primary pharmacological effects by direct stimulation of postsynaptic dopamine receptors but did not also have opposite effects on presynaptic dopamine neurons, clinical evaluations with this drug could provide a clearer test of the postsynaptic dopamine receptor supersensitivity hypothesis of tardive dyskinesia. One recent report (28) indicated that the drug bromocriptine, a dopamine receptor agonist that has not yet been reported to have potent presynaptic inhibitory effects, did increase symptoms of Huntington's chorea whereas apomorphine has previously been reported to decrease these symptoms (22).

2. The dose of apomorphine that we administered (0.75–6.0 mg) may have been below that required to produce increases in dyskinetic symptoms. Researchers studying dogs (29) reported that a higher dose of apomorphine was required to produce stereotyped behavior than emesis in this species.

The neuroendocrine strategy, which has been increasingly used in biological psychiatric research, has not been previously extended to the investigation of tardive dyskinesia. We used these neuroendocrine tests as an additional way to assess dopaminergically mediated response in tardive dyskinesia. If patients with tardive dyskinesia had shown a greater change in their prolactin and growth hormone responses to apomorphine than our schizophrenic and normal control groups, this would have provided strong support for the hypothesis of dopamine receptor supersensitivity. However, our negative results showing that apomorphine did not produce greater neuroendocrine responses in these patients do not disprove the receptor supersensitivity hypothesis. The neuroendocrine responses we measured may not be the best biochemical indicator of dopamine-receptor supersensitivity in the nigrostriatal dopamine system, which would be clinically manifested as a movement disorder, but may only reflect functioning of dopamine receptors in the tubuloinfundibular dopamine system (where receptors for dopaminergic activity would influence the secretion of prolactin and growth hormone). Previous research has demonstrated that with repeated neuroleptic administration tolerance develops to the increase in homovanillic acid seen after a single dose of neuroleptics (30, 31) but not to the prolactin response to neuroleptics (32). This may reflect differences in the adaptation of different areas of the brain to repeated neuroleptic administration.

The positive results with *d*-amphetamine may have implications both for theory and clinical testing. Since amphetamine exerts its major effect through release and blockade of reuptake of catecholamines in presynaptic neurons, more attention should be given to possible defects in the functioning of presynaptic neurons in tardive dyskinesia. Of greater clinical significance, however, is our finding that amphetamine could elicit recognizable symptoms of tardive dyskinesia in some patients who had very minimal or subclinical signs of the disease. Since the symptoms of tardive dyskinesia vary considerably from day to day, routine clinical evaluations might easily miss the diagnosis in some of these patients. In cases in which there is some reason (clinical history or length of neuroleptic administration) to presume the possibility of tardive dyskinesia, an I.V. amphetamine test might be a useful additional diagnostic test to help uncover early cases of the disorder. For those patients who demonstrate symptoms of tardive dyskinesia during this provocative test, a more thorough evaluation can be pursued and remedial measures—reduction of neuroleptic doses, extended drug holidays, and therapeutic trials with drugs that might decrease tardive dyskinesia symptoms—could be instituted to try to prevent the progression of the disease.

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Cholinergic Influences in Tardive Dyskinesia

BY CAROL A. TAMMINGA, M.D., ROBERT C. SMITH, M.D., PH.D., STEPHEN E. ERICKSEN, M.D., SIDNEY CHANG, M.D., AND JOHN M. DAVIS, M.D.

The authors conducted placebo-controlled double-blind studies of physostigmine, choline, and deanol in 12 patients with tardive dyskinesia. Physostigmine and choline both had a positive therapeutic effect on tardive dyskinesia, but the authors note that interpretation of these results is not entirely clear because they found that the sedation effect of physostigmine may nonspecifically reduce the intensity of tardive dyskinesia symptoms. Deanol was not found to be effective in this group of patients; the authors suggest that this drug should not be assumed to be generally effective unless effectiveness is verified by a large placebo-controlled double-blind study.

THERE IS BOTH basic and clinical evidence suggesting that the cholinergic as well as the dopaminergic system may be involved in extrapyramidal movement disorders (1). Diseases like parkinsonism, Huntington's chorea, Gilles de la Tourette's syndrome, and tardive dyskinesia respond with movement exacerbation or remission to cholinomimetic and cholinolytic agents. The familiar example is Parkinson's disease, in which movements worsen with drugs that increase brain acetylcholine effect (e.g., physostigmine) and are benefited by anticholinergic drugs (2, 3). Studies in experimental animals demonstrate a mutual antagonism between cholinergic and dopaminergic neurons within the neostriatum (4–6); these preclinical studies substantiate inferences made from the observed human pharmacology.

Tardive dyskinesia has been hypothesized to be a dopamine-supersensitive disease (7). Gerlach and associates (8), Fann and associates (9), and Klawans and Rubovits (10) found that physostigmine, a cholinomimetic agent, benefited symptoms, but Gerlach and associates and Klawans and Rubovits also demonstrated that two anticholinergic drugs, biperiden and scopolamine, aggravated tardive dyskinesia movements. A preliminary report from Davis and associates indicated that oral choline used in a precursor loading strategy alleviated tardive dyskinesia (11). Deanol, a postulated but controversial acetylcholine precursor, has been used in the treatment of tardive dyskinesia. Many uncontrolled studies and single case reports detail therapeutic trials of deanol (12–18); some report a positive therapeutic effect. We are not aware of any double-blind placebo-controlled studies validating the clinical impression that deanol helps tardive dyskinesia.

This paper reports results from double-blind placebo-controlled trials of cholinomimetic agents. Physostigmine was used in an acute single dose trial to assess the response of tardive dyskinesia movement to a cholinergic agent. The acute effect of an I.V. dose of physostigmine may indicate the potential efficacy of the treatment of tardive dyskinesia symptoms with chronic cholinergic agents. Chronic administration of deanol was evaluated because of the reports of its efficacy and its purported cholinergic actions. Since there is a body of evidence from animal studies that oral choline does indeed increase brain acetylcholine (19–21), we also carried out a therapeutic trial of oral choline.

SUBJECTS AND METHOD

Twelve patients with tardive dyskinesia were included in this study. The majority came from long-term mental health facilities; 2 were referred from the community. Specific characteristics of the population are summarized in table 1.

On admission to the study the patients were withdrawn from all medication and allowed to be drug-free for 2 weeks (patient 5 continued to receive 100 mg of phenytoin sodium [Dilantin] three times a day). Each gave voluntary consent to the procedures. Since the whole protocol consisted of a number of different acute and chronic drug trials, the sequence was arranged so that there was a 5–7-day drug-free period following any chronic dose of medication and a 2–3-day period following any single-dose drug trial.

The tardive dyskinesia (TD) rating scale used was an

Presented at the 129th annual meeting of the American Psychiatric Association, Miami Beach, Fla., May 10–14, 1976.

At the time this research was started, all of the authors were with the Illinois State Psychiatric Institute, Chicago, Ill. Dr. Tamminga is now Assistant Professor of Psychiatry, University of Chicago. Dr. Smith is now Chief, Section of Behavioral Neurochemistry, Texas Research Institute of Mental Sciences, Houston, Tex. Dr. Ericksen and Dr. Chang are Ward Chiefs and Dr. Davis is Director of Research, Illinois State Psychiatric Institute. Dr. Davis is also Professor of Psychiatry, University of Chicago. Address reprint requests to Dr. Tamminga at the University of Chicago, Department of Psychiatry, 950 E. 59th St., Chicago, Ill. 60637.

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TABLE 1
Characteristics of the 12 Tardive Dyskinesia (TD) Patients Studied

Patient	Sex	Age	Psychiatric Diagnosis	Areas of Most Prominent TD Symptoms	Areas of Other Prominent TD Symptoms
1	Female	68	Chronic schizophrenia	Mouth, tongue, eyes	Hands, feet
2	Female	63	Chronic schizophrenia	Hands	Mouth
3	Female	57	Chronic schizophrenia	Mouth, tongue, waist	Hands
4	Female	33	Schizophrenia; mental retardation	Face	Hands
5	Female	49	Chronic schizophrenia	Mouth	Hands
6	Female	59	Chronic schizophrenia	Mouth, tongue, face	Feet
7	Female	59	Chronic schizo-affective schizophrenia	Mouth	Feet
8	Female	72	Chronic schizophrenia	Face	Hands
9	Female	73	Chronic schizophrenia	Mouth, tongue, face	Hands, feet
10	Male	52	Chronic schizophrenia	Hands, feet	Mouth
11	Male	34	Chronic schizophrenia	Hands	Tongue, face
12	Male	25	Schizo-affective schizophrenia	Face	Waist

observational scale in which movements of specific body parts were rated from 0–4; the TD scale rated both parkinsonian and tardive dyskinesia movements (see the article by Smith and associates in this Special Section of the *Journal*). The scale was developed by one of us (R.C.S.) in collaboration with Dr. George Crane. Patients were rated at least three times a week by trained raters who had an interrater reliability of .80–.95. During the initial evaluation period ratings were done daily and videotaping was done twice a week. We continued to videotape during acute and chronic drug trials at critical times and rated them in series at a later time. We found that evaluating videotapes in this manner fostered a focus on the comparative change in movements.

Three drugs that have been purported to affect central cholinergic mechanisms (deanol, choline, and physostigmine) were used with behavioral evaluations. Deanol was evaluated in a chronic schedule with a double-blind placebo-controlled design in doses beginning at 500 mg a day and increasing to 1250 mg a day over a 3-week period. Daily ratings were done in the morning and afternoon and twice-weekly interviews were conducted and videotaped.

Choline was used in a preliminary nonplacebo trial with a limited number of patients. The drug was given orally in doses beginning at 3 g a day, increasing to 18 g over a 3-week period. Ratings were done daily and videotaping twice weekly. Blood samples were collected for choline levels and prolactin values before each dose increase.

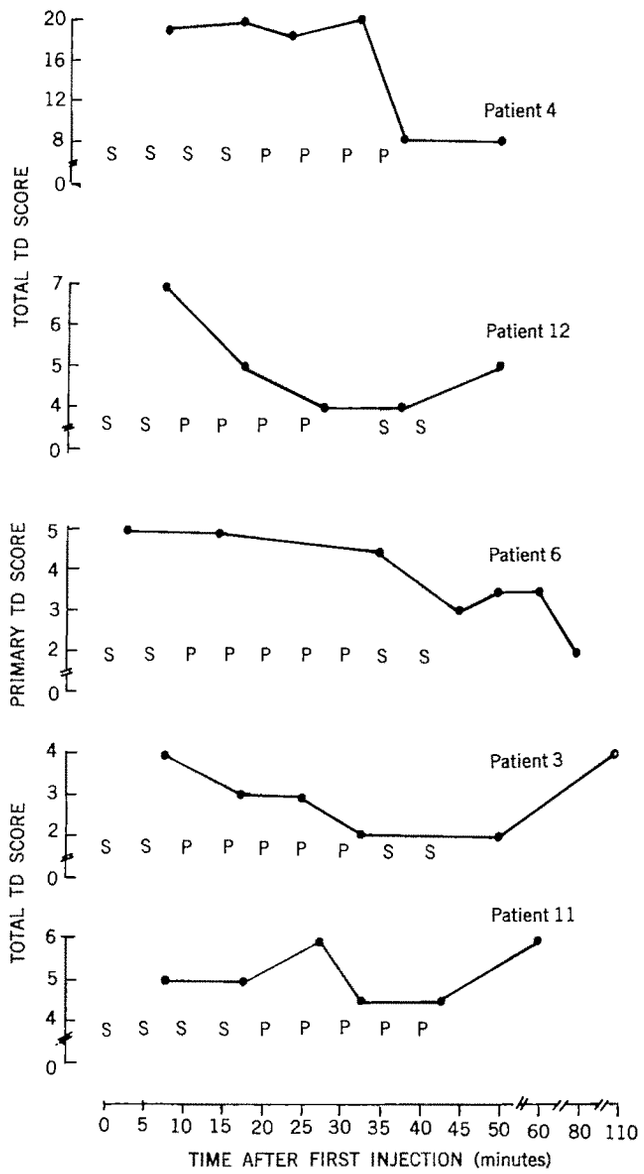
Physostigmine was given in an acute dose. Sixty minutes before the test the patient was given 1 cc of methscopolamine as a peripheral cholinergic blocker. Before the study the patients rested in their rooms for a number of hours. The procedure entailed inserting a butterfly catheter into a forearm vein with a syringe of heparinized saline attached; the patient was given 30 minutes to acclimate to the test procedure before beginning. Physostigmine was then administered in a .5-ml I.V. bolus every 5 minutes with a variable number of initial injections of sterile saline. Up to 2.0 mg of physostigmine was given; the drug injections were stopped before patient discomfort rose as evaluated by the nonblind observer. One of us prepared and gave the medication and remained nonblind; another rated the movements throughout the test and remained blind to the drug given and to the number of initial saline injections. Using the same clinical method, I.V. neostigmine and I.V. methohexital sodium (Brevital) were given as control drugs, one to control for peripheral cholinergic action and the other for sedation, respectively.

Physostigmine

Physostigmine, a central acetylcholinesterase inhibitor, was tested to document previous reports of the effect of this drug in reducing symptoms of tardive dyskinesia. As shown in figure 1, each of 5 patients had some decrease in movements within 10–20 minutes of active drug administration; this ranged from 20%–80% suppression of predrug levels. These data are consistent with previously reported studies, which generally show that physostigmine reduces dyskinetic movements in a tardive dyskinesia population. Neostigmine, a drug that has only peripheral acetylcholinesterase activity, produced no effect on the movements of 2 patients (see figure 2).

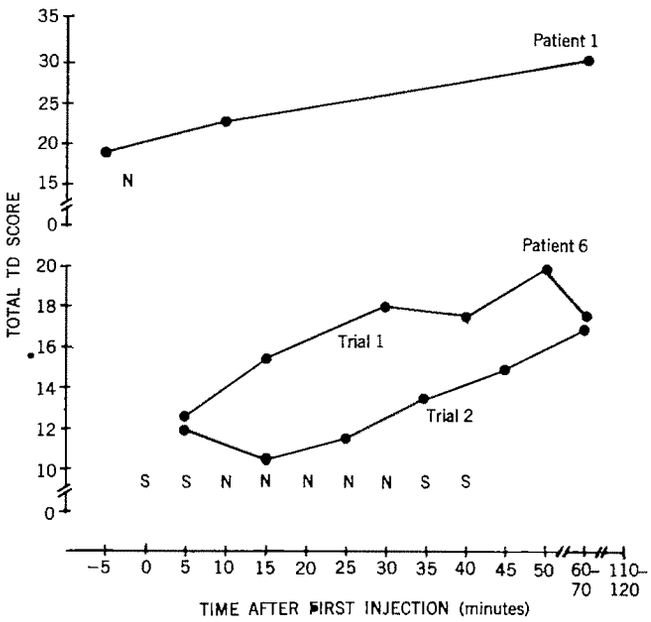
During the course of the study our attention was drawn to the effect of an activation/sedation factor on tardive dyskinesia movements. The response of one patient (patient 6) to physostigmine necessitated this consideration. Her movements were initially suppressed with physostigmine, but on repeated testing with electromyography (EMG) (a test that includes repetitive pinprick-like stimulation) the expected movement diminution was blocked (see figure 3). This result suggests that the stimulation of the EMG test counter-

FIGURE 1
Effects of I.V. Physostigmine on TD Symptoms of 5 Patients*



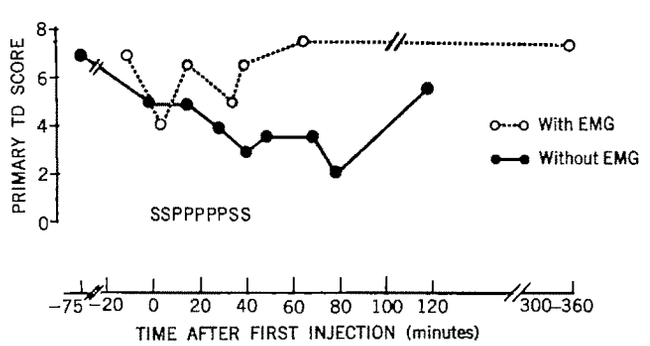
*P indicates administration of .5 mg of physostigmine; S indicates administration of saline.

FIGURE 2
Effects of I.V. Neostigmine on TD Symptoms of 2 Patients*



*N indicates administration of .5 mg of neostigmine; S indicates administration of saline.

FIGURE 3
Effects of I.V. Physostigmine on TD Symptoms With and Without EMG Testing in Patient 6*



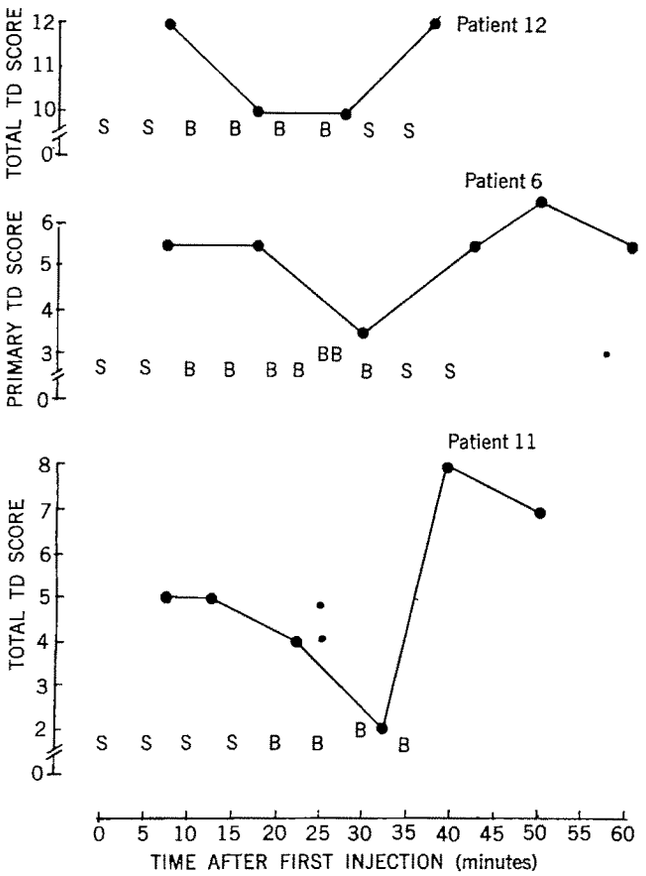
*P indicates administration of .5 mg of physostigmine; S indicates administration of saline.

acted the physostigmine effect and, furthermore, that some of physostigmine's effect might be due to the sedative properties of the drug. Other data also indicated that symptoms of tardive dyskinesia might fluctuate with activity level. It is known, for instance, that tardive dyskinesia movements disappear with sleep. Furthermore, in a survey of symptoms of 198 patients in a state hospital population conducted by one of us (R.C.S.) a positive relationship was found between patients with a higher activity level and those with greater dyskinesia symptoms. Therefore, we wondered whether some of the physostigmine effect in reducing tardive dyskinesia symptoms could be due to its sedative properties.

To test this possibility, further I.V. methohexital sodium, a short-acting barbiturate, was used as a control for sedation with the same clinical protocol. The methohexital-induced sedation caused a similar fall in the TD scores of 3 patients; this fall coincided with methohexital's sedative effect (see figure 4).

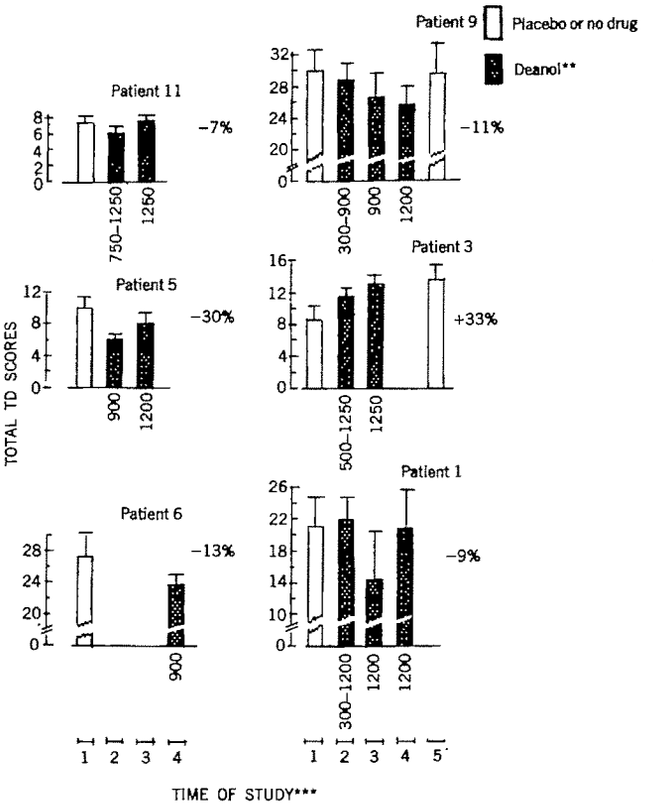
That the sedative properties of physostigmine may play a role in the suppression of tardive dyskinesia movements appears to be a previously unconsidered factor. It can be added, however, that our clinical observation seems to be able to distinguish between movement remission with physostigmine and with methohexital. We found that the stimulation of talking to the patients rather than just observing them in a

FIGURE 4
Effects of I.V. Methohexital Sodium on TD Symptoms of 3 Patients*



*B indicates administration of 1.0 mg of methohexital sodium (Brevital); S indicates administration of saline.

FIGURE 5
Effects of Deanol on TD Symptoms of 6 Patients*



*The average percentage change in movements with deanol treatment is indicated to the right of each graph.
**The doses of deanol are given in mg.
***1=placebo; 2=week 1; 3=week 2; 4=week 3; 5=washout period.

drowsy state could distinguish between the movement suppression with physostigmine and that with methohexital. With the methohexital-sedated patients such verbal arousal caused their movement remission to disappear; however, with physostigmine sedation arousal did not readily diminish the movement suppression.

In summary, it has been shown that physostigmine reduced tardive dyskinesia movements in this population of 12 patients by 20%–80%. We found a similar degree of movement remission with methohexital sodium, however; this makes the interpretation of the physostigmine data less clear. Previous studies have not controlled for physostigmine sedation. Although it is probable that physostigmine suppresses tardive dyskinesia movements, delineation of the mechanism—whether by sedation or specific cholinergic activity—needs continued careful evaluation.

Deanol

Our results show that deanol did not improve tardive dyskinesia to any substantial degree in the 6 patients who received it. Figure 5 gives the total TD scores for each of the 6 patients averaged for each drug and placebo week. The percent differences in the TD scores between the average of the deanol weeks and the placebo week is indicated to the right of each graph. Although deanol produced a variable reduction in TD symptoms in some patients, exacerbation occurred in others. The symptom reduction that did occur in some patients did not persist with continued treatment and did not appear to be dose related. When we looked at primary symptoms alone (buccolingual-masticatory), we also found no overall beneficial effect of deanol.

What is particularly striking in deanol's effect is the variability in movement ratings on a day-to-day basis. Figure 6 illustrates the range of TD scores in 2 patients who were taking 500–1250 mg of deanol a day and underlines the wide day-to-day variability in TD symptoms. Our conclusion is that the effect of deanol on reducing the symptoms of tardive dyskinesia is not great in the doses used in this study.

Choline

In a preliminary nonblind trial of oral choline in 4 patients with moderate to severe TD symptoms the data indicate some therapeutic effect. Two of the 4

FIGURE 6
Week-to-Week Variability in TD Ratings of 2 Patients Receiving 500–1250 mg of Deanol a Day

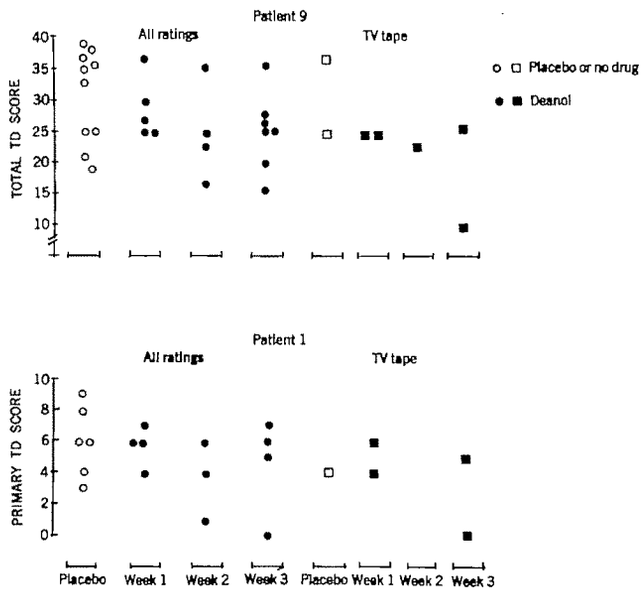
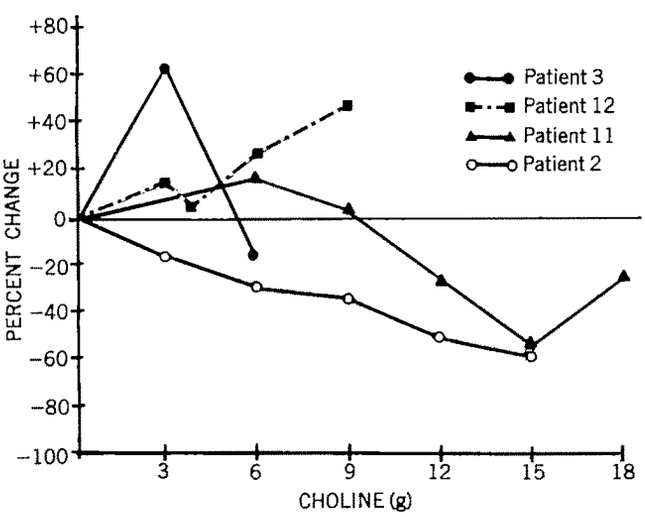


FIGURE 7
Percent Change from Total Baseline TD Scores in Response to Oral Choline in 4 Patients*



*Patient 12 became agitated and depressed, patient 3 became depressed, and both were withdrawn from choline. Patient 11 and patient 2 continued in the study to a therapeutic dose range.

patients showed a trend toward improvement at doses higher than 6 g a day (see figure 7). Unfortunately, administration of the drug had to be stopped in 2 patients due to depression before an adequate dose was reached. In neither of these patients was any substantial improvement noted. Patient 12 was agitated by the depression, which likely exacerbated his tardive dyskinesia movements. We have previously suggested that drugs which increase central acetylcholine may cause depression (22). That this depression was a side effect of oral choline treatment is supported by two factors: 1) the depression remitted with drug withdrawal, and 2) atropine reduced the depressive symptoms while the patient was receiving choline. Depression could have been a predicted side effect of cholinomimetic drug treatment according to the cholinergic-adrenergic balance theory of depression (23). This mood change with choline also tends to confirm the central site of action of choline in humans. Our preliminary findings of therapeutic benefit with choline in tardive dyskinesia need placebo-controlled trials before a definitive statement can be made about its therapeutic effect.

DISCUSSION

There is evidence both from our work and from others of a cholinergic influence in tardive dyskinesia. Our study shows that physostigmine and choline benefit tardive dyskinesia, but the interpretation of these results is not entirely clear because of a methodological variable that surfaced during the study, namely, that sedation may nonspecifically reduce the intensity of tardive dyskinesia symptoms. This observation surfaced serendipitously, as detailed above, when EMG testing was done during a physostigmine test and the expected movement suppression did not occur. These results indicate that sedation may have been a factor in previously reported studies with physostigmine. In our opinion future studies of tardive dyskinesia must include a sedative control for drugs with sedative properties in order to separate the nonspecific movement suppression due to sedation from the specific anti-TD action of a cholinergic agent.

However, on the basis of clinical evidence, we suspect that physostigmine produces an effect beyond sedation because the movement-suppressant physostigmine effect persisted even with arousal, whereas with methohexital this was not the case. Our preliminary findings with choline tend to support the idea of positive therapeutic efficacy of cholinergic agents in tardive dyskinesia. We began using choline as a long-term cholinomimetic agent because of evidence that it increases central acetylcholine levels. Our preliminary results indicating a positive therapeutic effect, although suggestive, are not sufficient to draw definitive conclusions about the general therapeutic efficacy of choline in tardive dyskinesia. Control studies are needed to document choline effect. Our clinical observation of severe depression in 2 patients warrants continued careful observation.

The findings with deanol, as tested in this placebo-controlled double-blind design, failed to confirm previous nonblind trials of the drug that suggested a treatment effect of deanol in tardive dyskinesia. There are a number of important methodological artifacts that may introduce limitations into the interpretation of results from uncontrolled trials of treatment regimens in tardive dyskinesia. First, it has been shown that there is a

large placebo effect with any treatment regimen used in tardive dyskinesia. In previous work with amantadine we found a 50% decrease in tardive dyskinesia symptoms in an uncontrolled study; later controlled studies with amantadine showed that this decrease was a placebo effect (24, 25). In addition, studies with tardive dyskinesia patients frequently include positive environmental changes as a necessary accompaniment to movement assessment (for example, increased staff contact or transfer to a well-staffed research ward), all of which generally alleviate movements. There is a second important reason why controlled studies are necessary. Although tardive dyskinesia symptoms are often permanent, there is a certain spontaneous remission rate following neuroleptic withdrawal. Obviously, this spontaneous remission factor can confound drug response results.

These reasons make it imperative that a double-blind methodology be used in all drug studies in tardive dyskinesia. Deanol use is further complicated by the fact that its pharmacological action as an acetylcholine precursor is not yet firmly established; there continue to be conflicting data as to its action (18). Therefore, our largely negative results with deanol may not be strong evidence against the cholinergic influence in tardive dyskinesia because deanol is not a confirmed acetylcholine precursor.

In summary, although deanol may be effective in treating isolated patients, we can say that the drug should not be assumed to be more generally effective unless this is verified by a large double-blind placebo-controlled study.

Normal regulation of extrapyramidal function may depend on a sensitive balance between dopamine neurons and acetylcholine fibers in the basal ganglia. Previous clinical studies have demonstrated the association of acetylcholine with dopamine in extrapyramidal movement disorders. We have provided data in this study that add both to the pharmacological characterization of cholinergic influences in tardive dyskinesia and to the evaluation of therapeutic agents. The importance of working out the effects of cholinomimetic agents lies in the potential contribution of chronic cholinergic drugs to the treatment of tardive dyskinesia.

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Electrophysiological (H-Reflex) Studies of Patients with Tardive Dyskinesia

BY JOHN W. CRAYTON, M.D., ROBERT C. SMITH, M.D., DAVID KLASS, M.D., SIDNEY CHANG, M.D., AND STEPHEN E. ERICKSEN, M.D.

The authors studied the H-reflex recovery curves of 31 schizophrenic patients with tardive dyskinesia in response to acute administrations of apomorphine, amphetamine, or physostigmine and compared them with the curves of chronic schizophrenic patients without tardive dyskinesia and normal volunteers. Their most unexpected finding was the absence of an H-reflex in 9 of the 31 patients with tardive dyskinesia. They also found a relationship between severity of tardive dyskinesia and the value of the facilitatory peak H_x: patients with more severe tardive dyskinesia symptoms had significantly higher values for H_x.

TARDIVE DYSKINESIA is a disabling disorder that increases the morbidity associated with chronic psychotic illness. Despite the intensified interest in this disorder in recent years, there is as yet no consensus about its etiology or underlying pathophysiology, nor have there been consistent results with numerous treatment approaches taken by various investigators. Tardive dyskinesia has been improved in some patients by treatment with agents that reduce the efficacy of dopaminergic transmission, such as haloperidol (1), reserpine (2), and manganese (3), and by substances that enhance cholinergic activity, such as physostigmine (4–7), deanol (8), and choline (9). However, results are not consistent, and some patients continue to be refractory to all treatment.

It seems possible that if more were known about the underlying pathophysiology of tardive dyskinesia, treatment could be tailored to the specific disease process. With this possibility in mind, we initiated an electrophysiological study of this disease in an effort to more carefully delineate possible underlying pathophysiological mechanisms. Our approach has been to assess the excitability of the spinal alpha motoneuron pool, since this has been shown to be altered in a variety of movement disorders (10). Furthermore, we have shown that some patients with schizophrenia have altered spinal reflex excitability patterns (11, 12). We wished to determine if chronic schizophrenic patients who also had tardive dyskinesia could be distinguished from those without tardive dyskinesia and from normal subjects.

SUBJECTS

Our subjects were 31 patients with a diagnosis of chronic schizophrenia who also had tardive dyskinesia. Twenty-three of the patients were long-term inpatients at Manteno State Hospital, Manteno, Ill. The other 8 patients had been admitted to a special research unit at the Illinois State Psychiatric Institute (ISPI) for clinical evaluation and treatment of tardive dyskinesia. The 31 patients were identified for the study through a survey of the incidence of tardive dyskinesia in the Illinois state hospital system carried out by one of us (R.C.S.). Data for these 31 patients were compared with data for chronic schizophrenic patients without tardive dyskinesia at Manteno State Hospital and with data on normal healthy volunteers. Voluntary consent to the procedures was obtained from all patients.

METHOD

Patients were rated as to tardive dyskinesia (TD) and parkinsonian symptoms as described in this special section of the *Journal* by Smith and associates. Patients were divided into three groups according to whether they received high, medium, or low TD scores on the scale they describe. Patients with high ratings had total TD scores of more than 10; medium, 5 to 9; and low, less than 5.

Spinal monosynaptic reflex testing was carried out by the procedure described by Hugon (13). The patient reclined in a large easy chair with his or her leg

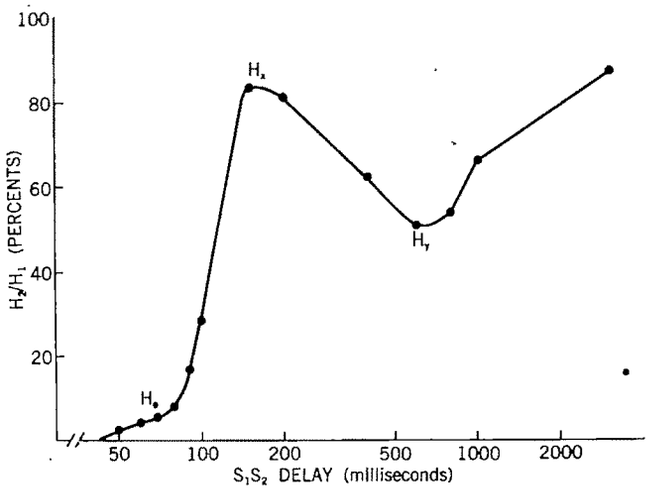
Presented at the 129th annual meeting of the American Psychiatric Association, Miami Beach, Fla., May 10–14, 1976.

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The authors would like to thank Sheila King and Richard Rued for technical assistance.

FIGURE 1
H-Reflex Recovery Curve for 19 Normal Volunteer Control Subjects*



*The principal features of the curve studied are the initial return of recovery (H_o), the "secondary facilitatory" peak (H_x), and the following trough (H_y).

extended and flexed at an angle of approximately 30°. The I-A afferent sensory fibers were stimulated with a bipolar surface electrode placed in the popliteal fossa; the reflex activity was recorded by surface electrodes placed over the distal tendon and belly of the soleus muscle. Recruitment curves were generated for each patient by gradually increasing the stimulus intensity and measuring the amplitude of the M or direct muscle response and the H or reflex response (14).

The alpha motoneuron excitability recovery cycle was determined by giving paired stimuli (S_1S_2) at delays of between 50 milliseconds and 5 seconds at a stimulus intensity that gave a near-maximal H-response. The ratio of H_2 to H_1 was determined at each stimulus delay for from 3 to 5 determinations at each point. For purposes of statistical comparisons, three principal features of the recovery cycle were studied: H_o, which was the S_1S_2 delay time at which there was an initial return of excitability; H_x, the maximum H_2/H_1 value between 100 and 300 milliseconds; and H_y, the lowest value for H_2/H_1 in the epoch between 300 and 600 milliseconds' delay. (See figure 1.)

Much of our interest centers on H_x, which is increased in patients with Parkinson's disease (10, 15, 16) and reduced in some schizophrenic patients (12). The peak H_x is thought to be partially influenced by descending supraspinal influences; there is also some evidence that it is affected by altered dopaminergic tone (17, 18).

Selected patients at ISPI were treated with acute administrations of apomorphine, amphetamine, or physostigmine. The subjects treated with apomorphine were studied within 1 to 2 hours of the administration of the drug. The patients studied during the physostigmine administration were studied continuously during the repeated injections of 5 mg of physostigmine or saline at 10-minute intervals over the course of 1 hour.

TABLE 1
Presence or Absence of H-Reflex in 30 Schizophrenic Patients with Tardive Dyskinesia

TD Score Status	H-Reflex	
	Absent	Present
Low (N=7)	0	7
Moderate (N=8)	1	7
High (N=15)	8	7

RESULTS

Absence of H-Reflex in Tardive Dyskinesia

Nine schizophrenic patients with tardive dyskinesia failed to have any H-reflex activity (see table 1). In 1 of the 31 patients the presence or absence of an H-reflex was equivocal because the amplitude of the response was similar to background electrical activity. For the 30 subjects in whom the presence or absence of H-reflex activity could be unequivocally determined the absence of H-reflex activity was highly correlated with TD score: 8 of the 9 subjects who lacked an H-reflex had high TD scores; the ninth had a moderate TD score. All of the patients with low TD scores had H-reflexes ($\chi^2=8.0555$, $p<.02$).

In order to determine whether the absence of an H-reflex was a reflection of the effect of dyskinetic movements of the patient at the time of the study we reevaluated 3 patients at times when they showed less movement. Each patient repeatedly failed to show any H-reflex response despite all efforts to elicit one by moving the stimulating and recording electrodes and by facilitation of the reflex via the Jendrassik maneuver (19).

H-Reflex Recovery Cycle

An important difference between our normal volunteer control subjects or the schizophrenic control subjects without TD and TD subjects was the greatly increased run-to-run and test-retest variability in the TD patients. The coefficient of variability (SD×100) for the normal subjects for a set of 5 determinations of H_x was in the range of 4%–8%. Therefore, the recovery cycle is a highly constant neurophysiological feature in normal subjects. In contrast, the coefficient of variability range for TD subjects was 9%–109% (mean, 43%). Some of this variability was undoubtedly due to dyskinetic movements that affect spinal reflex pathways, but the variability was not substantially different when subjects were tested on days when they had high or low TD ratings. Furthermore, there was no consistent relationship between TD scores and the coefficient of variability. In fact, 3 of the subjects with the highest TD scores had low coefficients of variability.

Recovery cycles of motoneuron excitability were studied in the 22 TD patients who had H-responses (including the 1 patient whose response was equivocal). Comparative analysis of H_o, H_x, and H_y values of the

TABLE 2

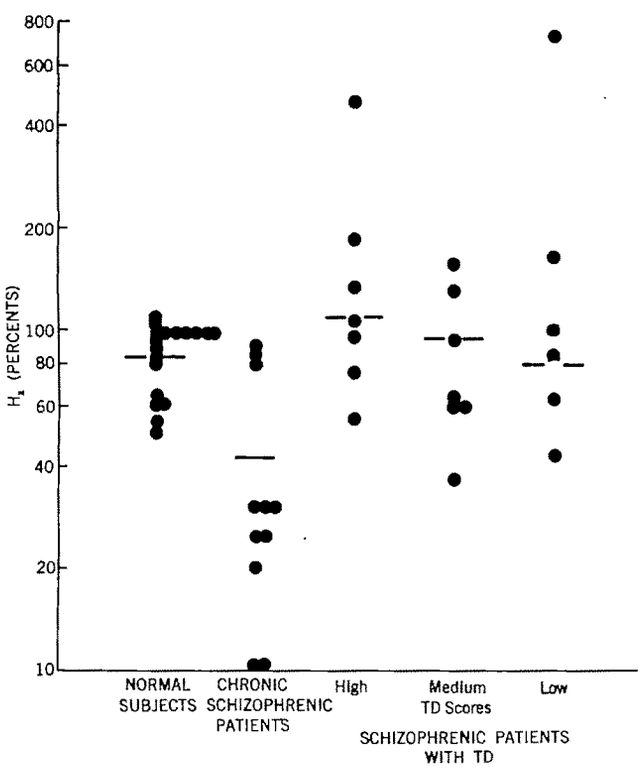
H-Reflex Recovery Values for 19 Normal Volunteer Control Subjects, 11 Chronic Schizophrenic Patients Without TD, and 20 Schizophrenic Patients with TD*

Item	H-Reflex Recovery Values					
	H_o (milliseconds)		H_x (percents)		H_y (percents)	
	Mean	SD	Mean	SD	Mean	SD
Normal control subjects	83.2	20.8	86.7	20.7	38.5	20.8
Chronic schizophrenic patients without TD	—	—	55.7	29.3	34.6	32.7
Schizophrenic patients with TD						
High TD score (N=6)	55.3	67.8	109.3	46.8**	18.4	34.2
Moderate TD score (N=8)	100.8	31.7	80.8	30.0	34.5	19.5
Low TD score (N=6)	162.5	87.6***	72.3	28.7	35.0	29.2
Total	91.8	78.5	86.7	37.3**	27.1	29.8

*Extreme values of 747 and 488 were excluded from this analysis.
**$p<.05$ when compared with chronic schizophrenic patients without TD by two-tailed t test.
***$p<.05$ when compared with normal subjects by two-tailed t test.

FIGURE 2

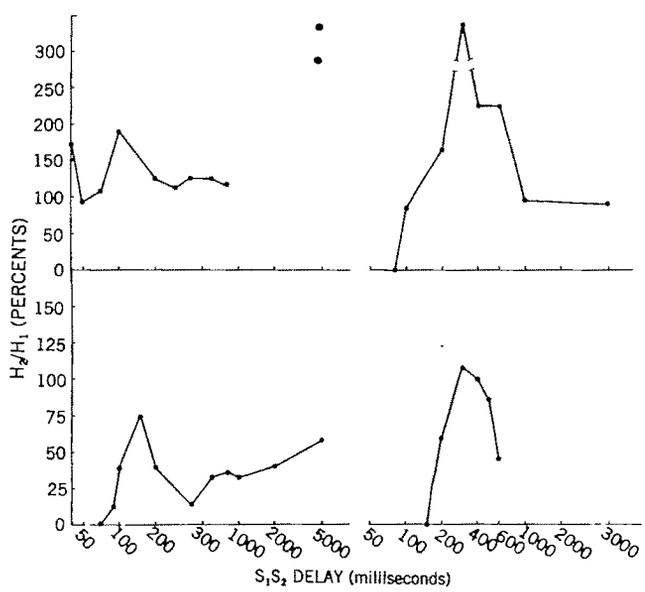
H_x Values of 19 Normal Volunteer Control Subjects, 11 Chronic Schizophrenic Patients Without TD, and 22 Schizophrenic Patients with TD*



*The two extreme values of H_x were excluded from calculations of the group means (horizontal lines).

FIGURE 3

H-Reflex Recovery Curves for 4 Schizophrenic Patients with High TD Scores*

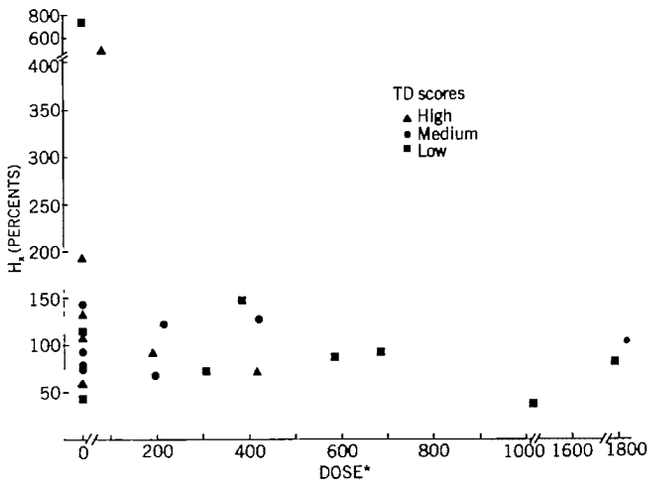


*Note the great variability in the curves and the differences between these curves and the one shown for 19 normal subjects in figure 1. Also note the generally higher values for peak H_x in these 4 patients.

recovery cycle between these TD patients and 19 control subjects is shown in table 2, and the values of H_x are displayed in figure 2. Examples of H-reflex curves of 4 schizophrenic patients with high TD scores are shown in figure 3. Chronic schizophrenic patients had lower values for H_x than control subjects, but patients with TD had higher values. Even when the two extreme values of 747 and 488 were excluded from the analysis, two-tailed t tests suggested that the patients with high TD scores differed significantly from both normal control subjects and chronic schizophrenic patients without TD (see table 2).

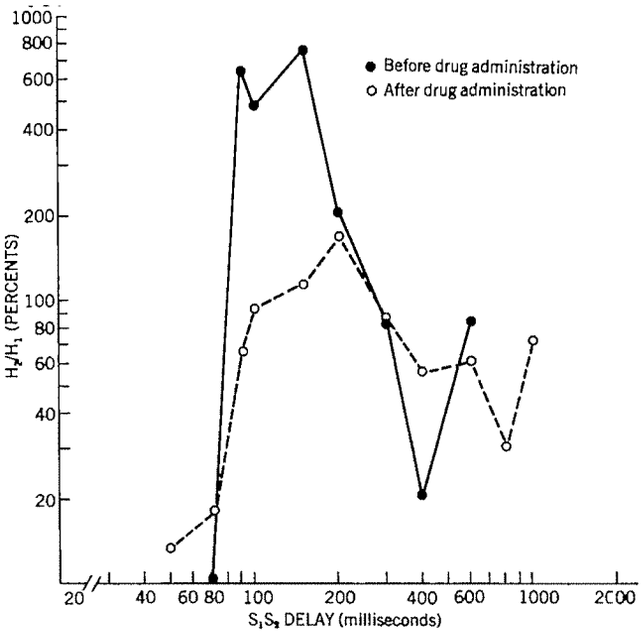
Previous studies of schizophrenic patients (11, 12) have indicated that H_y is of little discriminative value because of its great variability. Similarly, in this study we found no significant relationships between H_y and TD scores. Mean values of H_y in patients with TD were similar to those for normal subjects and chronic schizophrenic patients without TD. It should be noted, therefore, that the significant separation of values be-

FIGURE 4
H_x Values and Medication Dose for 31 Schizophrenic Patients with TD, According to TD Score



*Patient medication was calculated in terms of chlorpromazine dose in mg per day or an equivalent dose of other neuroleptics.

FIGURE 5
Effect of Infusion of 15 mg of I.V. Amphetamine Sulfate on the H-Reflex Recovery Curve of a Schizophrenic Patient with TD*



*The most prominent change is a reduction in the value for the peak H_x.

tween schizophrenic patients with and without TD occurs in only one part of the recovery cycle.

Medication Effects

For the experimental group as a whole there was no significant relationship between recovery curve or presence or absence of an H-reflex and current medication or total phenothiazine equivalents during the patient's lifetime. Nor was there a significant correlation between the amount of phenothiazine and TD scores in the whole group. However, if only the patients receiving psychotropic medication are considered, there was a significant correlation of higher drug doses and lower TD scores. The relationship of TD scores to H_x and current medication is illustrated in figure 4. As discussed above, H_x tended to be higher in patients with high TD scores. Figure 4 illustrates that dose of current medication may also be a contributing variable. Six of 7 patients with high TD scores and 6 of 7 with medium TD scores were receiving less than 250 mg of chlorpromazine or equivalent per day ($\chi^2=6.61$, $df=2$; $p<.05$).

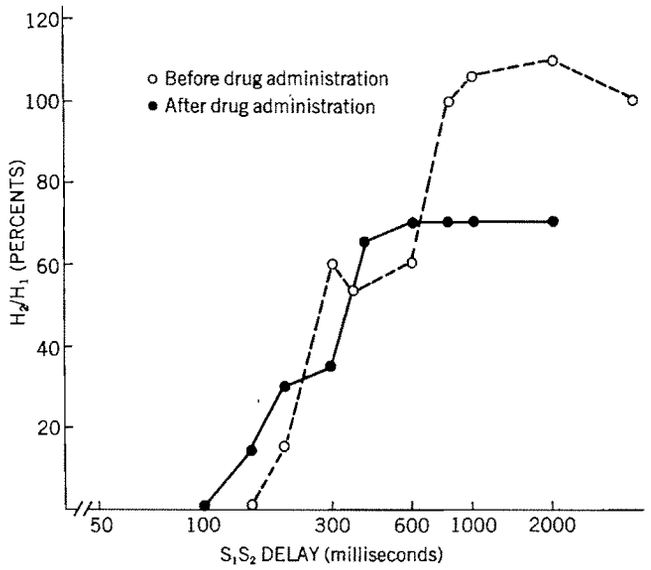
We therefore sought a relationship between H_x and dosage. Six of 16 patients with H_x values greater than 120 (38%), including the 2 patients with very high H_x scores, were receiving less than 250 mg of chlorpromazine or equivalent per day, while only 2 of 7 patients with H_x values greater than 120 (29%) were receiving more than 250 mg of chlorpromazine or equivalent per day. Although not statistically significant, these data may suggest a trend toward lower H_x with increasing dosage in these patients.

To test the hypothesis that tardive dyskinesia is a disorder of receptor supersensitivity to dopamine, selected patients were given infusions of the dopamine agonists amphetamine and apomorphine. The patient whose H-reflex recovery curve is shown in figure 5 had been admitted to the unit with a presumptive diagnosis of tardive dyskinesia, but she had little in the way of objective signs of the disorder. During the infusion of amphetamine her excitability curve shifted markedly downward, from 747 to 166 (see figure 5), and she developed marked signs of tardive dyskinesia. In this case the patient had a markedly abnormal recovery curve in the absence of clinical signs of tardive dyskinesia. Dopamine receptor stimulation induced florid TD signs, while at the same time motoneuron excitability as measured by the H-reflex was reduced.

Dopamine receptor stimulation with apomorphine in the patient whose recovery curve is shown in figure 6 also produced a reduction in the value for H_x. Before the infusion this patient had a value for H_x of 110%, but after infusion it dropped to 76%. There was no appreciable change in this patient's clinical TD scores during the infusion.

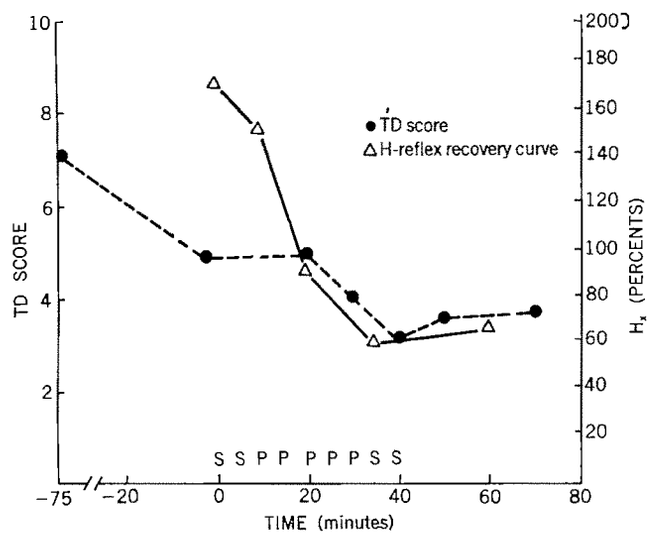
The contribution of cholinergic mechanisms to TD and H-reflex measurements was assessed by studying patients before and during administration of physostigmine. The patient whose recovery curve is shown in figure 7 showed a small drop in TD scores during physostigmine infusion. This patient's H_x showed a marked drop during the infusion that approximately paralleled the drop in TD scores. The same patient was also studied after treatment with 900 mg of deanol a day for 2 weeks. There was little change in the recovery cycle following deanol treatment. Similarly, there was little change in the patient's TD scores during that time. Deanol in this subject appeared to have little effect on either spinal reflex excitability or on the TD score.

FIGURE 6
Effect of Infusion of 4 mg of I.V. Apomorphine on the H-Reflex Recovery Curve of a Schizophrenic Patient with TD*



*In this patient there was a reduction in the curve values starting at about 600 milliseconds; note that the entire curve is shifted to the right in this patient; the pre-drug-administration H_x occurs at about 1000 milliseconds instead of at the normal 100–300 milliseconds.

FIGURE 7
Effect of Physostigmine Infusion on the H-Reflex Recovery Curve and TD Score of a Schizophrenic Patient with TD*



*S=saline infusion; P=physostigmine infusion.

DISCUSSION

Previous attempts to treat tardive dyskinesia have relied primarily on a clinical description of the dyskinesia and certain hypotheses about the underlying pathophysiology of tardive dyskinesias. The present study was undertaken to determine if a delineation of neuromuscular pathophysiology might define specific abnormalities in the disorder and electrophysiologically characterize certain groups of patients who might be specifically responsive to certain medications. Our data indicate that there is considerable heterogeneity of the electrophysiological findings among the patients diagnosed as having tardive dyskinesia. In some patients we failed to elicit an H-reflex. Others showed alpha motoneuron excitability alterations, with a tendency for extremely high values of H_x.

The absence of an H-reflex in 9 of 31 patients, all but 1 of whom had high TD scores, was an unexpected finding. In more than 60 normal control subjects and more than 250 schizophrenic patients without TD we have yet to find a subject in whom an H-reflex could not be elicited. An absent H-reflex may occur for several reasons. First, it may be an indication of a peripheral neuropathy. Ioku and associates (20) found that a significant percentage of patients with peripheral neuropathy had extremely small or absent H-reflexes. We found no clinical evidence for peripheral neuropathies in these patients, but in previous studies we have shown that schizophrenic patients have an increased incidence of degenerative changes in motoneurons (21, 22) and that they have slowed conduction velocities. According to a paper being prepared by Goode and associates, schizophrenic patients also have reduced numbers of distal motor units.

However, another possible explanation for an absent H-reflex is that the reflex is suppressed by excessive inhibition via spinal or supraspinal mechanisms (17). Further studies of various inputs to the spinal monosynaptic reflex, such as gamma motoneuron activity and the activity of Renshaw circuits, may clarify the role of these inhibitory mechanisms in tardive dyskinesia.

A third possibility to be considered in explaining the absent H-reflexes is that the absence is related to the dyskinetic movements themselves and not to some basic underlying pathophysiological process. It is known that movement alters motoneuron excitability (23, 24). However, there are several lines of evidence that would argue against this formulation. First, those patients studied repeatedly at times when they had high and low TD scores did not show any tendency to afford a correlation between the amount of movement and the H-reflex elicited. Second, chronic schizophrenic patients with considerable hyperactivity have H-reflexes. These include patients with so-called hyperponesis, or baseline electromyographic activity in muscle in the absence of apparent movement (25). Third, the Jendrassik maneuver, which involves brisk movement of the upper extremities and is a means of enhancing spinal reflex activity, did not potentiate H-reflexes in those patients who had no H-reflex. Similarly, since Jendrassik-maneuver potentiation of spinal reflexes is based on upper extremity movement, one might expect that spontaneous movement in TD patients would potentiate the reflex response and could not be an explanation for the absence of an H-reflex.

The major finding in our examination of the H-reflex recovery curve was a relationship between severity of TD and the value of the facilitatory peak H_x. When compared with normal volunteers or chronic schizophrenic control subjects, patients with high TD scores had significantly higher values for H_x. Arguments similar to those outlined above have suggested that the increased values for H_x are not simply related to an increase in alpha motoneuron excitability.

In patients with Parkinson's disease there is an increase in the value for H_x that tends to return toward normal in patients treated either by thalamic surgery or by L-dopa (26). Some patients with chronic schizophrenia tend to have low values for H_x, and these low values can be reversed with phenothiazine medication (7). These data, taken together, suggest that the facilitatory peak H_x is at least partially under the control of a dopaminergic influence, with the height of the peak inversely related to dopaminergic tone. This formulation is supported by results with amphetamines and apomorphine, both of which tended to reduce H_x in the patients studied.

However, these data are not easily compatible with the hypothesis that tardive dyskinesia is due to supersensitivity of dopamine systems in the basal ganglia. If H_x reflects the activity of dopamine systems, then low values of H_x would be associated with high TD scores. Our findings were just the opposite. It is possible that an additional neuronal influence mediated by other neurotransmitters, such as acetylcholine or γ-aminobutyric acid (GABA), are implicated in TD pathophysiology. Physostigmine infusion in the patient whose recovery curve is shown in figure 7 tended to reduce the value for H_x in parallel with the time course of reduction of the TD score.

It is apparent from these studies that there is considerable heterogeneity both clinically and neurophysiologically in tardive dyskinesia. It seems likely that a number of different abnormalities of neurotransmitter systems, including dopaminergic, cholinergic, and others, are involved in its development. Our studies have described certain neurophysiological characteristics of subjects with TD and the effects of dopamine and acetylcholine agonists on these factors. However, there is good evidence that each neurotransmitter has a variety of functions in the CNS. Dopamine acts as both an excitatory and an inhibitory transmitter in the basal ganglia, according to microiontophoretic studies (27), and acetylcholine appears to exert both an excitatory and an inhibitory effect at functionally different receptors in the brain (28). Therefore, it should not be surprising if the administration of a drug that increases dopamine blockade, for example, would have a variety of physiological effects based on blocking both dopamine excitation and inhibition.

No simple view of dopamine activity in tardive dyskinesia is compatible with the various electrophysiological and clinical findings. Further studies of TD patients may delineate certain subpopulations of patients who respond selectively to one or more pharmacological agents and contribute to the early identification of patients at risk for the development of serious neurological impairment as a result of psychotropic drug use.

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The Relationship Between Parkinsonism and Tardive Dyskinesia

BY JES GERLACH, M.D.

The author analyzes parkinsonism and hyperkinesia in psychiatric patients with tardive dyskinesia before and during treatment with α-methyl-p-tyrosine (AMPT, a dopamine antagonist), biperiden (an acetylcholine antagonist), and baclofen (a GABA agonist); and in patients with paralysis agitans and L-dopa-induced hyperkinesia. AMPT and baclofen had similar influences on oral dyskinesia, resulting in reduced frequency, unchanged or slightly reduced amplitude, and increased duration of each movement. The author concludes that: 1) reduced dopaminergic activity may be the primary pathogenetic background for tardive dyskinesia; 2) dopaminergic hypersensitivity and/or cholinergic hypofunction is necessary before hyperkinesia breaks through; and 3) the neurotoxic effects of neuroleptics may be associated with age-dependent changes in nigrostriatal regions representing oral innervation.

IT HAS OFTEN been stated that the literature on neuroleptic-induced neurological side effects lacks precision and uniformity of description and classification (1–3). This apparent difficulty can be explained; although the neuroleptic-induced disturbances of movement can be roughly classified as acute dystonia, parkinsonism, and tardive dyskinesia (4), in reality they are far from being separate, well-defined syndromes. There is a considerable amount of both clinical and pathogenetic overlap. In an attempt to shed light on this problem we studied the relationship between parkinsonism and hyperkinesia in 1) neuroleptic-treated patients with tardive dyskinesia before and during treatment with α-methyl-p-tyrosine (AMPT, a dopamine antagonist), biperiden (Akineton, an acetylcholine antagonist), or baclofen (Lioresal, a γ-aminobutyric acid [GABA] agonist), and 2) patients with paralysis agitans and L-dopa-induced hyperkinesia.

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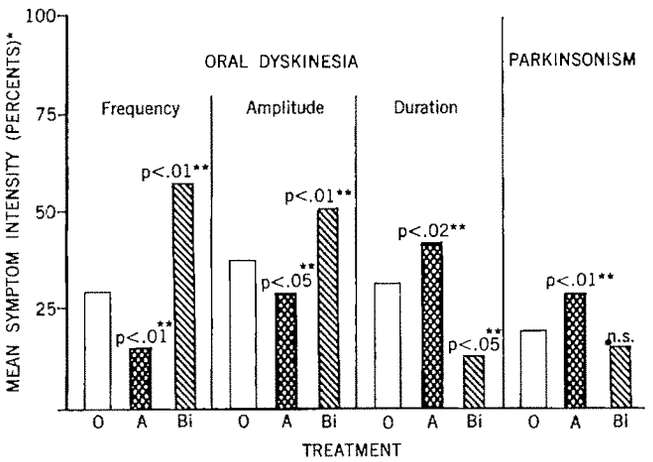
TARDIVE DYSKINESIA DURING ANTIDOPAMINERGIC AND ANTICHOLINERGIC TREATMENT

Tardive dyskinesia is mainly localized in the oral area as the so-called bucco-linguo-masticatory syndrome (2, 5, 6). These oral dyskinesias may be described with respect to the following movement elements: frequency (or degree of hypermotility when frequency cannot be measured), amplitude, and duration of each separate tongue protrusion and/or mouth opening (7). The relationship among these elements differs from patient to patient. At times the tongue protrusions are rapid and rhythmic (representing the real hyperkinetic movements of tardive dyskinesia) and at times they are slow, prolonged and almost continuous (more related to parkinsonism).

We studied these movement elements and the degree of parkinsonism in 24 psychiatric patients with neuroleptic-induced tardive dyskinesia (7). During the whole study the patients continued with their previous psychopharmacological treatment. Four g/day of AMPT (an inhibitor of catecholamine synthesis) was given for 3 days, and 12 mg/day of biperiden (an acetylcholine blocker) was given for 3 weeks. The study was carried out with crossover and a blind evaluation of the neurological condition with the help of videotape.

As shown in figure 1, AMPT significantly reduced the frequency (or the hypermotility), whereas biperi-

FIGURE 1
Oral Dyskinesia and Parkinsonism in 24 Patients Before Treatment (O) and During Treatment with AMPT (A) and Biperiden (Bi).



*Mean percentage of the maximum possible score on the rating scale used (7).
**By Wilcoxon test for paired differences.

den increased the hypermotility. The effect of AMPT on the amplitude varied; in 9 cases the amplitude was decreased, in 13 cases it was unchanged, and in 2 cases it was increased. The reduction in amplitude occurred in patients who showed increased parkinsonism following AMPT; the increase occurred in 2 young patients who showed no signs of parkinsonism following AMPT. Biperiden significantly increased the amplitude of oral tardive dyskinesia. The duration of each tongue protrusion or mouth opening was increased by AMPT in 6 cases; in the other cases duration was unchanged. In some cases the total protrusion time was decreased because of a decreased number of protrusions. Biperiden, as would be expected from its effect on motility, significantly reduced the duration of each protrusion.

Sixteen of the 24 patients had shown obvious signs of parkinsonism during previous treatment. In 14 of these 16 patients tardive dyskinesia and parkinsonism occurred simultaneously. AMPT slightly but significantly increased the parkinsonian score, with an aggravation of all classical parkinsonian features (bradykinesia, rigidity, tremor, and salivation). Biperiden had no significant effect on parkinsonism.

This study suggests the following:

1. AMPT, primarily through an inhibition of dopamine neurotransmission, reduces the frequency of tardive dyskinesia. This corresponds to results obtained with other dopamine inhibitors (6, 8–10).

2. Anticholinergic antiparkinsonian drugs increase both the frequency and the amplitude of tardive dyskinesia (11–13).

3. AMPT leaves the amplitude unchanged or only slightly reduced; furthermore, AMPT often increases the duration of each separate tongue protrusion or mouth opening. This leads to the conclusion that tardive dyskinesia may be determined primarily by a functionally decreased activity of the dopaminergic system and that the hyperkinetic movement element does not break through until so-called dopaminergic hypersensitivity develops or until anticholinergic treatment is started (7).

This study also shows that tardive dyskinesia and parkinsonism often coexist in the same subject (see also 13, 14) and that patients under long-term treatment with neuroleptics often show signs of parkinsonism that are partly refractory to anticholinergic treatment (15). Although in some cases parkinsonian symptoms may be irreversible (16), they are less obtrusive than those of tardive dyskinesia.

TARDIVE DYSKINESIA DURING BACLOFEN TREATMENT

Traditional inhibitors of dopaminergic neurotransmission (receptor blockers and inhibitors of storage or synthesis) may suppress tardive dyskinesia but may at the same time accentuate the real pathogenetic process behind it (6, 7). New forms of treatment for this late complication of neuroleptic therapy are therefore strongly indicated.

It has recently been shown in animal experiments that GABA agonists can inhibit impulse flow in the nigrostriatal dopaminergic pathways (17, 18) and counteract neuroleptic-induced increases in dopamine turnover (19, 20). There is, therefore, a pharmacolcgical basis for supposing that GABA-stimulating drugs such as beta (*p*-chlorophenyl)-GABA (baclofen) would be able to ameliorate tardive dyskinesia.

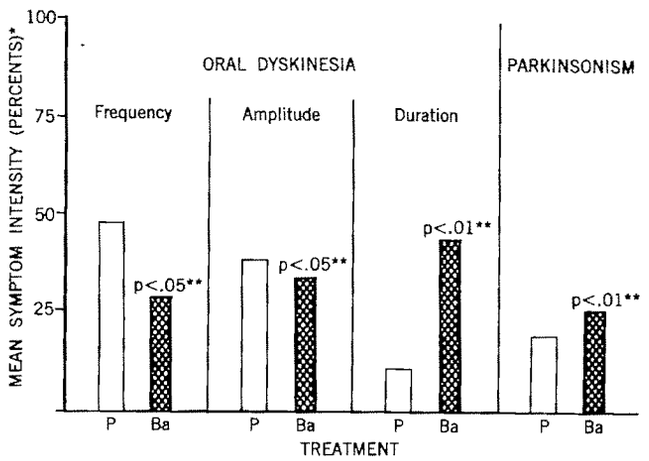
We have substantiated this supposition in a double-blind crossover clinical study. Eighteen chronic psychiatric patients aged 47–79 years (median 66) were treated in a randomized sequence with baclofen and placebo. Baclofen and placebo were each given for 3 weeks; effects were evaluated with the aid of videotape at the end of each phase.

The study showed that baclofen (given in doses of 20–120 mg/day, median=75 mg) had both antihyperkinetic and parkinsonism-eliciting effects. Figure 2 shows the average score of patients on three movement elements of oral dyskinesia and parkinsonism. Frequency was reduced in 10 of 18 patients and unchanged in 8. Amplitude was reduced in 8 patients and was unchanged in 10. The duration of each mouth opening was increased in 9 patients and unchanged in 9.

This pattern of response to baclofen corresponds quite well to that of AMPT (see figure 1) and is in accordance with the observation that, from a biochemical point of view, baclofen acts somewhat like AMPT, namely, by inhibition of dopamine neurotransmission (20).

Two-thirds of the patients in this study showed obvious signs of parkinsonism together with tardive dyskinesia. Baclofen slightly but significantly increased

FIGURE 2
Oral Dyskinesia and Parkinsonism in 18 Patients During Treatment with Baclofen (Ba) and Placebo (P).



*Mean percentage of the maximum possible score on the rating scale used (7).
**By Wilcoxon test for paired differences.

parkinsonism; bradykinesia was aggravated in 4 patients, rigidity in 2, tremor in 5, and akathisia in 5.

Baclofen, however, has more disturbing side effects. Sedation, muscular weakness, and confusion were observed in half of the patients. These side effects, which appear particularly in elderly patients, sometimes begin before the antihyperkinetic effect, thus limiting the drug's usefulness.

Therefore, it must be concluded that baclofen has more theoretical than practical therapeutic interest. Further research efforts must concentrate on finding by animal experimentation other GABA agonists without these side effects (i.e., with a more selective action on the GABA receptors of the basal ganglia) and clarifying on the clinical level whether GABA agonists may potentiate the antipsychotic effect of receptor blocking neuroleptics (see 20) and thereby make it possible to reduce the dose of these neuroleptics, as has been done by using AMPT (21, 22).

PARALYSIS AGITANS AND L-DOPA-INDUCED HYPERKINESIA

It is well known that the choreoathetoid movement disturbances often seen in parkinsonian patients treated with L-dopa are of the same type as those seen in neuroleptic-induced tardive dyskinesia (23, 24). The only difference is that tardive dyskinesia is primarily limited to the oral area, whereas L-dopa hyperkinesia usually has a more general distribution. An examination of the localization and intensity of L-dopa hyperkinesia and parkinsonian symptoms shows an independent course of development of oral and extraoral hyperkinesia that suggests the need to distinguish between dyskinesia in the two regions (25).

Oral hyperkinesia shows an increasing rate of occurrence and intensity with age, independent of the degree of parkinsonism and extraoral hyperkinesia. This suggests that age-related phenomena facilitate the development of oral hyperkinesia. The occurrence in older subjects of spontaneous hyperkinesia (senile dyskinesia) involving primarily the oral region (26) supports this view. Pathological studies have demonstrated the presence of a characteristic somatotopic organization of the substantia nigra, the striatum, and their internuclear connections (27, 28). Thus, when neuroleptic-induced tardive dyskinesia involves the oral region almost exclusively, it is reasonable to presume that the possibly irreversible neurotoxic effect of neuroleptics consists of an accelerated progression of age-related changes in the oral somatotopic area of the basal ganglia.

When hyperkinesia of the extremities first appears and parkinsonian symptoms disappear, the hyperkinesia is positively correlated with the severity of the parkinsonism in the respective extremities. This holds both for the patient population as a whole, in which hyperkinesia of the extremities is most pronounced among younger patients, most disabled by parkinsonism, and for the individual patient, in whom hyperkinesia is most pronounced in those body segments most affected by the disease.

This supports the hypothesis that a primary dopaminergic deficiency lies behind hyperkinesia and suggests a causal relationship between parkinsonism and tardive dyskinesia. The biochemical and neurohistological nature of this relationship, including an explanation of the fact that not all parkinsonian patients treated with L-dopa or all neuroleptic-treated psychiatric patients develop hyperkinesia, is not yet fully clarified. Particular problems in this area include the biochemical processes underlying postsynaptic hypersensitivity (especially the importance of the dopamine-sensitive adenyl cyclase), the implications of the presence of two distinct types of dopaminergic receptors in the neostriatum, and the involvement of other neurotransmitters (including GABA).

CONCLUSIONS

The pathophysiology of involuntary hyperkinesia in neuroleptic-treated psychiatric patients and in L-dopa-treated parkinsonian patients appears to consist of a primary dopamine deficiency (pharmacological or organic) and a secondary relative hyperactivity in the dopaminergic system (dopaminergic hypersensitivity) that may be functionally identical to a hypoactivity in the cholinergic system. The GABA system is also involved in the regulation of motor functions, and the GABA analogue baclofen influences dyskinesia in the same way as α-methyl-p-tyrosine, an inhibitor of dopamine synthesis. Baclofen, however, is not suitable for treatment of old psychiatric patients with tardive dyskinesia because of its tendency to induce sedation, muscular weakness, and confusion. Possible morpho-

logical changes of irreversible tardive dyskinesia are still unknown, but it is reasonable to assume that the neurotoxic effect of neuroleptics is related to age-dependent changes in the oral somatotopic area of the basal ganglia.

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Paradoxical Response to Dopamine Agonists in Tardive Dyskinesia

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The authors conducted an extensive pharmacological analysis of a patient severely affected by tardive dyskinesia. No drug treatment gave lasting clinical improvement. Several agents recently recommended for this condition, dimethyl aminoethanol, clozapine, and thioridazine, failed to modify the dyskinesia. Reserpine caused a worsening of the symptoms. A paradoxical and unexpected improvement was observed with apomorphine injections and with low-dosage oral L-dopa. These two drugs may have acted by stimulating presynaptic inhibitory dopamine receptors.

THE NEUROCHEMICAL lesion in tardive dyskinesia is not well understood, and it has not been established that all patients with the disorder have the same neurotransmitter defect(s) or will respond to the same drugs. A heterogeneity of disease mechanisms has been suggested by some authors who use the highly variable responses of patients to drug treatments as their major line of evidence (1). Consequently, there is a need for extensive use of many drugs in individual patients, both to find agents to control the abnormal movements and to provide a pharmacological profile of the disorder. Since the movements are especially variable in patients with mild forms of tardive dyskinesia, severely affected subjects are the most suitable for long-term studies. This report describes the treatment of one such patient, who took 19 different drugs over a period of 2 years. Some of the drugs were used in attempts to control the abnormal movements. After several unsuccessful clinical trials other drugs were given experimentally so that their effects on the dyskinesia could be observed and more rational treatments could then be provided.

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CASE REPORT

Ms. A was 36 years old when she was admitted to the Neuropsychiatric Institute, University of Michigan Medical Center, in February 1973. The only relevant incident in her medical history was a poorly documented episode of "subclinical poliomyelitis" at age 15. Her psychiatric illness began at age 27, when she became psychotic and was treated with ECT. The next year she became psychotic again and received neuroleptic drugs. Between the ages of 28 and 36 she was hospitalized at least 12 times and received continuous outpatient treatment in the intervals. The details of her drug treatments during this 8-year period are not known completely. There is documentation, however, that at various times she received 800 mg/day of thioridazine, 40 mg/day of trifluoperazine, and 400 mg/day of chlorpromazine, each for several months. At age 32 the first symptoms of her dyskinesia were noted in the form of frequent eye blinking and oral movements. From age 32 to age 36 she received intensive neuroleptic drug treatment, and her dyskinesia progressively worsened. For 4 months before her admission to our unit she was receiving the following drugs simultaneously: 400 mg/day of chlorpromazine, 40 mg/day of trifluoperazine, 48 mg/day of perphenazine, and 10 mg/day of benzhexol.

At the time of admission she was not psychotic. Her main psychological complaints were severe anxiety about her deteriorating marriage and distress at her abnormal movements. A careful drug history was not obtained at this time, and the patient was diagnosed as having a hysterical tic, based on the observations that her movements became more severe when she was anxious or under stress and on psychodynamic interpretations of her personal history. During her first 6 months of hospitalization she was treated in psychotherapy; haloperidol was given intermittently when her symptoms became disabling. During this period two important observations were made: 1) control of movements could be achieved temporarily with haloperidol, but they later returned even though the drug was continued; and 2) the abrupt discontinuation of haloperidol was sometimes followed by the disappearance of the movements for several days. In August 1973 a new resident and a new consultant saw the patient. Her drug history was obtained and the diagnosis of tardive dyskinesia was made.

The movement disorder involved the patient's oral-facial muscles, axial muscles (neck and trunk), and both upper and lower limbs. Frequent blinking and spontaneous blepharospasm were present; grotesque facial grimacing occurred to-

gether with rolling tongue movements and lateral movements of the mandible. These oral-facial-lingual movements were complicated by bruxism, leading to the breaking of several teeth and to occasional tongue-biting. Poor oral hygiene, drooling of saliva, and excoriation of her skin around the mouth and chin were present. When her oral movements were very severe, she was unable to eat solid food because of dysphagia. As a result of inadequate food intake and continuous dyskinetic movements she lost weight (from 50 kg to 38.6 kg) and appeared emaciated. Other features of her dyskinesia included ballistic head movements, spasm of her cervical muscles, truncal flexion, opisthotonos, abdominal rigidity, and choreoathetoid movements of her arms and legs. The overall clinical picture resembled an advanced stage of Huntington's chorea (2). When Ms. A became anxious or psychotic, the movements were exaggerated; they always disappeared during sleep. The movements could be suppressed also by competing voluntary activity; e.g., all the facial movements disappeared during forced protrusion of the tongue. Ms. A's neurological status was otherwise unremarkable except for mild rigidity of all limbs and symmetrically decreased limb reflexes. Further neurological evaluation, including skull X-ray, EEG, radioisotopic brain scan, pneumoencephalogram, and lumbar puncture yielded normal results. There was no clinical evidence to support the diagnoses of Huntington's chorea, Wilson's disease, or dystonia musculorum deformans.

DRUG TREATMENTS AND RESULTS

Group 1: Miscellaneous Sedatives

Ms. A did not improve with any of the following treatments: 90 mg/day of phenobarbital for 21 days, 400 mg/day of amylobarbital for 11 days, or 100 mg/day of diphenhydramine for 10 days. The two barbiturates produced only a decrease of anxiety symptoms without a major decrease in the dyskinetic movements. The effect of the antihistaminic drug was similar and did not suggest that central nervous system (CNS) histamine mechanisms might be implicated in the disorder. The β-adrenergic receptor blocking drug propranolol, which is effective in some extrapyramidal disorders such as familial tremor and lithium-induced tremor (3, 4), did not improve the dyskinesia when given in a dose of 160 mg/day for 8 days. The antidepressant drug desipramine, given for 11 days, did not improve or worsen the movements but did precipitate psychotic behavior. The minor tranquilizers diazepam and chlorazepate, given for 14 and 21 days, respectively, had no definite effect on the movements.

Group 2: Cholinergic and Anticholinergic Agents

Physostigmine salicylate was given intravenously in a dose of 1 mg 10 minutes after peripheral anticholinergic blockade with 2 mg of scopolamine methyl bromide intravenously. Within 10 minutes the patient developed severe rigidity and increased movements. This reaction was terminated by 2 mg of intravenous atropine sulfate.

Dimethylaminoethanol was given because of reported success in treating some patients with tardive dyskinesia (5, 6). No clear improvement in the dyskinesia was obtained with a dose of up to 1500 mg/day over 19 days. Serious peripheral cholinergic side effects occurred in the form of rhinorrhea, excessive salivation, and bronchorrhea (7).

Benztropine, an anticholinergic drug, was given in doses of 4–6 mg/day over 2 weeks. This drug caused a doubtful mild improvement in the dyskinesia together with a mild organic brain syndrome that cleared when the drug was stopped. When given in conjunction with haloperidol, benztropine did not improve the duration of control of the dyskinesia observed with haloperidol alone.

Group 3: Functional Antagonists of Dopamine

Reserpine was given because of previous reports that it and tetrabenazine were effective treatments in some patients (8, 9). At a dose of 2 mg/day for 8 days reserpine caused a marked increase of movements and severe rigidity developed along with hypotension and bradycardia.

Thioridazine at a dose of 400 mg/day for 20 days caused no improvement in the symptoms. This drug was used because of reports that tardive dyskinesia rarely occurs in patients receiving thioridazine (10).

Clozapine has been reported as dramatically effective in controlling some cases of tardive dyskinesia (11). When given at a dose of up to 1000 mg/day for 18 days, this new neuroleptic did not alter the symptoms.

Haloperidol treatment in doses of 20 mg/day produced good control of the dyskinetic symptoms on several occasions. A mild rigidity and parkinsonian facial appearance developed as the abnormal movements abated. This control persisted for 4 to 6 weeks, after which the movements returned at full intensity, despite continuation of the haloperidol. When the dose was increased to 30 or 40 mg/day, the movements were controlled again but for only 1–2 weeks before the dyskinesia reappeared. When haloperidol was then discontinued, the movements stopped rapidly within 48 hours, remained absent for 10–14 days, and then reappeared over a 2- to 3-day period. This sequence of events was documented on three separate occasions; it does not appear to have been described previously in other patients with tardive dyskinesia.

Group 4: Dopaminergic Agonists

Amantadine at 300 mg/day caused no improvement or worsening of the dyskinesia when given for 6 days.

d-Amphetamine was given once only in an oral dose of 10 mg. It caused an increase of activity and hostility together with a worsening of the dyskinesia.

L-Dopa was given in gradually increasing doses over a period of 21 days. Ratings of the dyskinesia were obtained daily by the ward staff using the Visual Analog (10-cm line) Scale of Global Severity (12). Figure 1 shows that doses between 2 and 4 g/day reduced the severity of the movements. The mean rating for 12 days when the dose was 0–2 g was 67.6 (SD=17.4) com-

FIGURE 1
Ratings of Dyskinesia Severity During Treatment with L-Dopa

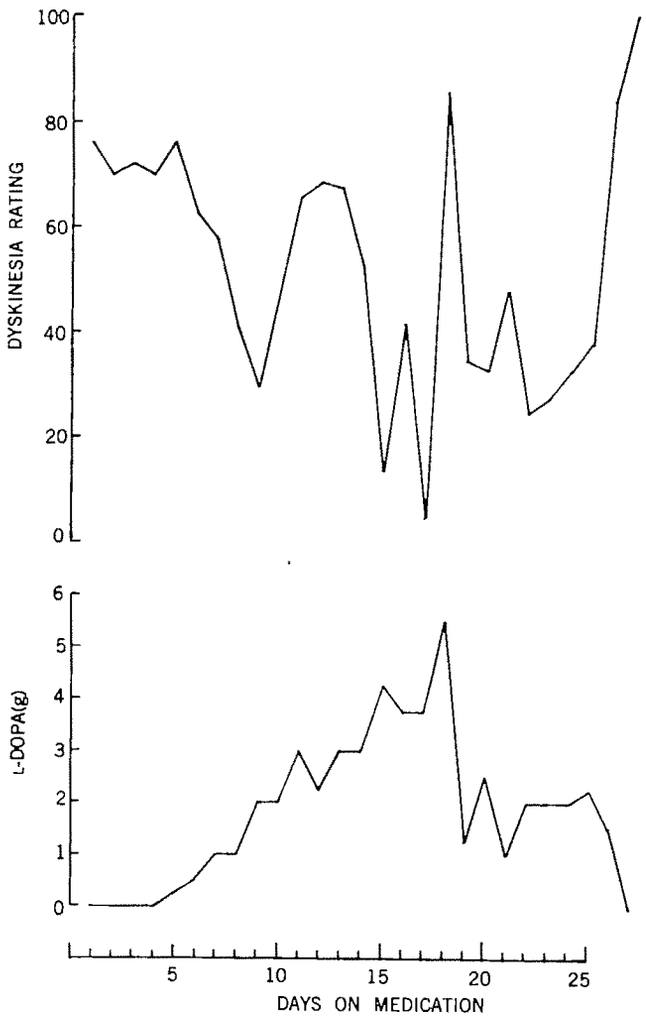
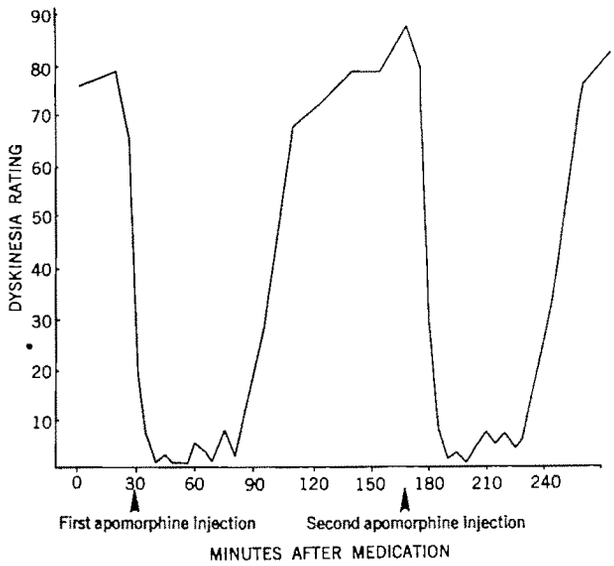


FIGURE 2
Ratings of Dyskinesia Severity After Two Injections of 4 mg of Apomorphine HCl



pared with 42.5 (SD=19.8) for 12 days at 2–4 g ($p<.01$). During this trial of L-dopa the movements worsened at a dose of 5.5 g given for 1 day, then returned to low levels at 2–3 g/day. On the final 2 days, the dose was reduced to 1.5 g and then to zero, and the dyskinesia returned at maximum intensity. During another trial with high dosage L-dopa the patient developed writhing choreiform movements and psychotic behavior.

Apomorphine, a dopamine receptor agonist, was given to determine the possible role of dopamine receptor supersensitivity in causing the dyskinesia. We expected that apomorphine would worsen the movement disorder. A dramatic and paradoxical improvement of the dyskinesia rather than a worsening followed the administration of apomorphine. In doses from 2 to 6 mg subcutaneously the drug caused a marked reduction in the movements within 10 minutes. On many occasions the movements disappeared almost completely, remained absent for up to 60 minutes, and then returned again at full intensity over a 10- to 15-minute period. Figure 2 shows this response to two injections of apomorphine. Placebo injections did not modify the movements. This effect of apomorphine was observed repeatedly. As this was the only effective treatment besides haloperidol, the patient received 2–6 mg of apomorphine subcutaneously every 2–6 hours for 2–4 weeks. Each injection was followed by suppression of the movements as shown in figure 2. After 5–7 days, however, a loss of effectiveness was noted, and responses to the drug became progressively briefer and less complete. Over these prolonged treatment periods tolerance to the emetic effect of apomorphine developed within 48 hours or less whereas the motor suppression persisted for much longer. This effect of apomorphine was antagonized by the concurrent administration of haloperidol. With prolonged apomorphine treatment the patient became dysphoric and her suicidal ideation increased.

The severe dyskinesia that occurred with high-dosage L-dopa treatment was terminated within 10 minutes by 6 mg of apomorphine subcutaneously, as described by Duby and associates (13) in parkinsonian patients.

DISCUSSION

The diagnosis of tardive dyskinesia in this patient was supported by the history of high-dosage intake of neuroleptics, by the clinical features of the movement disorder, and by the neurological evaluation, which excluded Huntington's chorea and other possible differential diagnoses. The patient's responses to drugs cannot be used at present to make the diagnosis, in view of the many negative reports associated with all of the recently proposed treatments for this disorder.

A consistent finding in this patient was the lack of

meaningful response to placebo and to nonspecific sedatives (barbiturates and benzodiazepines). Similarly, other psychotropic agents (diphenhydramine, propranolol, and desipramine) did not affect the movements.

The responses to drugs affecting acetylcholine mechanisms were investigated in view of the hypothesis that tardive dyskinesia is caused by dopamine (DA) receptor hypersensitivity, with cholinergic hypofunction in the striatum (14). In this patient, in contrast to some reports (14, 15) but in agreement with others (16, 17), physostigmine aggravated the movements and precipitated severe rigidity. Atropine reversed this acute effect of physostigmine, but benztropine given chronically did not have any definite action against the movements. That the alleged precursor of acetylcholine, dimethylaminoethanol, was ineffective is in contrast to some reports (5, 6, 18, 19) but in agreement with others (20–22). The worsening of movements and the precipitation of rigidity by reserpine was similar to the acute effect of physostigmine. It should be remembered that reserpine increases functional cholinergic activity, in addition to depleting the storage of dopamine, norepinephrine, and serotonin (23). These observations do not suggest that cholinergic hypofunction was related to the dyskinesia in this patient; conversely, they tend to indicate that cholinergic hyperfunction was present, since physostigmine and reserpine exaggerated the movements and rigidity as they do in Parkinson's disease (24, 25).

The neuroleptic drug thioridazine has strong anticholinergic activity in addition to its blockade of DA receptors (26). Clozapine, a new neuroleptic, also has strong anticholinergic properties (26). Tardive dyskinesia is said to occur rarely, if at all, with these drugs; clozapine in particular may relieve dyskinetic symptoms in some patients (10, 11, 27). Neither drug was effective in this case over periods of 2–3 weeks.

Haloperidol, on the other hand, was very effective in controlling the movements, at least for several weeks before "escape" occurred. This breakthrough phenomenon has been described previously with neuroleptics in tardive dyskinesia (28), and it has been speculated that changes in postsynaptic DA receptor sensitivity could be responsible for the effect (29). The sudden decrease in symptoms seen in this case when haloperidol was stopped cannot be explained easily, although there is no doubt about the clinical observations, which were made on three separate occasions. The definite effect of haloperidol provides good evidence for a role of activated DA receptors in the dyskinesia.

The DA precursor L-dopa had a biphasic action on the dyskinesia. At moderate doses of 2–4 g/day some definite improvement was noted. At higher doses, however, the dyskinesia was aggravated and other dyskinetic movements appeared as described in parkinsonian subjects (30). Adverse behavioral side effects of L-dopa occurred as well and were similar to those described in parkinsonism (31). This biphasic response pattern to L-dopa has been observed also in studies of animal motor activity (32).

The unexpected inhibitory effect of apomorphine on the dyskinesia suggests that DA mechanisms were involved in this case. Since apomorphine is a direct agonist of postsynaptic DA receptors (33), a worsening of the movements was anticipated. The antagonism of apomorphine by haloperidol tends to confirm the fact that apomorphine acted by stimulation of dopaminergic receptors. Recent pharmacological evidence for presynaptic receptors that inhibit synaptic DA release has been described, and it is possible that apomorphine caused its effect through activation of these presynaptic inhibitory DA receptors (34). Tolosa (35) has reported a similar inhibitory effect of apomorphine in Huntington's chorea and tardive dyskinesia. We have also observed inhibitory effects of apomorphine on the abnormal movements in patients with Huntington's chorea, spontaneous facial dyskinesia, and Gilles de la Tourette's disease (36). The antagonism of L-dopa-induced dyskinesia observed in this patient and in parkinsonian subjects (13) also supports a possible presynaptic site of action for apomorphine. In addition, the biphasic motor activity response of rats to L-dopa (32) is very similar to the biphasic effect of apomorphine (37); thus, the partial suppression of dyskinesia found in this patient with low doses of L-dopa (figure 1) could also be mediated through presynaptic inhibitory DA receptors. Gerlach and associates (17) observed a worsening of tardive dyskinesia with 1.2 g/day of L-dopa plus a peripheral decarboxylase inhibitor, but they did not report any improvement with lower doses.

SUMMARY AND CONCLUSIONS

The pharmacological studies in this patient support the concept of DA receptor activation in tardive dyskinesia (improvement with haloperidol, worsening with high doses of L-dopa, and paradoxical "presynaptic" improvement with low doses of L-dopa and with apomorphine). The mild improvement seen with benztropine and the worsening caused by physostigmine suggest that this patient could fit the "hypercholinergic hyperkinetic syndrome" described by Gerlach and associates (17). Our experience with this patient confirms the view of Granacher and associates (38) that " 'tardive dyskinesia' is a complex syndrome, and [it is] very likely that a variety of pathophysiologic mechanisms will be demonstrated. Systematic pharmacologic characterization of tardive dyskinesia in large numbers of patients remains to be done" (p. 926).

Although this patient experienced no definite benefit from any of the drugs tested, the striking temporary improvement caused by apomorphine (figure 2) suggests that other long-acting agents such as piribedil (39) may be clinically useful. The effectiveness of apomorphine that we observed was similar to that reported in two cases of dystonia musculorum defor-

mans (40) and in acute dyskinetic-dystonic reactions to haloperidol (41). Further study of agents that act presynaptically to improve tardive dyskinesia, such as apomorphine and α-methyl-*p*-tyrosine (17), is needed.

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Relationship Between Age and Tricyclic Antidepressant Plasma Levels

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Older depressed patients treated with imipramine or amitriptyline developed higher steady-state plasma levels of imipramine, desipramine, and amitriptyline. In imipramine-treated patients this finding was associated with a decreased rate of drug elimination from plasma. These findings provide at least a partial explanation for the increased susceptibility of the older patient to tricyclic antidepressant side effects and also provide a pharmacological rationale for use of lower dosages in this age group.

IT IS WELL KNOWN that clinical response to drug treatment is often altered in the elderly. Depressed geriatric patients frequently exhibit such toxic responses to tricyclic antidepressants (TCA) as postural hypotension, urinary retention, tachycardia, congestive heart failure, and organic confusional mental states. The occurrence of confusional episodes is of particular importance because it can be a confounding aspect of the tendency to misdiagnose and overdiagnose dementia in depressed geriatric patients. The fact that the elderly have greater susceptibility to TCA cardiotoxicity is usually attributed to the increased prevalence of underlying cardiac disease in the older age groups. However, it is also possible that age-related changes in drug elimination play an important role in TCA cardiotoxicity.

It has been shown that the disappearance from plasma of drugs as diverse as phenylbutazone (1), antipyrine (2, 3), penicillin (4), barbiturates (5), and propranolol (6) is slowed with age. Aside from benzodiazepines (7, 8), the effects of aging on psychotropic drug metabolism have received little attention; we have been unable to find reports of a relationship of TCA metabolism to age.

In this paper we report a study of age and TCA metabolism in patients treated with imipramine or amitriptyline. Our findings that older patients tend to have higher plasma levels and long plasma disappearance half-lives of the administered drug and its demethylated metabolite have practical implications for the treatment of depressed geriatric patients.

METHOD

Plasma TCA levels were determined in patient samples from two separate studies. The patients in the amitriptyline series were participants in a double-blind 6-week outpatient trial, and the imipramine patients were predominantly inpatients treated in a general hospital psychiatric service. Patients with serious underlying medical illness or alcoholism were excluded. The age and sex distributions of patients in the two series differed because the amitriptyline patients received treatment in a university-based depression research clinic while the imipramine patients were treated at a Veterans Administration hospital. The 35 amitriptyline patients ranged from 21 to 68 years of age, and the 23 imipramine patients ranged from 27 to

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78 years. All patients participating in these studies gave informed consent according to protocols approved by the human experimentation committees of the university and the hospital.

In the amitriptyline study, following a 1-week drug-free interval amitriptyline administration was begun at a dose of 25 mg three times a day for the first 5 days and then increased to the final dosage of 50 mg three times a day. Blood was drawn at 2-week intervals and a plasma sample frozen for subsequent determination of amitriptyline and its metabolite, nortriptyline. Only patients who received 150 mg of amitriptyline a day for the final 4 weeks of the protocol were included. Inspection of the data obtained at the end of 4 and 6 weeks of treatment from these patients shows consistent amitriptyline and nortriptyline levels for each patient, indicating that patient compliance was excellent and that a steady-state level of amitriptyline and nortriptyline had been achieved.

The imipramine patients were participants in an investigation of TCA pharmacokinetics with the principal objective of assessing the relationship of imipramine elimination and desipramine disappearance half-lives to age. Most of the patients were treated with 150 mg of imipramine a day for a minimum of 21 days so that steady-state conditions were achieved (confirmed by multiple plasma drug determinations before the plasma disappearance half-life studies of imipramine and desipramine were undertaken). Previous psychotropic medications were discontinued over the first week of imipramine treatment. Chloral hydrate as a hypnotic agent and aspirin and acetominophen as analgesic agents were permitted if necessary. The plasma disappearance half-lives were computed from at least four determinations over a 48–72-hour period following discontinuance of chronic therapy.

The plasma TCA levels were determined by gas chromatography using a nitrogen detector as described by Cooper and associates (9). This method has a coefficient of variation of ±5% across the range of 4–250 ng/ml.

Imipramine elimination half-life and desipramine disappearance half-life were calculated using a standard program for fitting a logarithmic decay curve in linear form. It must be recognized that levels of desipramine in plasma obtained following the administration of imipramine are a function of metabolic generation of desipramine from imipramine and disappearance of desipramine from plasma. Therefore, the metabolic and excretory elimination of desipramine as we have studied it differs from a standard elimination half-life, and we refer to the disappearances as the imipramine *elimination* half-life and the desipramine *disappearance* half-life in recognition of the distinction.

RESULTS

Steady-state plasma levels of the two parent compounds, imipramine and amitriptyline, both showed significant positive correlations with age (see table 1). In the case of the demethylated metabolites, only the desipramine steady-state plasma levels showed a positive correlation with age; the nortriptyline steady-state levels did not. In the imipramine series the group of patients over 65 years of age (who were all nonsmokers) had significantly higher imipramine and desipramine mean steady-state levels than a group of younger nonsmoking patients (see table 2). Smokers were eliminated from this imipramine comparison because of preliminary findings that imipramine and desipramine steady-state levels may be affected by smoking (10).

The age differences in imipramine and desipramine levels occurred despite the fact that all of the younger patients were receiving 150 mg of imipramine a day but the dosage of the older group ranged from 50 to 150 mg a day (mean daily dosage of 92 mg). Dosage modification was dictated by patient tolerance to side effects (2 patients over the age of 70 experienced substantial postural hypotension, necessitating a reduction in their imipramine dosage to 50 mg a day). In the amitriptyline-treated outpatients the mean levels of only the parent compound were significantly higher in the elderly patients (see table 2).

TABLE 1
Correlations of Steady-State Plasma Levels and Plasma Disappearance Half-Lives of TCA with Age

Item	Correlation Coefficient (r)*	
	Plasma Level Versus Age	Plasma Half-Life Versus Age
Imipramine	+.54**	+.15
Desipramine	+.30**	+.62***
Amitriptyline	+.42**	—
Nortriptyline	+.01	—

*The Pearson correlation coefficients (r) were computed from samples of 18 patients treated with 150 mg of imipramine a day and 35 patients treated with 150 mg of amitriptyline a day.
**p<.05.
***p<.01.

TABLE 2
Steady-State Plasma TCA Levels of Young Versus Old Patients

Item	Mean Plasma TCA Level (ng/ml)*			
	Young Patients		Patients Over 65 Years of Age	
	Mean	SEM	Mean	SEM
Imipramine	32.3	6.7	83.8	20.9**
Desipramine	21.2	4.5	57.2	22.2**
Amitriptyline	81.7	18.2	138.7	24.1***
Nortriptyline	122.8	35.9	122.8	16.5

*All of the patients in these two age groups were nonsmokers. The old versus young comparisons are based on 5 patients in each age group treated with imipramine and 6 patients in each age group treated with amitriptyline.
**p<.05.
***p<.01.

TABLE 3
Plasma Disappearance Half-Lives of Imipramine and Desipramine of Young Versus Old Patients

Item	Mean Plasma Half-Life (hours)*			
	Young Patients		Patients Over 65 Years of Age	
	Mean	SEM	Mean	SEM
Imipramine	19.0	3.6	23.8	1.5
Desipramine	34.2	2.7	75.8	18.2**

*Nonsmoking patients treated with imipramine in each group.
**$p<.05$.

Analysis of the plasma disappearance data for imipramine and desipramine is presented in tables 1 and 3. The disappearance half-life of desipramine is strongly correlated with patient age, while imipramine half-life correlates positively but not significantly (see table 1). It can be seen that the desipramine disappearance half-life of the over-65 age group is more than twice that of the younger group. The mean imipramine elimination half-life is only moderately longer in the elderly patients (see table 3).

Since dosage on a mg/kg body weight basis could influence plasma TCA levels, the relationship of patient weight to age was examined. No correlation of body weight with age was found for either the amitriptyline or the imipramine group. This, together with the fact that some of the elderly patients received lower imipramine dosages, excludes the possibility that the age-related differences in steady-state levels are attributable to an increased dosage (mg/kg) with age.

DISCUSSION

The cardiotoxicity of the TCAs is a current cause of concern because of the potential for lethal tachyarrhythmias and rare sudden deaths. The more common side effects attributable to their anticholinergic properties also constitute important clinical problems, especially in the elderly. Although the susceptibility of the older individual to undesirable effects of tricyclics has generally been regarded as a nonspecific intolerance of the aged to pharmacological agents, the evidence presented here would suggest that decreased biotransformation with resultant excessively higher plasma and tissue levels provides a rationale for employing lower doses of TCAs in the elderly.

Initially, TCAs were regarded as exceedingly safe drugs, with such relatively benign side effects as dry mouth, tachycardia, and occasional urinary retention in the elderly male. These compounds are concentrated in highly perfused organs (including the heart [11, 12]), and clinical reports have described cardiac arrhythmias and, rarely, sudden death in patients undergoing TCA treatment (13). The 2-hydroxy metabolites of imipramine and desipramine have been shown to produce significant cardiotoxicity in the dog at doses and tissue concentrations commonly attained in clinical use (14). It would appear that all TCAs have the capacity to produce dose-related changes in cardiac function and ECG. The dose-related ECG changes include prolongation of the PR and QRS intervals, nonspecific T wave changes, and clinical tachyarrhythmias, the most serious of which are ventricular premature contractions, heart block, and ventricular fibrillation (15, 16). The fact that a national conference was held in 1975 to examine the existing evidence on TCA cardiotoxicity, to review current FDA-approved therapeutic indications, and to revise dosage recommendations for these drugs (17) underscores the importance of establishing more precise guidelines for their use.

Only recently has it been recognized that the rate of metabolism of some psychoactive drugs decreases with advancing age. In particular, the pharmacokinetics of the benzodiazepines and barbiturates are influenced by age (5, 7, 8). This may be due primarily to a gradual decrease in specific activities of drug metabolizing enzymes in the liver as well as a possible loss of liver mass in relation to body size. Thus the older the patient, the greater the tendency to develop higher plasma and tissue levels at a given dose. Higher TCA blood levels may partially explain the increased incidence of overall side effects noted in older patients, as well as the apparently greater susceptibility of the aged to cardiac arrhythmias with TCA therapy.

In addition to these well-documented cardiovascular toxicities, adverse effects of TCAs in the CNS, with behavioral manifestations, have been described; these are often more difficult to recognize clinically. For example, Davies and associates (18) reported confusion in 13% of all patients receiving TCAs and noted that confusional episodes occurred in 35% of their patients over 40 years of age. It seems plausible that the higher incidence of this side effect among the older patients can be explained at least in part by the pharmacokinetic changes associated with aging that we observed in this study. There is a tendency among many clinicians to prematurely reject the diagnosis of depression and discontinue antidepressant treatment when organic signs appear in elderly patients. Our data suggest that downward adjustment of TCA dosage would frequently be the correct therapeutic decision in such a situation.

In the average patient, steady-state drug levels of TCAs will not be achieved for 5 to 10 days because of the prolonged elimination half-lives (averaging from 20 to 35 hours in the general patient population). Since 3 of our patients (all of whom were over 70) had desipramine disappearance half-lives greater than 80 hours, application of "5 times rule" (96% of steady-state is reached by 5 times the elimination half-life [19]) indicates that many such patients will not achieve a steady-state plasma TCA level for 2 or 3 weeks. Furthermore, at the usual therapeutic doses such patients would develop very high plasma and tissue drug levels.

Our data do not yet permit quantification and definition of toxic plasma levels for individual patients, but the results nevertheless have overall implications for dosage strategies in older patients. It seems prudent to reduce TCA dosage from that usually recommended by approximately a third to a half. Thus imipramine and amitriptyline dosage should have a maximum limit of 100 mg a day for most patients 65 years or older. For example, imipramine should be started at 25 mg daily at bedtime, with progressive increments every 4–7 days until 100 mg a day is reached or troublesome side effects intervene, whichever occurs first. As mentioned, because of the longer mean half-life in the elderly patient, steady-state levels may not be achieved for up to 2 or 3 weeks in the occasional patient (half-life more than 60 days). Therefore, more frequent and more intensive monitoring of the elderly patient is indicated during initial treatment. Because the lag period before onset of clinical antidepressant effect may include the time to reach steady-state, the standard 3-week period for completion of an adequate therapeutic trial must be extended and sometimes doubled in some depressed geriatric patients.

It is of interest that in the amitriptyline-treated patients nortriptyline plasma levels showed no age relationship (see tables 1 and 2). Although this finding will require further investigation, it is possible that clinical treatment of depressed patients over a wide age range with nortriptyline might be less complicated than with the other TCAs.

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Anhedonia and Schizophrenia

BY MARTIN HARROW, PH.D., ROY R. GRINKER, SR., M.D., PHILIP S. HOLZMAN, PH.D., AND LAWRENCE KAYTON, M.D.

The authors administered semi-structured interviews to 187 psychiatric inpatients to determine the role of anhedonia in schizophrenia. The interviews were tape-recorded and then given blind ratings for anhedonia on a 7-point scale. Schizophrenic patients had significantly more anhedonia than nonschizophrenics, although many of the latter group had anhedonic tendencies. Most of the difference between the groups resulted from high anhedonia scores for chronic schizophrenics; less anhedonia was found in the acute schizophrenic patients. The data indicate that anhedonia is not necessary or unique to schizophrenia but is a prominent factor in chronic schizophrenia.

OBSERVATIONS have been made by Rado (1), Rado and Daniels (2), and Meehl (3, 4) about a marked defect in pleasure capacity, sometimes referred to as anhedonia, in schizophrenia. These views are in accord with some recent biochemical theories of schizophrenia that involve formulations about excess dopamine and the limbic system (5). Thus formulations that posit diminished experience of pleasure as a factor in the pathogenesis of schizophrenia are rooted in both clinical observation and biological inference.

In Meehl's view (3, 6), mechanisms by which anhedonia could influence the clinical picture in schizophrenia involve features such as a general weakening of positive or pleasurable emotions, an emphasis on negative components of ambivalence, and exaggerated interpersonal fears. These could lead to interpersonal distortions, cognitive slippage, and autism.

Statements about anhedonia often emphasize the lack of pleasure in interpersonal situations. Anhedonia is usually viewed as a long-term, quasi-stable, or trait-like feature present since childhood. Thus the schizophrenic is seen as an individual who from childhood on has found little reward in interpersonal situations and little reinforcement for much of the behavior for which others are routinely reinforced. The hypothesized deficiency could then be responsible for such features in the later schizophrenic as isolation from other people and withdrawal from social situations and withdrawal from reality.

It has not been established, however, that anhedonia is present in all schizophrenics. Although interpersonal aversiveness and withdrawal from social contacts are prominent in some adolescents who later become schizophrenic, there is no evidence that they are a necessary condition for schizophrenia. Indeed, a number of theorists and investigators have reported on schizophrenics who lack histories of a shy, withdrawn adolescence (7–11).

Another question concerns the specificity of anhedonia to schizophrenia. Is anhedonia also present in disturbed nonschizophrenic patients, and perhaps in normal people as well? Analysis of a sample of disturbed nonschizophrenic subjects would help to answer this and other questions.

A related issue is whether anhedonia may be characteristic of only specific types of schizophrenic pathology (e.g., of young chronic schizophrenics but not of young acute schizophrenic patients). If this is the case, anhedonia may be associated with the course of deterioration in schizophrenia for the select group of schizophrenic individuals who go downhill drastically and later become chronically ill and deteriorated (12).

The empirical literature in this area is very scant to date. We have attempted, during the course of research on schizophrenia, to investigate formulations related to anhedonia (13). A previous report from this project by Grinker (14) noted that anhedonia and depression are separate components in schizophrenia. Kayton and Koh (15), from the same project, used experimental tasks involving pleasant and unpleasant words and found evidence that schizophrenics as a group may have a decreased tendency to use features of pleasantness in their mnemonic organization.

In a series of studies, Watson (16–18) attempted to develop measures of anhedonia and to use them in an investigation of the relationship between anhedonia and physiological variables (16, 17). His results sug-

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gested that anhedonia is not a function of hyporeactivity or learning efficiency (17, 18).

Thus despite the importance of anhedonia in several modern theories of schizophrenia, there is a paucity of systematic research in this area. The present investigation is an attempt to determine whether anhedonia really does play a prominent role in schizophrenia. In particular, we will provide data on whether anhedonia is 1) a prominent characteristic of schizophrenia, 2) present in all schizophrenic patients, 3) found in nonschizophrenic patients, 4) a necessary condition in specific select types (or subdiagnoses) of schizophrenia, and 5) related to other characteristics of schizophrenia or to other clinical or personality variables.

METHOD

Sample

The subjects were 187 relatively young recently admitted psychiatric inpatients from the Michael Reese Hospital (91 males, 96 females). The sample included 128 schizophrenic and 59 nonschizophrenic patients. Five major schizophrenic groups were studied in detail: acute schizophrenics, N=19; paranoid schizophrenics, N=27; chronic schizophrenics N=58 (relatively young schizophrenics with long-standing psychopathology, slow hospital improvement, and other features of chronicity); schizo-affectives, N=11; and acute-chronic schizophrenics, N=13 (moderately well-functioning chronic schizophrenics with a current hospitalization for a sharp, acute break). The nonschizophrenic patients included depressives, N=26; individuals with severe personality disorders, N=16; and patients from varied other diagnostic groups, N=17.

The mean age of the patients was 22.43 years, with a range of 14–34 years. There was no significant age difference between the schizophrenic and nonschizophrenic patients. Almost all of the schizophrenic patients were on phenothiazine or phenothiazine-like medication (e.g., haloperidol). The patients were predominantly from Hollingshead-Redlich (19) social classes I, II, and III.

Procedure

Semi-structured interviews were conducted as early as possible during hospitalization to obtain information on anhedonia, symptom status, social and instrumental functioning, family life, and other aspects of psychopathology and personality. Since the research focused on anhedonia, the interview included a detailed investigation of "fun" and pleasurable experiences in childhood and more recently. The interviews, conducted by experienced psychiatrists who had no prior knowledge of the patients' history or diagnosis, were tape-recorded for later assessment. The 7 items on the scale were as follows:

1. Pleasure in accordance with external events.
2. Occasional but rare pleasure in accordance with external events.
3. Occasional pleasure but occurs discordant with external events.
4. Lack of humor.
5. No convincing report of ever having fun.
6. No joy, exuberance, or knowledge of shared happiness.
7. Constant sadness or boredom.

Ratings for anhedonia (and other clinical variables) were made by consensus of the research team after listening "blind" to the tape recordings. The ratings for anhedonia were made on the 7-point scale. Interrater reliability on anhedonia for two trained raters was .77, with no significant differences between their mean ratings ($t=0.30$, $df=21$, $p>.50$). The scale for anhedonia is part of the Schizophrenia Trait Inventory, a series of rating scales used to assess key clinical dimensions that may be related to schizophrenia (13, 20).

RESULTS

Differences on the Anhedonia Scale

Table 1 presents the data on the mean scores for each of the major diagnostic groups. As can be seen in the table, patients of all types showed anhedonic tendencies; 40% or more of the patients from each major diagnostic group had ratings in the range that indicates anhedonic tendencies. The combined schizophrenic group differed significantly from the nonschizophrenic group on anhedonia ($p<.01$). However, some schizophrenic subgroups showed large differences and other schizophrenic groups did not differ significantly from the nonschizophrenic group. For example, the two acute schizophrenic groups (acute schizophrenics and schizoaffectives), who in general had a better premorbid history, did not differ significantly from the nonschizophrenics. However, the chronic and paranoid schizophrenic groups showed significantly more anhedonia than the nonschizophrenic patients. The paranoid schizophrenics were primarily young, chronic paranoid schizophrenics. Thus the two groups were primarily chronic schizophrenics under the age of 30 years.

There were no significant differences on anhedonia between the two major nonschizophrenic groups (depression and personality disorder). The relatively high anhedonia scores of some nonschizophrenics suggest that anhedonia is not unique to schizophrenia; it is common among many types of psychiatrically disturbed patients.

Relationship to Other Clinical and Personality Characteristics

Table 2 presents the relationship between the patients' ratings for anhedonia and variables from the other major areas studied. These results provide fur-

TABLE 1
Mean Anhedonia Scores for the Major Diagnostic Groups

Group	N	Mean	SD
Nonschizophrenics	59	3.66	2.17
All Schizophrenics	128	4.73*	2.09
Schizo-affective	11	4.64	1.69
Acute	19	4.11	2.31
Acute-chronic	13	4.15	2.41
Chronic	58	4.93*	2.06
Paranoid	27	5.07*	2.00

*Significantly different from nonschizophrenics ($p < .01$, t test).

TABLE 2
Correlations Between Anhedonia and Variables Representing Key Areas

Variable	Correlation with Anhedonia
Confusion	.24*
Thought regulation	.35*
Perceptual regulation	.24*
Depression	.19*
Sleep disturbance	−.08
Appetite disturbance	.06
Suicide attempts	.10
Marital status	−.14**
Age	−.02
Premorbid work performance	.20*
Premorbid school performance	.21*
Adjustment prior to onset	.14**
Affection-intimacy	.33*
Drug history	.02
Loneliness	.34*
Prior hospitalization	.14**
Lack of improvement	.23*
Slow onset	.16**
Early onset	.15
Sexual experiences	.12
Sexual satisfaction	.05
Dependency	−.06
Organization	.38*
Identity problems	.19*

*$p < .01$.
**$p < .05$.

ther clues as to the nature of anhedonia and its relationship to schizophrenia.

Anhedonia and depression. A critical issue is whether anhedonia is merely a concomitant of depression: our data suggest that there is a low but significant relationship between anhedonia and ratings of depression ($r=.19$, $p<.01$). This association suggests that depression accounts for a small percentage of the variance of anhedonia. In fact, no single variable we investigated accounts for a major percentage of the variance associated with anhedonia. Rather, several different types of variables show some relationship to anhedonia; these relationships are described below.

Symptom status. The clinical data (the first three items in table 2) suggest that anhedonic patients were more symptomatic ($p<.01$). The greater symptomatic distress of anhedonic patients in certain areas may have influenced the overall ratings for anhedonia slightly for these patients, although it is unlikely that the increased symptomatology alone can explain the pattern of significant relationships between anhedonia and variables in other nonclinical areas.

Chronicity and social functioning. The pattern of correlations indicates that the anhedonic patients were more chronically ill and had greater premorbid social difficulty than those without anhedonia. These data are consistent with the results reported earlier, which suggest that anhedonia is more prominent in chronic schizophrenics, even those who are relatively young. Anhedonia was related to early onset of disorder ($p<.02$) and to lack of improvement during the early period of the current hospitalization ($p<.001$). In the social area, the anhedonic patients tended to be unmarried ($p<.03$), lonely ($p<.001$), less affectionate toward others ($p<.001$), and had had few or no sexual experiences ($p<.06$). None of these variables accounted for a large percentage of the variance associated with anhedonia, but there was a consistent relationship between anhedonia and variables associated with chronicity and social isolation.

Prehospital competence. Another area that had a low but definite relationship to anhedonia is prehospital competence. The anhedonic patients tended to have had poorer functioning and less coping capacity before hospitalization than nonanhedonic patients. They had poorer overall organization ($p<.001$) and more identity problems ($p<.006$). Their adjustment prior to the overt onset of their disorder was poorer ($p<.05$), as were their premorbid school and work performance ($p<.002$). These variables round out a picture of less competence in the anhedonic patients.

DISCUSSION

Anhedonia in Schizophrenic and Nonschizophrenic Disorders

In previous reports based on smaller samples and with less systematic samples of control patients other investigators and members of our group have noted evidence of anhedonia in many schizophrenic patients. The results of this study, which show high levels of anhedonia in certain groups of schizophrenics, are only partly in accord with these reports. Our evidence indicates that 1) anhedonia is not characteristic of all types of schizophrenics, 2) anhedonia is also found in many disturbed nonschizophrenics, and 3) anhedonic characteristics seem to be present in mild to moderate and at times moderate to severe degrees in a variety of patients. These results raise questions about generalizations that have been made in the past concerning the relationship between anhedonia and schizophrenia.

Anhedonia may be a concomitant of other features (e.g., a certain type of chronic depressive outlook on life) that accompany a variety of different types of dis-

turbances. The data suggest that it is not present in all psychopathological conditions.

The results do not support Meehl's hypothesis that anhedonia is an inevitable feature of schizophrenia. However, the role of anhedonia in schizophrenia still must be taken into account, since it was more frequent in the schizophrenic group as a whole and there was a strong trend for specific schizophrenic subgroups to show anhedonia. These results suggest that anhedonia may play some part in the overall picture of schizophrenia and may be a key factor in chronic schizophrenia. Hence a modified and more modest view of the Meehl hypothesis of anhedonia centered around certain specific subgroups of schizophrenics is still feasible.

Anhedonia in Chronic Schizophrenia

The prominence of anhedonia in chronic schizophrenic patients can be viewed in several ways. One possibility is that anhedonia is a consequence of chronicity or of deterioration; thus schizophrenics who have become "chronic" begin to appear pleasureless during the course of their disorder. However, the evidence suggests that while chronicity could play some role, it is not enough to account for all of the relationship between anhedonia and chronic schizophrenia. Many patients' histories indicate the presence of anhedonia in childhood, before the onset of overt schizophrenic symptoms.

A second possibility is that anhedonia is a condition of dementia praecox, i.e., "true" or chronic schizophrenia. Anhedonia was extremely common in our more chronic schizophrenic subjects. In addition, anhedonia was more frequent in schizophrenics who were unmarried and had poorer premorbid social functioning and in those who had poorer premorbid work and school performance. These relationships are consistent with a view (still undocumented) that anhedonia is a significant aspect of the subgroup of schizophrenia characterized by poor premorbid functioning and that the schizophrenics without anhedonic tendencies who have good premorbid characteristics are cases of schizophreniform psychosis.

A third possibility, which is consonant with our data, is that anhedonia is important at an early age and is one of the factors that influence the course of the disorder toward chronicity. This third formulation suggests that anhedonia is not a major etiological factor in schizophrenia; however, once a person becomes psychotic, the presence of anhedonia (and other associated factors) becomes important in determining the chronicity of the illness. Thus anhedonia could have a negative influence on prognosis or it could be a concomitant of other factors that negatively influence the course of the disorder.

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Behavioral Toxicity and Equivocal Suicide Associated with Chloroquine and Its Derivatives

BY MICHAEL I. GOOD, M.D., AND RICHARD I. SHADER, M.D.

Although the antimalarial agents chloroquine, hydroxychloroquine, and amodiaquine are widely used to treat a variety of medical conditions, their behavioral toxicity and lethality are not generally recognized. Therapeutic doses sometimes cause psychosis, delirium, personality change, and depression. Since moderately low overdoses of chloroquine can result in rapid death, such behavioral effects could lead to accidental or state-dependent overdosage and death.

THE INCREASING role of the psychiatrist in the general hospital (1) has paralleled an expanding awareness of medication-induced behavioral changes among nonpsychiatric patients and an increasing number of drug-related hospital admissions (2). It is now widely known that certain drugs, such as steroids and digitalis, cause psychiatric symptoms.

The behavioral toxicity and the lethality of chloroquine have received relatively little attention in the American medical and psychiatric literature. It is one of a small number of medications that can not only significantly alter mental status but also cause death from moderate overdoses. This drug thus has particular significance for the psychiatrist, internist, toxicologist, and suicidologist. Chloroquine and its derivatives are also of special interest at the present time because of their widespread use in rheumatology and the upswing in their use as antimalarials as Americans travel more readily to countries in which malaria is endemic.

BEHAVIORAL TOXICITY

Chloroquine use is associated with psychosis, personality change, depression, and delirium. At least 10 cases of chloroquine psychosis (3–10) and 1 case of "personality change" (11) have been reported in the English-language literature since 1958. In a study by Mrňa and associates (12), 6 of 21 patients receiving chloroquine (28%) developed neurotic-like disturbances (4 neurasthenic and 2 hysteriform). Psychotic symptoms reportedly have developed after total doses of 2.0 to 6.0 g of chloroquine sulfate or phosphate and after an elapsed time from initial dose ranging from 2 to 40 days (usually 4 days). These cases included patients without a history of psychiatric disturbance. The psychological changes reversed upon drug discontinuation, usually within about a week (9), but not for 8 weeks in one inconclusive case (6), and with recurrence of symptoms even after discontinuation in another case (10). Reinstitution of therapy usually failed to reproduce significant symptoms (3), but not in all cases (10). The psychosis is probably toxic and caused by the drug itself rather than by malarial infection or concurrent factors (13). The possible role of premorbid mental susceptibility or idiosyncrasy has been considered but remains undetermined (3, 5, 9). Age and sex appear to offer no predisposition. Significantly, patients had some insight into their condition in the early stages (8).

It should be noted that secondary psychiatric symptoms may mask early delirium due to chloroquine and give the false impression of a purely psychogenic illness (14). For example, Mustakallio and associates (5) reported that a 40-year-old forester treated with chloroquine for atopic dermatitis became preoccupied with a panicky fear of "going mad" and could no longer control his intentional behavior, which became characterized by various irrational and compulsive impulses, noisiness, and aggressiveness. The patient described by Kabir (9) was also irritable and abusive. Brookes's patient (8) was depressed and confused but was aware that he was acting abnormally.

Depression has been reported in relation to treatment with chloroquine as well as its congeners, hydroxychloroquine and amodiaquine. Drew (15), for example, described his experience with a variety of side effects of antimalarials used in the extended treatment of rheumatoid diseases in 65 patients (56 women and 9 men) ranging in age from 19 to 71 years (average, 47 years). Sixty-two percent of the whole group developed psychological symptoms. Of the 21 patients on chloroquine sulfate, 43% developed such symptoms, as did 29% of the 28 patients on hydroxychloroquine sulfate and 72% of the 25 patients on amodiaquine. (Some patients received more than one drug course.) Daily doses of the drugs were comparable, averaging about 150 to 200 mg. The duration of treatment averaged about 1 to 1½ years and ranged from about 2

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months or less to 3 or 4 years. Drew noted that his series was largely composed of women of menopausal age and that some symptoms could thus be related to factors other than a direct toxic effect. Nevertheless, he observed that feelings of tiredness and depression were the earliest and most common toxic symptoms. Depression ranged from mild to severe and required treatment when the drug was continued. Several patients required antidepressant drugs. Three patients consulted psychiatrists; 1 received ECT. Other symptoms observed were claustrophobia, inferiority feelings, loss of sleep from noxious dreams, suspiciousness, and loss of affection. All these aberrations rapidly disappeared with drug withdrawal. Drew made no mention of suicidal ideation, cognitive impairment, or other signs of organic brain syndrome.

A previously published case (10) reported the occurrence of suicidal thoughts and an acute panic reaction with ego disorganization in a 15-year-old boy who was receiving chloroquine phosphate for a benign skin disorder. When the drug was begun he became drowsy and unable to think clearly. On the 11th day of treatment with a total of 4.25 g of chloroquine, the drug was discontinued because the patient developed depression with suicidal thoughts, restlessness, insomnia, and anorexia. His mood subsequently improved, and chloroquine was restarted on the 18th day, but severe depression recurred, and the drug was again stopped on the 28th day. On the 33rd day he was seen as a psychiatric emergency; he had uncontrolled outbursts of crying and self-accusation, ego-alien thoughts, and loose associations. Orientation and judgment reportedly were intact. There were some exacerbations of symptoms even in the month after the chloroquine was finally discontinued, although after 40 days the patient was improved.

The psychiatric literature to our knowledge includes only one report, by Rockwell (16), of either the behavioral toxicity or the lethality of chloroquine and derivatives. Rockwell included suicide as a possible psychiatric complication of chloroquine use and cited acute psychosis and delirium and their early manifestations as signs of its behavioral toxicity. However, he did not mention depression or other less acute or less dramatic mental changes or suggest any possible relation between behavioral toxicity and suicidal behavior. Reports of chloroquine-related depression, toxic psychosis or delirium, and irrational compulsive impulses raise the possibility of a relationship between chloroquine toxicity and lethal chloroquine ingestion.

Of related interest is the Amok syndrome sometimes seen in males in Southeast Asia, Indonesia, Papua New Guinea, the Phillipines, Africa, and other tropical areas. Amok is a sudden, often psychotic disturbance that is associated with homicides and suicides and is apparently culturally bound. Although its etiology is unknown, it is interesting that infections such as malaria and "toxic" processes have been hypothesized as possible physical causes (17). Antimalarials could play a role in this tropical syndrome.

Reports of the effects of chloroquine on the EEG vary (12, 18–20). In general it appears that chloroquine acts as a cerebrocortical stimulant that increases EEG frequencies, but it also decreases voltage and may occasionally cause seizures.

LETHALITY

It is ironic that chloroquine is more lethal than its forerunner quinacrine, which is generally considered to be more toxic (21). Chloroquine generally causes poisoning within hours; toxic symptoms or death have reportedly occurred as early as 30 to 45 minutes after oral overdoses (22–24). Such deaths usually have been considered secondary to hypersensitivity, with the drug itself described as relatively safe. In fact, however, the margin of safety is narrow: lethal doses reported for chloroquine sulfate are as low as 1.0 to 1.5 g, usually less than 8 or 10 g, and most frequently 2 to 4 g (25–30). Survival is rare without prompt emergency treatment following overdosage (31–33). Despite reports of the lethality of chloroquine, most medical and toxicological textbooks and references do not mention this fact (34). Hydroxychloroquine, which was introduced in 1955 and is considered less toxic than chloroquine, can also cause fatal poisoning (35, 36).

REVIEW OF REPORTED OVERDOSES

A review of the literature on chloroquine and hydroxychloroquine ingestions yielded reports of at least 134 overdoses, more than three-quarters of which led to death (22–59). (In some instances in the remaining cases the outcome was not mentioned.) Two lethal cases involved hydroxychloroquine (35, 36). Among adult cases in which the sex of the deceased was specified there was an approximately equal number of fatalities among men and women.

Fifteen cases were apparently unintentional overdoses in children (22, 25, 26, 37, 41–43), and there is at least 1 report of a clearly accidental overdose in an adult (46). Eighteen cases, 1 of which involved hydroxychloroquine (35), were apparent abortion attempts (27, 29, 35, 45, 49). Three additional deaths resulted from normal therapeutic use (29).

Many of the remaining cases are more difficult to classify and raise questions. Some of the ingestions considered to be suicides or suicide attempts seem equivocal in retrospect (24, 25, 47, 48, 50, 52). In fact, investigations of the possibility of murder were initiated in some fatal cases but homicide was eventually ruled out. The number of early reports of deaths from chloroquine ingestions suggests the need for a concerted pursuit of an additional clue to explain the lethal outcomes. In all cases reported, however, the overdoses were ultimately described or labeled as definite suicides or parasuicides, clear accidents, or abortion attempts, or they were simply not considered fur-

ther. For example, Kiel (39) reported 13 cases, 6 of which were acknowledged to be questionable suicides. Yet, as if to resolve the mystery, he then concluded that suicide was probable in such cases without specifying other possible explanations.

It is known that therapeutic doses of certain medications can alter or impair consciousness in some patients. Aspects of consciousness susceptible to toxic effects include perception, attention, information processing, memory, and affective state, any of which in turn can influence motivation and behavior. Consequences of behaviors performed during a toxic state may be quite contrary to what an individual would have intended if free from toxic effects. This concept has been used to account for certain cases of drug overdosage in which self-destructiveness either probably was not intended at the time of overdosage or, if intended, probably would not have been carried out under drug-free conditions (60).

We have been unable to locate in the psychiatric literature any reports of apparent suicide by chloroquine ingestion, and the cases reviewed above do not report employing a "psychological autopsy" (61) or similar procedure in reviewing the deaths. Nor is there any mention, with one exception (31), of psychiatric evaluation of patients who survived chloroquine ingestions. Wilkey (25) observed that chloroquine poisoning may be unsuspected until the toxicologist's report is seen and concluded that accidental therapeutic overdosage may be underdiagnosed in some areas. The possible facilitating role of behavioral toxicity in chloroquine deaths in which the mode of death is unclear but accidental or suicidal ingestion is likely needs further evaluation.

We recommend that the mental status of patients be evaluated before administration of chloroquine and related drugs and periodically during their use and that there be more detailed investigation of the nature and cause of antimalarial overdosage.

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Social Adjustment Profiles of Female Drivers Involved in Fatal and Nonfatal Accidents

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Previous research by the authors has shown that male drivers involved in fatal and nonfatal accidents can be differentiated from the general population on the basis of background, behavior, and social adjustment. The results of the present study suggest that female drivers involved in serious crashes do not differ from the overall female driving population.

ON THE BASIS of a considerable amount of research conducted over the past 2 decades, it now seems fairly well established that certain personal and behavioral characteristics of drivers are significantly associated with traffic violations, injuries, and fatalities (1, 2). However, what may not be well known is that most of the research relating driver characteristics to traffic accidents and violations deals almost exclusively with men. In large measure this is because men are much more frequently involved in traffic accidents, especially those involving driver fatalities. (According to data from the Maryland State Police, in 1975, 85% or 304 of the 357 drivers killed in motor vehicle accidents in the state of Maryland were men.)

For the past 9 years we have been conducting in-depth studies of fatal and nonfatal traffic accidents. To date, several articles and reports derived from this continuing investigation have been published (3–5); however, most of them dealt exclusively with male drivers. One of the most prominent and consistent findings is that fatally injured, legally culpable male drivers significantly differ from a comparable normative sample on a variety of behavioral traits that may be subsumed under the general heading of social obstreperousness. The prior existence and prominence of such traits is supported by the subjects' driving records, which indicate a high incidence of convictions for moving violations, and by excessive blood alcohol levels at autopsy in many cases.

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That the above relationships currently hold true among culpable male drivers can scarcely be doubted. The question remains, however, as to the generalizability of such findings with respect to female drivers. The meager evidence available on this issue is equivocal and for the most part limited to studies of the incidence of past driving offenses rather than the occurrence of fatal or nonfatal accidents (1, 2). This article represents a preliminary effort to shed light on this important issue.

METHOD

Sample

All of the fatal cases involving female drivers were drawn from the population of driver fatalities in the Baltimore metropolitan area between October 1968 and October 1974. With relatively few exceptions, the 30 fatalities reported herein represent a virtual census of their type occurring within the temporal and geographical constraints stated. A requirement for inclusion in the study was the death of the driver within 24 hours of the accident.

Because of the relatively small number of cases available for study, we felt that the research goals could be furthered by investigating 24 potentially fatal accidents that resulted in only slight injuries to the female drivers.

Procedure

After referral of an eligible case from the Chief Medical Examiner's office, we conducted a multidisciplinary investigation into the circumstances surrounding the accident. This included physical autopsies of the victims, including determination of blood alcohol levels; inspection of the accident site and the vehicle(s) involved; inspection of police reports and Motor Vehicle Administration records; and, whenever possible, comprehensive psychological interviews (both structured and unstructured) with relatives and friends of the victim.

This article deals primarily with the results of the Katz Adjustment Scales (R-form) (KAS) (6). This instrument consists of 205 scaled items that permit a retrospective, quantitative description, by an informant, of the subject's individual and social behavior. The KAS provides scores on 18 cluster-analytically derived dimensions pertaining to psychiatric symptoms as well as social activities. The recent availability of normative data (7) obtained from a systematic random sample of male and female residents of a nearby Maryland county has greatly increased this instrument's general utility.

In each case the KAS was completed by an informant (usually a spouse, parent, sibling, or other close relative) who had been in close contact with the subject during the weeks and months prior to her fatal, or potentially fatal, accident. In accordance with standard instructions, informants were asked to describe the subject as she had appeared to them during the few weeks immediately before the accident in question.

Data Analysis

All completed KAS forms were scored by computer. Raw scores on each scale were subsequently transformed to standardized T scores based on the normative data for females supplied by Hogarty and Katz (7). These T scores have a mean of 50 and a standard deviation of 10 in the normative group, and the transformation affords ready intergroup as well as intragroup comparisons.

RESULTS

Demographic and Situational Variables

Although the two groups of female drivers (surviving and fatally injured) differed significantly on only one of the demographic or background variables studied, we are reluctant to combine the data in several instances since the small samples involved preclude sensitive tests of significance.

The mean age of the dead drivers was 41.3 years ±16.9; that of the surviving drivers was 36.0 years ±17.1. The groups had a similar racial composition, with 28% being black and the rest white. Thirty-seven percent of the fatally injured drivers were either separated or divorced, compared with only 4% of the surviving drivers ($p<.05$). According to the Hollingshead and Redlich two-factor index (8), social class distribution was similar for both groups; 14% were in class V, 34% in class IV, and 52% in class III or above.

Because the previous driving records of the two groups were remarkably similar, we will present data for the sample as a whole. Unlike the driving records of the fatally injured male drivers (5), the records of the female drivers were relatively unblemished. Only 22% had had one or more previous convictions for speeding, and only 9% had had two or more. Only 4% had had their licenses suspended, and only 2% had had their licenses revoked. None of the subjects had been convicted of drunken driving, and only 24% had had one or more convictions for a moving violation of some other type. According to Motor Vehicle Administration records, 17% had had previous accidents; in this respect, the female drivers resembled the fatally injured male drivers.

Turning now to circumstances surrounding the accident itself, 38% of the surviving drivers but only 3% of the fatally injured drivers had been wearing seat belts (with or without upper body restraint) ($p<.01$). Seventy-one percent of the surviving drivers but only 43% of the fatally injured drivers were involved in single-car accidents; this difference is not statistically significant. In addition, nearly 92% of the surviving drivers and 80% of the fatally injured drivers were legally at fault.

Blood alcohol levels were determined at autopsy for the 30 fatally injured drivers. Unlike the male driver fatalities previously reported, 67% of these female

TABLE 1

Mean T Scores on the Katz Adjustment Scales (R-form) for Two Samples of Female Drivers Involved in Fatal and Nonfatal Accidents

Katz Adjustment Scales	Fatal Accidents (N=16)	Nonfatal Accidents (N=18)	Combined Sample (N=34)
Belligerence	58.0	53.1	55.4
Verbal Expansiveness	51.6	47.0	49.2
Negativism	50.4	49.3	49.8
Helplessness	49.0	49.8	49.4
Suspiciousness	49.8	48.1	48.9
Anxiety	49.9	46.7*	48.2
Withdrawal and Retardation	47.3	49.1	48.3
General Psychopathology	51.9	46.4	49.0
Nervousness	51.5	48.3	49.8
Confusion	50.7	47.2**	48.8
Bizarreness	50.1	48.8	49.4
Hyperactivity	50.3	45.2*	47.6
Stability	53.9**	53.9**	53.9*
Level of Performance of Socially Expected Activities	57.4*	57.1**	57.3*
Level of Expectations for Performance of Social Activities	55.6**	56.6*	56.2*
Level of Free-Time Activities	48.8	46.3	47.5
Dissatisfaction with Free-Time Activities	46.9	52.2	49.7
Dissatisfaction with Performance of Socially Expected Activities	45.8**	49.0	47.5

*Significantly different from normative mean of 50 ($p<.01$).
**Significantly different from normative mean of 50 ($p<.05$).

drivers had no measurable blood alcohol levels, and an additional 10% had levels less than .10 mg/100 ml, the point at which driving ability is definitely impaired (9). Thus the incidence of alcohol involvement appears to be only about half that found in a comparable series of male driver fatalities (5).

For the 24 surviving drivers, accurate determination of blood alcohol levels was much more problematical. However, on the basis of confidential interview data and police notes, it appears that no more than 17% of these accidents could be described as alcohol related.

Katz Adjustment Scales Profiles

KAS profile data were available for only 16 fatally injured drivers and 18 surviving ones; in the rest of the cases there was no reliable or sufficiently cooperative informant. However, subsequent correlational analyses suggested that the persons for whom KAS profiles were available were generally representative of their respective total groups.

Table 1 presents the mean T scores on each of the 18 KAS scales for each group separately and for both groups combined. Combination of the data in this fashion may be considered permissible on two grounds: 1) the nonfatal accidents had the *potential* for fatal injury, and 2) there were no statistically significant differences between the two groups on any of the 18 KAS scales.

Perhaps the most important feature of the summary statistics in table 1 is their essential *lack of deviancy* from the normative population in any undesirable or pathological sense. In contrast with comparable data on male drivers (5), which indicated significantly elevated mean scores on several quasi-undesirable characteristics, the present data on female drivers suggest that any deviancy present is of a positive sort, i.e., the female drivers appear, on the average, to be more emotionally stable and socially competent than the female comparison group (7). Unlike the male drivers, the present sample did not have elevated scores on the scales measuring verbal expansiveness, negativism, hyperactivity, and general psychopathology.

COMMENT

As Nunnally (10) has aptly noted, one of the truisms in psychological assessment is that men and women tend to differ in almost all respects. Despite this fact, many researchers persist in combining data on men and women. It would be interesting to know how many potentially significant findings have been overlooked through the mutual cancellation of divergent and sex-specific trends.

The more than adequate level of social adjustment that appears to characterize the female drivers as a group is in striking contrast to the somewhat undesirable traits of their typical male counterparts. While both sets of findings could be dismissed as mere artifacts of the assessment procedure or as biases associated with available norms, the implications of the profile differences are amply supported by equally striking differences in past driving records. The women's records were relatively unblemished; the men's records were not. Moreover, alcohol involvement, that tired explanation for automobile crashes that really does not explain anything (11), was much more common among the male drivers.

The implication of the present findings is that female drivers involved in fatal or other serious accidents are not particularly different from other female drivers, at least not in terms of any of the undesirable traits that seem to distinguish their male counterparts. Viewed in the aggregate, the available data on the female drivers provide no suggestion of social maladjustment, dangerous lifestyles, or poor driving habits. Indeed, there is slight evidence to the contrary. Faced with such unexpected findings, we can only conclude that the women in our samples were just unlucky—they happened to be at the wrong place at the wrong time. If such a tentative conclusion is borne out by further investigation, it could have important implications for theories of accident causation.

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This section contains reports of new clinical or laboratory observations, single cases of unusual interest or importance, descriptions of pilot studies, suggestions for future research, etc. Reports should be submitted in duplicate, typed double-spaced throughout, and may contain 1,000 words, no more than 1 table, and a maximum of 8 pertinent references. Figures cannot be used. Reports will be reviewed by at least one referee and the Editor; the author will receive a postcard informing him/her of the disposition of the submission. Reports will be edited for clarity and conformance with Journal *style.*

Platelet Monoamine Oxidase in Chronic Schizophrenia

BY WADE H. BERRETTINI, M.D., WOLFGANG H. VOGEL, PH.D., AND ROY CLOUSE, M.D.

The possibility that altered central nervous system monoamine function exists in the major psychoses has led to the investigation of platelet monoamine oxidase (MAO); it is hoped that peripheral MAO levels reflect brain levels. There appears to be mounting evidence that platelet MAO activity in chronic schizophrenics is decreased compared with that of normal individuals (1, 2). Other investigators have shown that there is no significant difference in MAO activity in acute schizophrenics (3, 4). This has led to the hypothesis that there are unknown, uncontrolled factors which are responsible for the differences seen in chronic schizophrenia; others claim that the difference may be a genetic marker for the disease (2).

Since most studies have used only one substrate concentration in their assays, which does not allow for kinetic interpretation of the enzyme, we studied MAO activity in platelets of chronic schizophrenics at various substrate concentrations to obtain information on maximum velocity (V_{max}) and K_m. These two constants provide additional information concerning enzyme differences.

Method

Subjects. Twelve chronic schizophrenic inpatients were selected for this study, 5 from Thomas Jefferson University Hospital and 7 from Albert Einstein-Southern Division Hospital. We selected patients who had been hospitalized for 2–5 weeks so that we would have an accurate record of drug intake. It was not considered necessary for patients to be drug free because the major antipsychotic drugs, such as phenothiazines and butyrophenones, do not alter MAO activity (1, 5).

The 12 patients (3 women and 9 men) had an average age of 32.1 years (range, 18–45). The average time since the initial diagnosis of schizophrenia was 8.4 years (range, 2–18). In all cases the clinical diagnosis of two psychiatrists was accepted. On admission, all patients were considered to have flat or inappropriate affect, delusions, and looseness of associations; 9 admitted to auditory hallucinations and 4 to suicidal/homicidal ideation. No patient had a history of depressive psychosis or use of antidepressant medication, and there was no evidence of iron deficiency anemia. Informed consent was obtained from all subjects.

Age- and sex-matched controls were selected from laboratory personnel and from the staff of both psychiatric inpatient units. These subjects had no personal or family psychiatric history and were not taking drugs.

Enzyme preparation. Approximately 10 cc of venous blood was drawn from a peripheral vein directly into two ethylenediaminetetraacetic acid (EDTA) treated vacutainer tubes. Platelets were isolated as follows. Whole blood was centrifuged for 30 minutes at 275 *g*. The platelet-rich plasma was separated and spun for 20 minutes at 1300 *g*. The resultant platelet plug was washed 2 times with 1 ml of Ringer's solution, suspended in 0.9 ml of Ringer's solution, and homogenized with 50 up-and-down strokes in a glass-Teflon homogenizer. These steps were carried out at 5° C. No more than 2 hours elapsed between venipuncture and platelet assay. Protein content of the homogenate was estimated by Miller's modification (6) of Lowry's method.

Assay. Using the method of Wurtman and Ax-

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The authors would like to acknowledge the assistance of Drs. R. Stohlberg, B.D. Evans, and T. Michaels, all of the Jefferson Medical College Department of Psychiatry.

TABLE 1
V_{max} and K_m Values for Platelet MAO from Chronic Schizophrenics and Controls

Group	Age	Sex	K_m(× 10^{-5}M)	V_{max} (nmol/mg protein per hour)	Years Since Diagnosis
Controls					
1	25	M	2.6	3.6	
2	25	F	3.6	3.8	
3	46	M	3.8	3.5	
4	20	M	2.8	8.2	
5	48	F	2.9	4.2	
6	29	M	2.3	3.9	
7	24	M	1.4	3.9	
8	31	M	1.8	2.7	
9	25	M	1.6	2.0	
10	54	M	0.9	1.8	
Mean (±SD)	31.7±9.6		2.4±0.9	3.7±1.7	
Chronic schizophrenics					
1	23	M	0.7	1.6	2
2	30	M	2.0	1.8	9
3	38	F	1.8	2.5	14
4	32	M	1.3	1.3	7
5	28	M	1.5	1.4	10
6	41	M	0.8	1.4	11
7	43	F	1.8	3.4	16
8	26	M	1.6	3.0	2
9	45	M	0.6	1.3	18
10	18	F	1.2	4.6	2
11	23	M	1.3	2.9	5
12	32	M	2.2	1.5	4
Mean (±SD)	32.1±8.4		1.5±0.5*	2.2±1.0**	8.4

*p<.01.
**p<.001.

elrod (7), 50 μl of the platelet homogenate were added to test tubes containing 200 μl of Sorensen's phosphate buffer (0.05 M, pH=7.4), with final concentrations of [2-^{14}C] tryptamine (New England Nuclear Corp., Boston, Mass.; specific activity, 48.5 mCi/mmol) ranging from 2.7 × 10^{-5}M to 1.3 × 10^{-4} M. Reactions occurred at 37° C and were stopped at 30 minutes by the addition of 0.5 ml of 2 N HCl. Metabolites were extracted into 6 ml of toluene and measured in 10 ml of a standard fluor in a Packard Model 3375 scintillation counter.

Results

As table 1 shows, both the mean K_m and the mean V_{max} of platelet MAO were significantly lower for chronic schizophrenics than for normal control subjects. Chronicity or severity of symptoms was not correlated with K_m or V_{max}.

Discussion

The finding of a lower V_{max} in patients with chronic schizophrenia is consistent with the reports of decreased platelet MAO activity in this disease (1, 2). The failure to find differences in studies of platelet MAO in acute schizophrenia is interesting given Rosenthal's finding of no increased incidence of schizophrenia in the families of acute schizophrenics (8). This is in direct contrast to the well-documented familial aspect of chronic schizophrenia. The difference seen in the V_{max} may be the result of a genetically determined variation in MAO.

This is, as far as we are able to determine, the first report comparing the platelet MAO K_m value in chronic schizophrenics to that in a control population. It is interesting to note that there appears to be a bimodal distribution of K_m values among the control population, while no such distribution was noted in the chronic schizophrenic patients, all of whom had a lower K_m value. These data are consistent with the hypothesis that there is a commonly occurring genetic variation of platelet MAO which is associated with chronic schizophrenia. Chronic schizophrenia may be associated with what Johnston calls an abnormal "type B" MAO (9). Evidence for this hypothesis comes from studies of platelet MAO and from analysis of MAO metabolites in the cerebrospinal fluid of chronic schizophrenics (10). Whether this association is related to schizophrenia in terms of pathogenesis must be determined in future investigations.

The data we have presented seem to indicate the existence of two distinct types of MAO—an MAO with a higher affinity for tryptamine and a generally lower maximum velocity and an MAO with a lower affinity for tryptamine and a higher maximum velocity. The first type occurs consistently in chronic schizophrenics and in one segment of the control population. One can speculate that it is this change in MAO which predisposes to chronic schizophrenia.

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Deanol in Tardive Dyskinesia

BY KENNETH L. DAVIS, M.D., PHILIP A. BERGER, M.D., AND LEO E. HOLLISTER, M.D.

Tardive dyskinesia is a syndrome of involuntary movements of the mouth and extremities that follows treatment with antipsychotic drugs. Pharmacologically induced supersensitivity of dopamine receptors may disturb the usual balance between dopamine and acetylcholine in the striatum, resulting in predominance of dopamine. Attempts to treat tardive dyskinesia by reducing dopaminergic activity have not been especially fruitful. Increasing cholinergic activity by administration of physostigmine produces a transient but definite improvement (1). In order to exploit this phenomenon in a clinically useful way, dimethylaminoethanol (deanol) was proposed as a precursor for acetylcholine, and several studies have reported beneficial effects from such treatment, usually in doses not exceeding 1500 mg/day (2–5).

We treated four patients suffering from tardive dyskinesia with deanol. Three patients had a stable buccolingual masticatory movement for 6 months to 1 year. These patients received up to 1600–2000 mg/day for 21 to 56 days, with no improvement in the frequency or intensity of their movements as compared with predrug movement frequency. Patients placed on 14 days of placebo following deanol had a movement frequency indistinguishable from the frequency during deanol administration. One patient with tardive dyskinesia of only 2 weeks' duration experienced a complete disappearance of all movements shortly after beginning treatment with deanol in a dose of 1 g/day. This remission persisted throughout a 21-day placebo period and the syndrome has not recurred to date, which indicates that the patient had a spontaneously reversible withdrawal dyskinesia. Our experience differs from the earlier more positive reports and indicates that selection of patients is critical, since cases of reversible dyskinesia might improve spontaneously. Deanol in the doses we used is not effective for well-established cases of tardive dyskinesia.

Deanol is not converted to choline in the brain, but must first be converted to choline in the liver, where it ultimately becomes phosphatidylcholine, a major constituent of brain lipids (6). Incorporated choline can be freed in the brain by hydrolysis of phosphatidylcholine, but the extent of this source of choline for synthesis of acetylcholine is undetermined (7). Choline chloride, theoretically more available for acetylcholine synthesis in the brain, has been more effective in our experience (8). If deanol were to be made equally available as a precursor, it would have to be given in much larger doses—probably 8–20 g/day. One of the patients who failed to improve when given 2 g/day of deanol for 51 days improved when placed on 20 g/day of choline chloride. Further studies will be required to compare deanol with choline as a truly effective precursor for acetylcholine in the brain.

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Response to Lithium in a Case of L-Dopa-Induced Psychosis

BY WILLIAM BRADEN, M.D.

There have been reports of psychoses induced by L-dopa in the treatment of Parkinson's disease (1). L-dopa, a precursor of dopamine, is thought to act by increasing dopamine synthesis in the central nervous system. Psychoses induced by L-dopa are thus of some theoretical interest because alterations of dopamine metabolism have been implicated in naturally occurring manic (2) and schizophrenic (3) psychoses. I would like to report a case of L-dopa-induced psychosis that apparently responded to lithium carbonate treatment.

Case Report

Mr. A, a 64-year-old retired man with Parkinson's disease, was admitted to a psychiatric hospital in December 1975 because of increasing hostility and suspiciousness. He had no history of psychiatric treatment, but the family had noted occasional suspiciousness toward business associates. Mr. A was described as a heavy drinker and a hard worker until the onset of Parkinson's disease in 1972. At that time he stopped drinking, had to leave his job, and felt bitter about the loss of his independence. He was treated with up to 8 g/day of L-dopa, with good remission of parkinsonian symptoms. However, he gradually became delusional, refusing on one occasion to start his car because, he said, "the Mafia had booby-trapped it." In April 1975 L-dopa was discontinued and he was started on a combination (Sinemet) of L-dopa and carbidopa, an inhibitor of peripheral decarboxylation thought to increase the amount of L-dopa available to the brain for conversion to dopamine. He appeared to do well initially on L-dopa/carbidopa, 750/75 mg per day, but gradually became concerned that his family was putting drugs in his food to interfere with the antiparkinsonian medication. He became openly hostile and verbally abusive toward family members. The dosage was gradually lowered to 250/25 mg per day. At this dosage the patient again had symptoms of parkinsonism and the hostility and suspiciousness recurred. He was admitted to the psychiatric hospital after striking his son.

On admission, he manifested depression and symptoms of parkinsonism. He denied delusional thinking. Initially he was treated with amitriptyline and appeared to do well on 100 mg/day. He was discharged but developed confusion and visual hallucinations and was readmitted in early February. The confusion and hallucinosis resolved when amitriptyline was withdrawn. He was somewhat angry and delusional, and his parkinsonism appeared to have worsened. The L-dopa/carbidopa dosage was increased to 500/50 mg daily and 5 mg/day of haloperidol was also prescribed. Three days later he was calm and reasonable but could not walk without falling. Haloperidol was discontinued and L-dopa/carbidopa was increased gradually to 1500/150 mg per day. One week later, he was walking well and seemed free of parkinsonian symptoms but he was restless, cheerful, talkative, and appeared manic. He soon became angry and threatening. The dosage of L-dopa/carbidopa was reduced to 1250/125 mg per day, and he was started on 1200 mg/day of lithium carbonate in late February (serum lithium level of 0.8–1.2 mEq/liter). His belligerence disappeared, his mood improved, and he was discharged. The emergence of dyskinesias led to reduction in L-dopa/carbidopa to 625/62.5 mg per day—a higher dose than he had previously tolerated without lithium.

He continued to do well until May, when he developed insomnia and suspiciousness and was readmitted. He was angry at his family because they had mentioned placement in a nursing home. An attempt was made to add chlorpromazine to his treatment, but the drug was discontinued after he developed a confusional state with benztropine. He was finally able to return home on a regimen of 1200 mg/day of lithium carbonate and 500/50 mg per day of L-dopa/carbidopa. His wife was instructed to leave the house when he behaved inappropriately (which proved to be unnecessary after the first few such events). He reportedly did well for another four months.

At that point I contacted the family, who said the patient had had some difficulties in October, apparently similar to his original problems. The neurologist had had him hospitalized and taken off all medications. He was started on amantadine, with excellent results for the next 2 weeks. He then attacked his wife with a knife and was committed to a state hospital.

Discussion

Several factors in addition to L-dopa contributed to this patient's psychopathology—his life-long suspiciousness, his perception of himself as helpless because of his neurological illness, and his wife's meek and fearful response to his outbursts, which was gratifying in that it allowed him to feel powerful. However, it is clear that the amount of manic and paranoid symptomatology was correlated with the L-dopa dosage. Although lithium did not completely eliminate his accusatory behavior, it did allow treatment with larger doses of L-dopa than he could have tolerated otherwise.

The use of lithium to control L-dopa-induced psychosis has been reported before. Lithium was effective in one patient who had manic symptoms (4). In anoth-

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er case, lithium controlled one patient's manic symptoms for only 2 weeks and was of minimal effectiveness in four patients with unclear symptomatology (5). Lithium may be useful when the dose of L-dopa cannot be reduced, especially since neuroleptics usually worsen parkinsonism symptoms.

Neuroleptics are thought to act by blocking dopamine receptors (3), so it is not surprising that they reverse both the therapeutic and toxic effects of L-dopa. In the case reported here, lithium apparently acted by a different mechanism, controlling the patient's manic and paranoid symptoms without noticeably worsening his parkinsonism (and without controlling L-dopa-induced dyskinesias). It has been suggested that lithium and L-dopa interact because each affects norepinephrine metabolism (4). Another possibility is that L-dopa induces behavioral changes by dopaminergic mechanisms, as is commonly thought, and that lithium acts at a different but functionally related brain site. This explanation is consistent with the wide overlap in clinical indications for lithium and the neuroleptics and should be considered in interpretation of research on model psychoses in animals.

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Abuse of Antiparkinsonian Drugs by Psychiatric Patients

BY KATHERINE MACVICAR, M.D.

Trihexyphenidyl (Artane) and benztropine (Cogentin) are anticholinergic drugs that are widely used in psychiatric populations to control extrapyramidal symptoms due to phenothiazines. Like atropine and scopolamine, for which they are synthetic substitutes, these drugs have the propensity to cause CNS side effects ranging from mild confusion to toxic delirium (1). Other physical signs of toxicity include the usual signs of atropinism: dilated pupils, flushed dry skin, constipation, tachycardia, elevated blood pressure, severe dryness of the mouth, and ataxia. Paranoid trends are the outstanding feature of the toxic psychosis, and disordered time sense and hallucinosis are almost ubiquitous. Trihexyphenidyl is also reported to have a euphoriant effect in some patients, which may predispose the drug to abuse (2).

However, deliberate abuse of these drugs in the United States is apparently quite rare; there are only a few reports in the literature. This has not been the case in other countries. In Britain, where trihexyphenidyl was available without prescription prior to 1964, it was young people for its hallucinogenic proper- On occasion it was also unwittingly ingested duals who believed that they were taking Poland there was a report of 20 cases of deliri- te in young people who had taken tri- nidyl for narcotic effects (4). The low incidence of abuse in this country must result in part from the fact that the drugs are available only on prescription; also, more pleasant and relatively cheap hallucinogens are readily available. Several major drug abuse clinics in the San Francisco area reported that they had not encountered patients who abused antiparkinsonian medication. However, a similar clinic elsewhere in the country documented several cases of toxic psychosis due to benztropine, one of which was a deliberate overdose by a schizophrenic patient (5).

There may, however, be another reason for the scarcity of such reports, a reason related to the nature of the population that is likely to have access to the medications. Aside from their use in the treatment of arteriosclerotic and idiopathic parkinsonism, where they have been at least partly replaced by L-dopa, these drugs are most frequently used in psychiatric populations; thus, the excitement, paranoid ideation, and hallucinosis characteristic of toxic reactions may be easily missed because these symptoms are commonplace and inherent in the primary diagnosis. In addition, the toxic symptoms generally remit within 24–48 hours after the drug is discontinued and may be gone before an adequate evaluation can be made. In fact, the patient I will describe was hospitalized with an acute toxic psychosis due to trihexyphenidyl, and the true etiology was unknown until the patient later revealed his intake of the drug. Other reasons given for the low incidence of reactions to antiparkinsonian medication are the relative youth of schizophrenic patients, the tendency of phenothiazine and antispasmodic side effects to cancel themselves out, and the

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pharmacologic resistance characteristic of schizophrenia (3).

Types of Toxic Reaction

Toxic reactions to antiparkinsonian drugs may be subdivided into 1) reactions to therapeutic dosages due to unusual sensitivity, and 2) reactions to overdoses. Toxic reactions have been reported in psychiatric patients who received the drugs in normal doses in conjunction with phenothiazines (6, 7). It has been suggested that narcotic users may be particularly sensitive to small doses of antiparkinsonian agents because narcotics diminish the rate of release of acetylcholine from nerve endings in both the peripheral and central nervous systems (5). There have been a number of reports (3, 8) of overdoses of the drugs with suicidal intent. Their use by presumably normal young people for "kicks" or for hallucinogenic purposes seems to be confined to other countries.

Misuse by Psychiatric Patients

This type of misuse has been reported by a few authors. Bolin (9) described a 32-year-old woman with marked hysterical features and masochistic preoccupations who developed a spastic torticollis, together with marked anxiety and depression. Trihexyphenidyl, 2 mg q.i.d., was effective in improving the symptom, but it also made her feel somewhat euphoric. As the stresses in her life increased, she began to take more of the drug, eventually reaching a dose of 24–30 mg/day. She then developed a toxic psychosis with marked paranoid preoccupation, confusion, and signs of atropinism. In this case, the euphoric effect led to ingestion of high doses of the drug.

Another case was reported (5) of a 24-year-old paranoid schizophrenic, a known abuser of other hallucinogenic drugs, who was taking 2 cc of I.M. fluphenazine enanthate every 2 weeks and 1 mg benztropine b.i.d. He developed a toxic psychosis after ingesting 12 benztropine tablets in order to get high. The same author cited three cases of narcotic addicts who overmedicated themselves with benztropine, precipitating toxic psychoses. These cases and the following one illustrate the potential hazards of antiparkinsonian medication in patients who have a history of drug abuse.

Case Report

The patient, a 30-year-old paranoid schizophrenic man, for 3 months had been taking 2 mg of trihexyphenidyl b.i.d. for alleged dystonic reactions to haloperidol. He did not reveal his misuse of trihexyphenidyl for some time, until he came to a point in his therapy at which he felt guilty about the misuse and wished to give it up. This patient was aware that phenothiazines tend to antagonize the effects of anticholinergic drugs and therefore refrained from taking phenothiazines the day before ingesting the trihexyphenidyl. He would take about 15 mg of trihexyphenidyl, which brought on a toxic psychosis with visual hallucinations that he found pleasurable, along with dry mouth, constipation, and nausea. On one occasion the patient took 50 mg of the drug and his behavior became so bizarre that he was picked up by the police and taken to a hospital in another area for observation. The true etiology of his psychosis was completely undetected; it was assumed to be an exacerbation of his preexisting schizophrenia. The patient reportedly introduced several of his friends to the drug, but most of them did not greatly enjoy the effects.

Aside from the euphoriant effect, a motive that he shared with the previously described patients, this man also enjoyed the feeling of being friendly and talkative that the drug gave him. It has been reported (3) that subjects feel a strong need to externalize the trihexyphenidyl experience and to communicate it to others, in contrast to responses to LSD and mescaline, which are dominated by an intense introspective preoccupation. The drug helped this patient to feel less isolated and also actively produced psychotic symptoms, which he usually experienced passively. At times the patient also used other psychedelic drugs to produce altered states of consciousness and hallucinations, presumably for similar motives. Apparently these benefits outweighed the unpleasant atropinelike effects of the drug, and he continued this pattern of use for some time.

Discussion

Deliberate abuse of antiparkinsonian medication for its euphoric and hallucinogenic effects has been reported only rarely in this country. However, there are many reports of abuse of proprietary medications containing belladonna alkaloids, which have very similar effects to the antiparkinsonian drugs. Since antiparkinsonian drugs have been abused in other countries, one must assume that the fact that they are controlled substances has prevented widespread misuse in this country. However, one should keep in mind the potential for abuse when prescribing these drugs, particularly with patients who have abused other drugs in the past.

These cases emphasize the need to exercise caution in prescribing antiparkinsonian drugs. It has been recommended (10) that they should not be prescribed prophylactically in patients taking phenothiazines but should be used only to bring extrapyramidal symptoms under control, and then in the lowest possible effective doses. Extrapyramidal symptoms rarely persist longer than 3 months; thus, patients on long-term medicati
usually do not require antiparkinsonian dru
should also have an index of suspicion in pati
keep requesting the drugs and in those with a
toward drug abuse.

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Danger of Overreliance on Computerized Cranial Tomography

BY CHARLES E. WELLS, M.D., AND GARY W. DUNCAN, M.D.

Each advance in diagnostic technology has the potential for misuse. Computerized cranial tomography (CT scan) has enormously expanded the physician's capacity for visualizing intracranial structures and is being used increasingly in the evaluation of neuropsychiatric problems (1). However, the CT scan, like all laboratory techniques, is subject to misapplication. We recently studied two patients who were misdiagnosed by highly trained physicians as having progressive dementias on the basis of relatively minor changes in the CT scan, in spite of conflicting clinical and laboratory data. We report these cases in order to 1) caution against overreliance on CT scan observations with our limited knowledge of this still-new technique, 2) highlight the danger of using an isolated laboratory finding to establish a diagnosis when confirmatory evidence is lacking, and 3) emphasize that dementia is a clinical syndrome (2), the diagnosis of which rests on the congruence of clinical and laboratory data.

Case Reports

Case 1. A 36-year-old man was admitted complaining of memory loss and confusion. He had been healthy until a motor vehicle accident 23 months earlier when his head, neck, and shoulders were injured; he was unconscious for only a few seconds. Afterward he complained of head and neck pain, but no serious damage was suspected. His performance as a self-employed plumbing contractor was unremarkable until 3 months later, when he began to concentrate poorly, made errors in estimates, and started to lose money. He gave up his business and since then has worked only intermittently at undemanding jobs.

The patient's complaints multiplied. His headaches, which were initially episodic and severe, improved, only to be replaced by disabling pains in his neck, shoulders, arms, and back. He developed memory failures, including losing his train of thought during conversations, finding himself in a room at home and not remembering how he got there or why, and forgetting to pay bills. His wife reported that his personality changed and his irritability increased. Psychological evaluation (done elsewhere) suggested organic dysfunction and CT scan was interpreted as showing mild to moderate cortical atrophy. He was diagnosed as having dementia, possibly secondary to trauma. The persistence of his disabling symptoms led to hospitalization for reevaluation.

The patient was alert and cooperative. Despite considerable pain, he attended to long interviews with interest and concentration, giving a detailed, complex history clearly and coherently and arranging details in correct temporal order. He easily recalled names of physicians, medications, and dates that marked the course of his medical care. He appeared both anxious and depressed but was unaware of these affects. Mental status examination increased his anxiety, but his performance was commensurate with his educational attainments. General and neurological examinations were normal.

Neuropsychological test data (Wechsler Adult Intelligence Scale [WAIS] grooved pegboard, Wechsler Memory Scale, memory for objects, trail making, Wisconsin card sort, Bender Visual Motor Gestalt Test, and tactual performance test) were inconsistent with organic brain dysfunction, and his estimated full scale IQ was 100. Personality data (Rorschach, Minnesota Multiphasic Personality Inventory, Thematic Apperception Test, and sentence completion) indicated inner turmoil bordering on panic, with anxiety, depression, and marked somatization.

The cerebrospinal fluid, radiographs of skull, chest, and spine, and EEG with nasopharyngeal leads were normal. CT scan was again interpreted as showing mild cortical atrophy.

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The authors wish to acknowledge the assistance of Denton Buchanan, Ph.D., who performed the psychological tests.

The mental status examination, neuropsychological testing, and other data except the CT scan failed to support the presence of organic dementia. The emphasis on cognitive dysfunction as a complaint, the communication of distress, the highlighting of failures, and memory gaps for specific periods were consistent with a pseudodementia which, with the manifest anxiety and depression, led to a diagnosis of serious posttraumatic neurosis.

Case 2. A 47-year-old man was seen for neuropsychiatric evaluation 4 weeks after an automobile accident because he and his wife would not accept the diagnosis of progressive dementia made earlier by a consulting neurosurgeon.

There was no history of neurologic or psychiatric dysfunction. The patient was a college graduate with a successful career who had functioned well in a highly skilled technical position until the automobile accident. He was not unconscious after the accident, but he complained of headache, appeared befuddled, and was hospitalized for observation. His symptoms abated, and he was discharged after 3 days. At home he experienced decreasing symptoms of headache, impaired concentration, low frustration tolerance, and unsteady gait. After 10 days he felt ready to resume work, but he experienced a severe episode of vertigo on bending that persisted for several hours. He was admitted to the neurosurgical service of another hospital, where the vertigo quickly subsided but his previous symptoms exacerbated. A build-up of high-amplitude slow activity with hyperventilation on EEG was interpreted as a "paroxysmal dysrhythmia," and "some mild cortical atrophy" was described on computerized tomography.

The patient and his wife were told that the CT scan showed brain atrophy which probably reflected a progressive dementing process, most likely predating the accident. Their unwillingness to accept the diagnosis resulted in referral for reassessment.

On admission to the hospital the patient complained of headaches, unsteady gait, fuzziness of vision, trouble concentrating, losing his train of thought, anxiety, and decreased frustration tolerance. He was alert, cooperative, anxious, and worried. He gave a clear, detailed, and accurate history, although he lost his train of thought several times. Each time he recognized this, called it to the examiner's attention, and proceeded appropriately. Although the tasks of the routine mental status examination appeared to give him trouble, his responses were accurate. The neurological examination was normal except for some unsteadiness when he turned quickly.

Skull X-rays, waking and sleeping EEG, and extensive otologic evaluation revealed no significant dysfunction. The WAIS, Wechsler Memory Scale, memory for objects, tactual performance test, Bender-Gestalt, trail making, sentence completion, MMPI, TAT, and Rorschach revealed no evidence of organic dysfunction. Full-scale IQ was 118 (verbal score=119, performance=114). There was considerable evidence of depression, anxiety, and somatization. Repeat CT scan revealed frontal convolutions slightly narrower and sulci slightly wider than usual.

The patient's symptoms were typical of those which follow mild cerebral trauma without brain damage. The symptoms had been exacerbated and prolonged by the fear that resulted from the episode of vertigo and the diagnosis of dementia. Clinical examination, behavioral observations, and psychological tests were inconsistent with dementia. He was discharged with a diagnosis of posttraumatic cerebral dysfunction with a good prognosis. Follow-up six weeks later showed resolution of most of his symptoms.

Discussion

The two patients we have described complained of symptoms that suggested dementia, but the history in each case was atypical of the usual dementing process (2), and mental status examination and behavioral observations were incongruent with dementia (3), as were ancillary diagnostic procedures except the CT scan. In other words, both patients presented clinical findings typical of pseudodementia (4–6) but were diagnosed as demented, almost solely on the basis of minimal features of the CT scan.

Computerized cranial tomography is a new and important diagnostic aid that has significantly increased our ability to measure brain size and contour safely and comfortably. However, standards of normality have not been firmly established (7), especially for varying ages, and even if they were, it would be dangerous to assume that a pattern noted on the CT scan invariably resulted in a specific clinical disorder. Fox and associates (8) observed that patients with moderate to severe dementia usually have moderate to severe brain atrophy demonstrable on CT scan. They suggested that absence of significant atrophy in dementia indicates a reversible process. We recommend that if a patient who appears demented has a CT scan revealing no more than minimal atrophy, the presence of dementia itself should be critically examined.

In the two cases we have described, the observations reported from CT scan probably represent normal variations of no pathologic significance. When the diagnosis of a clinical syndrome rests on an isolated laboratory observation, the physician should critically examine the reliability of the diagnostic procedure.

We reiterate that dementia is a clinical syndrome and the diagnosis rests on congruence among history, clinical behavioral observations, and ancillary diagnostic procedures. No single feature (history, mental status, psychological testing, EEG, or CT scan) establishes the diagnosis, and the physician who assigns preemptive diagnostic significance to a single measure is likely to err.

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EEG Sleep in a Child with Severe Depression

BY JUDITH KANE, R.N., M.S.N., PATRICIA COBLE, R.N., C. KEITH CONNERS, PH.D., AND DAVID J. KUPFER, M.D.

Depressive illness in children has become an area of increasing concern because it may severely impair an otherwise healthy child's scholastic and social adjustment (1). However, as numerous authors have pointed out, it is extremely difficult to diagnose depression in children because the clinical picture may vary depending on age and developmental level (2–4). Clinicians have long noted the prevalence of disrupted sleep as part of the symptom complex (5). EEG sleep studies in adults with depression have augmented our understanding of these depressive illnesses (6), but we are not aware of any such studies of children. We have begun to examine the EEG sleep of depressed children in cooperation with the inpatient children's psychiatric service in our clinical research setting. In this report we shall describe the EEG sleep and clinical data of a child who presented with severe depression.

Clinical History

This was the first psychiatric admission for Mary, an 11-year, 4-month-old girl who had been referred for evaluation of withdrawn behavior, increasing school difficulties over the past year, and suicidal threats over the previous several weeks. Her parents noted that she had become "very quiet and shy, answering only in whispers," her school performance had worsened, and she had fewer peer relations. At home she became exquisitely concerned about cleanliness and engaged in handwashing rituals and various phobic behaviors. On occasion she would lock herself in the bedroom or bathroom, refusing to come out or threatening to jump out the window. After an unsuccessful trial of individual psychotherapy, Mary's parents became frightened that she would carry out her suicidal threats and she was admitted to the children's psychiatric intensive care service.

Mary was a tall, slender girl with blond hair and some early breast development. Her demeanor was shy and demure. She appeared anxious, carried herself in a rigid, constricted manner, and was hypoactive in motor behavior. She was essentially nonverbal, avoiding eye contact and answering only direct questions with a nod of her head. Her affect was depressed. Her developmental history and medical history were unremarkable. Family history, as far as could be ascertained, was negative for the presence of major psychiatric disorders. Physical examination on admission, routine laboratory tests, chest X-ray, and EEG were all within normal limits. Psychological testing revealed a girl of average intelligence who did not appear to be suffering from any organic deficit or cognitive dysfunction. The predominant picture was that of an affective disorder, leading to a diagnosis of depressive reaction of childhood with suicidal ideation.

Mary was treated with both a tricyclic antidepressant and a phenothiazine (25 mg of imipramine, b.i.d. and 5 mg of trifluoperazine, h.s.). Her depressive symptomatology showed a marked improvement within 3 weeks and she was discharged on the same drug regimen.

Sleep Studies

Our techniques for studying sleep in this setting and our methods of scoring EEG sleep records have been reported elsewhere (6). Children visit the laboratory prior to their scheduled study for a "grand tour." They are shown the bedroom where they will sleep and are introduced to the laboratory staff. In addition, the nursing staff of the children's unit have been given a set of electrodes and a junction box that they routinely use to familiarize the children with the process of being "wired up" before coming to the sleep laboratory. During the actual study period, a member of the nursing staff not only accompanies the child to the sleep laboratory but stays in the area throughout the

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TABLE 1
Mean Sleep Characteristics of a Prepubertal Depressed Child

Measure	Mean Observed Values
Sleep latency (minutes)	55.5
Intermittent wakefulness (minutes)	20.5 (4.6%)
Time spent asleep (minutes)	443.5
Sleep efficiency (percent)*	82.7
Time in stages (percents)	
Stage 1	3.2
Stage 2	43.3
Stage 3	6.8
Stage 4	24.1
REM latency (minutes)**	87.0
Stage 1-REM (percent)	22.0
REM activity units***	147.0
REM density‡	1.88
Number of REM periods	3.5

*Calculated by dividing the amount of time spent asleep by the total recording period.
**Number of minutes from sleep onset until the onset of the first REM period.
***An estimation of rapid-eye-movements on a scale of 0–8/minute of REM sleep.
‡Determined by dividing the REM activity units by the REM time (minutes of REM sleep).

night. All-night EEG sleep recordings were obtained for 3 consecutive nights following the initial period of formal evaluation and at a time when Mary had been drug free for more than 14 days.

Results

The sleep pattern in this depressed child was characterized by a significant disturbance in sleep continuity. This disturbance did not appear to be restricted to any one type of arousal but was different on each of the study nights. Early morning awakening was the most prominent on night 1, difficulty falling asleep on night 2, and intermittent wakefulness on night 3. Her overall sleep efficiency (82.7% for nights 2 and 3) was significantly decreased when compared to normal values in this group, even when the first night was omitted because of the possible influence of adaptation to the laboratory.

While various measures of sleep architecture appeared to be consistent with those reported in normal controls, the relatively stable but persistently shortened REM latency was of particular interest (see table 1). Although normal values in this age group show considerable variability (137.5±34.5 minutes), the REM latency values for this child still appear to be at the lower end of that range and are significantly shorter than the mean value for this age group (7).

Discussion

In general, there is a paucity of information regarding EEG sleep of children who have a major childhood psychiatric disorder. A few studies do exist on various aspects of EEG sleep in psychotic and autistic children (8, 9), but we are aware of no reports that specifically address the sleep of children with depressive syndromes. There is much variability of normative EEG sleep values in children, but Mary's sleep did show some distinct aberrations, particularly the marked sleep continuity disturbance. The mean sleep efficiency of 82.7% for nights 2 and 3 is much lower than values reported in normal girls of this age. Also noteworthy is the abnormally short REM latency, which remained low regardless of the type or extent of sleep continuity disturbance experienced. Other REM sleep parameters, although variable, seemed to be more related to the type and severity of her sleep discontinuity.

These findings are very interesting; there are some similarities to findings in depressed adults. However, one cannot generalize from this single case study. Such findings can perhaps best serve to raise a number of relevant questions for future investigation.

1. What are the sleep characteristics of larger samples of depressed children and how do they compare with those of children in other diagnostic groups?
2. Are there any relationships between certain sleep parameters and specific symptomatology in childhood disorders?
3. Can developmental changes be distinguished from diagnostic changes in children?
4. Can certain alterations in the sleep patterns of children be used as more objective indicators of treatment strategy, particularly with regard to the efficacy of major pharmacologic intervention?

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Fluphenazine Decanoate During Pregnancy

BY MICHAEL F. CLEARY, M.D.

The introduction of long-acting phenothiazines for the treatment of psychotic patients over a decade ago (1, 2) was stimulated in part by the need to ensure consistency of medication intake and to avoid relapse and multiple hospitalizations (3).

There has been apprehension about such treatment for severely psychotic women during pregnancy. The responsibility for continuation of phenothiazine therapy rests with the clinician; manufacturers' comments in the *Physicians Desk Reference* usually indicate that the safety of the use of such drugs during pregnancy has not been established and that the possible hazards should be weighed against the potential benefits. The mechanism of the therapeutic action of these drugs is unknown. This paper describes the use of fluphenazine decanoate throughout pregnancy in a 32-year-old primiparous schizophrenic patient.

Case Report

When the patient was first seen in January 1972 she was 29 years old and had been married for 8 years to an engineer who was 5 years her senior. This was the first marriage for both and there were no children, although the patient had had a first-trimester miscarriage in 1968 after an automobile accident.

She had been in psychotherapy in another state and wished to continue. At the time of her self-referral, the patient had been taking oral phenothiazines for 5 years; her dosage then was 800 mg/day of chlorpromazine and 30 mg/day of trifluoperazine. In addition, she took 2 mg/day of trihexyphenidyl HCL. Her history included another automobile accident in 1965, in which she had suffered a concussion; after the accident she had a psychotic episode. Her first psychiatric hospitalization was at age 15. During a later hospitalization she had a course of 5 ECTs. She was one of a family of four; no one else in the immediate family had been mentally ill. The dosage of oral phenothiazines was progressively reduced during the 15 months after I began treating her but was increased when the patient began to appear irrational and expressed delusional ideas; she also became preoccupied with religious concepts and developed an interest in the occult. She managed to give up smoking but seemed to require larger doses of phenothiazines when she abstained from tobacco. She had stockpiled drugs from earlier prescriptions and once threatened to take an overdose after calling a suicide prevention center.

In October 1973, she was admitted to a private psychiatric hospital and was started on 2 cc of I.M. fluphenazine decanoate every 3 weeks because of her unreliable drug ingestion, her habit of stockpiling medication, and her suicidal tendencies. After her discharge from the hospital in November 1973, she continued to be seen in individual psychotherapy; the same dosage of fluphenazine decanoate was given until January 1975; 2 mg of benztropine mesylate b.i.d. was also prescribed but she began to take it only sporadically. She conceived in early January 1974, with an expected delivery date of October 8, 1974. The possible hazards of phenothiazine use early in pregnancy were discussed with the patient and her husband, and they concurred with the therapist's recommendation that the fluphenazine be continued. She had resumed smoking and was drinking four or five cocktails each evening. She developed occasional vomiting during the first trimester and experienced weight gain and elevated blood pressure (140/100) in the second trimester. Her cigarette consumption reached a peak of four packs daily. Latent homicidal ideas were noted late in her pregnancy, and her behavior continued to be bizarre, explosive, and unpredictable.

A healthy male infant weighing 7 pounds, 7 ounces (3.38 kg) was delivered by Caesarean section 9 days after the expected date following the failure of oxytocin to induce labor. The Apgar score of the baby was 8 after 1 minute and 10 after 5 minutes. The patient remained in the hospital for 6 days—fluphenazine decanoate (2 cc I.M.) was given 4 days postpartum. This dose was repeated 4 weeks later and the patient's regular dosage schedule (every 3 weeks) was then resumed.

Four weeks after delivery, the pediatrician thought the baby had minor extrapyramidal manifestations, as has been previously reported by Hill and associates (4). He had been breast fed for 5 days. The extrapyramidal symptoms—or withdrawal symptoms from fluphenazine—responded readily to diphenhydramine elixir. Two months after delivery, the infant was healthy and weighed 10 pounds, 4 ounces (4.66 kg). The patient developed somatic delusions, became agitated and paranoid, and was readmitted to a psychiatric hospital. After discharge from the hospital, she sought treatment from another psychiatrist and some months later left the state. She continued to write letters to her therapists that indicated the persistence of a schizophrenic thought disorder.

Subsequent communications included a phone call from the patient in June 1976 in which she said the baby, then 20 months old, was healthy. She also sent a photograph taken on his second birthday that showed an apparently well-nourished, well-developed, and alert child.

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Prompt Publication Policy

The *Journal* would like to remind authors who are particularly interested in early publication (whether to establish priority of an idea, reveal innovative results of new research, or for other pressing reasons) of its prompt publication policy. Under this policy, short (maximum of six double-spaced pages) manuscripts will be considered for publication in the Brief Communications section of the first available issue. These manuscripts must still undergo expert scrutiny before being accepted, and two months must be allowed for the printing process. However, the time required for publication of these short articles can be lessened considerably.

Authors who would like to have their manuscripts considered under this policy should indicate this desire, as well as the reasons for it, when they submit their manuscripts.

LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double-spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

On Existence of Dangerousness

SIR: The type of patient described by Victor R. Adebimpe, M.D., in "Physical Violence in the Concept of Dangerousness: A Case Report" (February 1977 issue) is familiar to all psychiatrists. For those who enjoy categorizing patients, Mr. A certainly fulfills the usual criteria of primary affective disorder, manic type. I have no reason to doubt that the description of Mr. A's behavior is accurate.

However, I have grave concerns about the attitude Dr. Adebimpe seemed to display, which reflects an apparent lack of concern with intra- or interpersonal dynamics or conflicts. The attitude also seems to betray a feeling that the doctor has more wisdom, prescience, responsibility, and authority than the patient, the judge, or the Constitution. Dr. Adebimpe apparently feels that the patient's disagreement with him was a significant indicator of illness. This animosity from the patient toward the doctor seems in some way to arouse within Dr. Adebimpe urges to protect the patient through further confinement, forced medication, and labeling him as dangerous.

Violence is frequently the result of interpersonal conflicts. The identification of one of the actors as "sick" only reinforces the behaviors of the antagonists, frequently resulting in ostracism of the "sick" person, as this case demonstrates.

If we, as a society, insist on the incarceration of individuals on the basis of presumed dangerousness, I for one would prefer to see persons locked up for driving while intoxicated or for child abuse. The whole notion that some physician can invoke a diagnosis as a basis for incarceration is frightening to me, and I think it should be frightening to anyone concerned with human liberty and freedom.

I applaud the attorney, judge, and patient in this case. The willingness of the attorney and judge to consider this patient's rights is refreshing, even if they did not, or could not, ignore the accusations of the physician.

Dr. Adebimpe mentions that a trial was held. I suspect he is referring to a commitment hearing, but his choice of the word "trial" is further evidence of the attitude that this patient was "guilty" of something. I would rather have seen a trial based on the patient's assault on his wife than a trial regarding his mania.

I do not blame Mr. A for not following the medication instructions, for failing to make or keep appointments for follow-up, or for "refusing to recognize his illness"—he seems to have had good reason to suspect his physician's allegiance. Although it may not have been intentional, the psychiatrist in this case gave primary concern to the wife's fears, not to the patient's rights. I hope Mr. A will someday encounter a therapist with whom he can feel safe and be safe. Only then will Mr. A be able to recognize his self-defeating behavior.

I must have missed some major point in the article, because I could not follow Dr. Adebimpe's final remarks. It seemed to me that Mr. A continued living with the burden of impending death (i.e., his coronary disease). In addition, Mr. A was apparently able to carry out an independent existence, to move, to apply for Veterans Administration benefits, and to refrain from aggression against the wife who abandoned him. In other words, the patient refused to comply with the doctor's predictions of dangerousness. Although I personally would not enjoy enduring the patient's posthospitalization existence, I do not see reason to invoke the powers of the state to protect him from that existence.

When Dr. Adebimpe states, "The answer is clear," I do not know what he means. What is the question? If the question is "Should all manics be locked up?" or "Should all mentally disturbed persons be locked up?" or "Should a person be locked up because a psychiatrist predicts violence?" my answer is a fervent no. However, if the question is "Should dangerous people be locked up?" I would say yes, but I would need evidence that mortal danger is present. I would require confinement not because someone is "sick," but because he/she is sick *and* dangerous as demonstrated by previous behavior. I would rely on legal definitions of offensive acts and penalize the individual for these acts, not for his/her illness. In any case, I would not call a psychiatrist into the court proceedings until the trier of fact had found the facts and made conclusions of guilt or innocence based on those facts. If we must release "sick" people who have committed no serious offense, so be it. Let there be consequences for behavior proportional to the offense and let them be uniformly applied.

We in Missouri are attempting to remedy past abuses of citizens' rights with Senate Bill 275; other states have made such changes already. But providing for due process is only a first step. Ultimately, we need to alter attitudes which hold that mentally disturbed persons should be subject to more, or more severe, sanctions than others who commit similar offenses.

LAWRENCE R. DOSS, M.D.
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Dr. Adebimpe Replies

SIR: A more leisurely reading of the article might reassure Dr. Doss that rather than adopting an omniscient posture, I attempted to acquaint the court with medical and psychiatric considerations that would have been applied to a decision for discharge had Mr. A been a voluntary patient. These included information about the intra- and interpersonal dynamics in the case. They were probability statements, not ex cathedra pronouncements, and none of the recommendations were intended to be instructions.

Psychiatric diagnosis was not invoked as a basis for incarceration. The patient's heart condition and lithium therapy required medical surveillance "ideally in an inpatient setting." His improved but continuing mania prevented him from seeing that assaulting his wife and daughter had been out of character. Therefore, "immediate release was ill-advised [because] treatment for a week or 2 beyond the 10-day period of the emergency commitment might give the patient a better chance for optimal response to therapy." His previous record of outpatient attendance was bad.

Obviously, the point of the article needs to be more concretely stated. There was no question of "refusing to comply with the doctor's prediction of dangerousness," since my testimony was that the risk of deliberate acts of violence was low. The critical issue is that if, as a result of mental illness, a man's actions are unintentionally detrimental to his own health and to the social and economic well-being of his family, do we need to wait for him to hold a gun to his head or theirs before deciding that some decisions must be made for him until he gets better? Opinions are still divided on the issue, because one of those decisions is that his liberty be temporarily curtailed.

This case illustrated some possible consequences of insisting on liberty at all costs. Mr. A became estranged from his wife and daughter, not the kind of "independent existence" that most people might envy. His heart condition became worse, and the decision to see a cardiologist was no longer optional. The necessity of discontinuing lithium carbonate showed that his physician's concern about monitoring the early stages of therapy with this drug was justified. The continuation of his mania for 2 more months may have been the result of outpatient treatment, but we cannot be sure. In this locality the average length of inpatient stay for his condition is much shorter than that.

The outcome in this case suggests that if we define dangerousness only as "the probability that the individual will inflict harm on himself or others in a specific physical manner," we are obliged to give some patients liberty at the risk of consequences that we would not ordinarily wish for ourselves. In addition, we may deprive them of the right to the type of treatment they themselves might have requested had their judgment been intact.

The question is, "Which is preferable: worsening untreated heart disease, prolonged episode of mania, forced estrangement from family and home, and transfer to another state, or 3 weeks of inpatient treatment?" Although some of these outcomes are the result of the unique court ruling, it is clear that when neither alternative is desirable, it is common sense to choose the lesser of two evils. Liberty is fine, but the best should not be the enemy of the good.

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Subcategorizing Gilles de la Tourette's Syndrome

SIR: On the basis of our study of 450 patients with Gilles de la Tourette's syndrome (1), we suggest that there are eight types or subcategories of movement disorders presently subsumed under this diagnosis. The syndrome is a chronic, multiple-tic condition that begins in childhood and is characterized by involuntary, sudden, rapid, and purposeless movements and vocalizations that can include sounds, words, and coprolalia.

The eight subtypes and their characteristics are as follows:

1. Transient tic of childhood. The symptoms are indistinguishable from the initial symptoms of Tourette's syndrome. The only difference is that the symptoms remit spontaneously within one year.
2. Subacute multiple tic of childhood or adolescence. The symptoms and clinical course are indistinguishable from Tourette's syndrome and last longer than one year, but complete remission occurs before or during adolescence.
3. Dystonic variant of Tourette's syndrome. This group includes patients whose movements have both a fast and slow or dystonic component. The tics of Gilles de la Tourette's syndrome are frequent, brief, rapid, sudden (lightning-like), and jerky. Patients in this subgroup have dystonic movements as well. These movements are slow, stretching, writhing, or manneristic squeezes, somewhat (but not exactly) like the symptoms characteristic of focal dystonia. The symptoms are not fully developed, however, and resemble partial, incomplete, or abortive forms of blepharospasm, torticollis, or writer's cramp. These dystonic symptoms respond less successfully to treatment with haloperidol.
4. Invariant tic symptoms beginning in childhood. These patients' symptoms begin in childhood and are characteristically more stable and constant than the variable and changing symptoms in Tourette's syndrome. In Tourette's syndrome, symptoms change slowly over time, with new symptoms replacing or being added to old ones. Symptoms wax and wane spontaneously, and the clinical course characteristically fluctuates. In this subgroup, although the symptoms also include variable, spontaneously changing multiple tics, the symptoms are fewer and less variable, show less spontaneous changing, wax and wane less, are more stable and stereotypic, and tend to remain the same throughout life. Our impression is that myographic recordings would differentiate tics from these muscular movements because they tend to be slightly stronger, definite, and vigorous. Vocal tics are less frequent and intense and have a different tonal character. These symptoms respond to treatment with haloperidol, although somewhat less successfully than the more classic tics of Tourette's syndrome.
5. Invariant tic symptoms beginning in adulthood. Symptoms in this group are similar to those described for group 4, except that they begin in adulthood, frequently during or after middle age. Only one or, occasionally, two tic symptoms are present.
6. Polymorphous perverse variant. These patients have a mélange of symptoms characterized by vivid, changing, and primitive sadomasochistic and polymorphous perverse sexual fantasies; excessive intense and bizarre thoughts and ideas; obsessive, compulsive, and echo phenomena; thought fixation; and fog states. A classic case of this type was described by Meige and Feindel (2) in a chapter entitled "The Confessions of a Victim to Tic," and several other case descriptions appear in the older literature.

7. Self-mutilation variant. Self-mutilation is a symptom of a small number of patients with symptoms otherwise similar to those of Tourette's syndrome. First described by Van Woert and associates (3) and suggestive of but not identical with the severe self-mutilation that occurs in children with Lesch-Nyhan syndrome, such patients have a tendency to severely bite their tongues, lips, or cheeks.

8. Treatment-resistant type. Patients in this group fulfill the classic criteria for Tourette's syndrome but fail to respond adequately to treatment. Despite considerable effort in titrating the dosage of haloperidol and the management of side effects over a long period of time, these patients (who represent less than 5% of the total group) develop side effects that offset the benefit of haloperidol. The side effects appear to be more of a problem than the tics. Clinically, these patients seem to be extremely sensitive to the extrapyramidal side effects of haloperidol. The possibility that they have another subcategory of movement disorder should be considered.

These variations in symptoms and clinical course may be different manifestations of essentially one illness or may represent different disease entities. These initial and preliminary clinical observations should be further evaluated by careful, controlled clinical and biochemical studies.

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ARTHUR K. SHAPIRO, M.D.
ELAINE SHAPIRO, PH.D.
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Improvement or Relief from Side Effects?

SIR: The article entitled "The Treatment of Acute Schizophrenia Without Drugs" by William T. Carpenter, Jr., M.D., and associates (January 1977 issue) raises several interesting questions. The authors suggest that these questions could be answered by careful scientific study, but their investigation was neither careful nor scientific. They acknowledged two major flaws in their study: 1) the failure to control treatment in either patient group, which reduces the value of any outcome comparison, and 2) differences in timing of follow-up, which also hinders meaningful comparison.

A third flaw involves the apparently haphazard use of medication. The authors indicate that drug prescription in the National Institutes of Health (NIH) sample was related to the patient's date of admission rather than symptom or prognostic status. If drug prescription was determined in this way, one must question both the objectivity of the staff in making key treatment decisions and the validity of the drug versus no drug comparison.

The authors conclude that treating acute schizophrenics without drugs is "feasible." However, the fact that the patients treated without drugs at NIH had a 35% rehospitalization rate within 9 months of discharge does not suggest that these patients did terrifically well.

The authors did not comment on the higher socioeconomic status of the NIH group compared with the International Pilot Study of Schizophrenia (IPSS) sample. Patients in the NIH group were significantly more likely to be in social classes I or II than the IPSS group ($p<.001$, chi-square test). Higher social class and mobility have been associated with better outcome (1–3).

The fact that, among those NIH patients treated with medication and then withdrawn from it (after an average of 46 days), significant improvement was noted during the no-drug period is not surprising given the following examples cited by the authors: "more spontaneity, fuller affect, less psychomotor retardation, and more social and work initiative." It appears that the authors were rating drug side effects, particularly akinesia (4), as psychopathology. This could certainly account for the "improvement" in these patients when drugs were withdrawn. No mention is made of examinations for extrapyramidal side effects or use of antiparkinsonian agents.

It is unfortunate when serious investigators address major issues with poorly designed studies and easily misinterpreted data that only serve to increase confusion.

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JOHN M. KANE, M.D.
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Dr. Carpenter and Associates Reply

SIR: We had hoped that our communication would encourage further consideration of several issues in the treatment of schizophrenia rather than evoking polemics. We have received several other communications that, by the nature of their praise or criticism, suggest we missed the mark. Perhaps this response will enable us to come closer to achieving our goal.

Our review of the literature suggests that the predominant treatment considerations in schizophrenic patients rest on a very narrow information base. What may seem to be important operating assumptions should actually be regarded as questions urgently in need of careful scientific study. In our report of observations and thoughts based on our experience with a treatment program at NIH, we carefully indicated that these observations were not from a study designed to compare different treatment approaches; we certainly did not represent our observations as providing any definitive answers. We did not have controlled treatment conditions in either patient group, and this does limit the value of the outcome comparisons. It was precisely for this reason that the question we addressed with these data was of limited scope. Was there evidence that patients receiving psychosocial treatment with little or no medication fared poorly compared with those receiving "usual" treatment?

A second flaw pointed out by Dr. Kane was the uneven

timing of follow-up evaluations. We called attention to this problem in our article, but in a footnote we presented data indicating that the unequal timing biased against the NIH patients and therefore did not undermine the conclusions we drew from these observations.

Dr. Kane discusses a third flaw, but his discussion seems to indicate a considerable difference in his views and ours on the clinical decision-making process. We discussed a probable indication (related to phasing in and phasing out of a particular treatment program) that the date of admission was an important variable in determining medication status in these patients. Many considerations enter into treatment decisions, and clinicians who view themselves as operating purely objectively without being influenced by the social setting in which they work are maintaining a view that is contrary to our understanding of the clinical decision-making process.

In regard to Dr. Kane's comment about the 35% rehospitalization rate, we did not claim that NIH-treated patients had done "terrifically well," nor did we provide a psychosocially oriented treatment during the aftercare period. Furthermore, the 35% rehospitalization rate does not compare unfavorably with a number of other studies in which maintenance antipsychotic medication was used. Dr. Kane ignores the immediately relevant information in our report, which indicated that the NIH patients had done at least as well as the drug-treated comparison group. Furthermore, those NIH patients who received antipsychotic drugs during hospitalization were slightly more likely to be rehospitalized during the follow-up period than patients treated without medication.

Whether one would expect schizophrenic patients to improve if medications were discontinued before discharge is a moot point. It is a common apprehension that clinical deterioration will occur when drugs are discontinued, and patients are generally left on medication throughout their hospitalization, even when clinicians expect the patient to take medication unreliably after discharge. Akinesia and sedation are often side effects of antipsychotic medication, and one would not be surprised to see the type of improvement we described as patients come off these drugs. However, it is important to consider this potential benefit in relation to clinical management. If, for example, side effects in some way mimic residual or defect symptoms of schizophrenia or induce apathy or demoralization, the immediate course of illness will be complicated by these side effects. We did argue that this was sufficient reason to discontinue drugs, but we did think that what happened when we took patients off drugs before discharge for purposes of the research protocols was worth mentioning.

Dr. Kane is correct in noting that there is a disproportionate representation of higher social classes in the NIH patients compared with the IPSS patients. This represents a minority of the patients and, taken in conjunction with the other extensive prognostic data, does not seem to undermine our conclusions. We apologize for this error and have since looked at the relationship between social class and outcome in the NIH patients. Using the nonparametric statistic tau and the Pearson product-moment correlation coefficient, we found no significant relationship between social class and outcome. A positive correlation was predicted, but the correlation coefficients were $-.17$ and $-.22$, respectively, both nonsignificant. This does not suggest that the influence of social class on course and outcome in schizophrenia has been overrated, but with relatively small numbers and with very few patients falling into the very lowest or very highest social classes, it is not a significant operating bias in this cohort.

We certainly share with Dr. Kane the view that serious questions should not be addressed with poorly designed studies and easily misinterpreted data, as our critique of the literature indicates. Our care in describing the nature of our data and the limitations of its interpretation was intended to discourage misinterpretation and to encourage open-mindedness regarding the optimum treatment of schizophrenia.

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More on Exhibitionism in Women

SIR: In "Genital Exhibitionism in Women" (April 1977 issue) Marc H. Hollender, M.D., and associates did not mention in their review of the literature a paper I have written on the subject. The case I analyzed (1) clearly shows that genital exhibitionism by women can be a sexual perversion that is almost identical with that seen in men. The exposure of the genitalia was accompanied by masturbation and orgasm; it was also motivated by castration anxiety. One characteristic difference was the need for a fetish during the act.

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GEORGE ZAVITZIANOS, M.D.
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A Middle Ground in Prophylactic Antiparkinsonian Medication

SIR: Recently, two children I had been treating with phenothiazines suffered dystonic reactions. In both cases I had warned the children's mother of this possibility, and I had supplied both mothers with small samples of benztropine mesylate. By the time the mothers called to express their concern they had already given the medication to their children; both crises passed within a half-hour. The mothers were very grateful that they had been prepared. One child was a 9-year-old boy being treated with 1 mg/day of haloperidol for Gilles de la Tourette's syndrome and the other was a 12-year-old boy being treated with thioridazine, 200 mg h.s., for severe childhood schizophrenia. Both boys are now receiving 2 mg/day of benztropine mesylate in addition to their phenothiazines.

I am writing about this situation because I continue to see both positive and negative communications regarding prophylatic antiparkinsonian agents. Many physicians believe that one should wait until problems occur before prescribing antiparkinsonian medication.

I like the idea of waiting until trouble occurs, but once it does the patient suffers while the family contacts the physician, who then has to call the pharmacy and prescribe the medication, which then must be picked up; meanwhile, the

patient is dystonic and very frightened. For that reason I now supply a small amount of antiparkinsonian medication to those patients I think are more likely to experience a dystonic reaction. This has worked out very well so far, as the two examples above illustrate.

I thought my experience might be of interest to other psychiatrists and physicians in view of the contradictory opinions on prophylactic antiparkinsonian medication. I feel a middle ground is a wiser course.

DIRK E. HUTTENBACH, M.D.
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Creative "Restraints"

SIR: In "Children's Rights on Entering Therapeutic Institutions" (February 1977 issue) Derek Miller, M.D., and Robert A. Burt, J.D., used psychological insights to arrive at practical solutions that I would like to support with some of my own experiences. At our center in Austin, Tex., we have not yet experienced the courts' intervention in limiting parents from obtaining treatment of a protesting child. In our open rural setting, with a policy against any kind of physical structure into which a child could be locked, we have successfully treated many children thought to require closed settings because they have run away repeatedly. We agree with the authors' emphasis on symbolism and relations as being more important than physical structures in dealing with this problem.

During the early stage of treatment, we work hard to develop relationships of sufficient quality to ensure that the child who may run away will return. We use symbolic means of restraint, requiring "runaway risks" to wear gray sweatsuits called "pajamas." Since, according to our lore, one cannot elope in pajamas, the potential runaway must somehow sneak clothes (which is not difficult). Although our sweatsuits would attract little attention in the community, only two children (both psychotic) have run away wearing "pajamas." We also require a child who threatens to run away to stay with staff. If he/she needs further structure, we tie him/her to a staff member with an easily broken piece of kite string; this restrains the child only symbolically but gives support against impulsivity. We can then selectively interpret the situation (and the symbiotic conflicts involved), to for example, a 6-foot-tall muscular 16-year-old complaining bitterly of being prevented from running off by being bound to a 5-foot-tall, frail female staff member.

It would be easy to run away from our center by feigning cooperation for a few hours and then slipping off into the woods near the highway. Such runaway episodes occur at a rate of about 1.5 a week (per 55 patients). Usually these episodes occur at specific stages of treatment and provide a useful focus for interpretive work. Occasionally, significantly high rates (along with other indicators) have led us to seek out and remedy program problems to which running away was a reaction, as the authors suggested. However, out of 228 primarily high-runaway-risk adolescents whose treatment we have undertaken in the past 3 years, only 21 (9.3%) have failed to return from various runaway episodes to continue treatment with us. Many closed facilities I have known have had higher rates of elopement.

These facts support the recommendation by Drs. Miller and Burt that to keep acting-out adolescents in treatment, one relies not on secure physical custody but on their obvious capacity, however protested, to recognize and use treatment that meets their needs. Certainly the adolescents at our center who have been "voting with their feet" recognize treatment with token restraints as the least restraining alternative.

MICHAEL S. WEINER, M.D.
Austin, Tex.

More on Atypical Facial Pain

SIR: I read "Atypical Facial Pain as a Defense Against Psychosis" by John F. Delaney, M.D. (October 1976 issue) with interest. It is a not infrequent clinical experience to find symptoms of somatization and pain in psychotic patients. However, by emphasizing psychodynamics Dr. Delaney has tended to neglect other facets of his cases, namely, the relationship (if any) of drug abuse or withdrawal to the onset and presence of hallucinations and confusion in these patients. He also did not deal with the possibility that these cases may represent a chance association between relatively common symptom complexes, i.e., atypical facial pain and psychosis.

I would like to present a summary of a case that illustrates an association between intractable headache and psychosis (1). The patient was first admitted to a mental hospital in 1958 when he was 36 years old. He had auditory hallucinations (mainly the devil's voice) in clear consciousness, as well as persecutory delusions and ideas of reference. There was no history of alcohol or drug abuse, head injury, or encephalitis, and physical examination was normal. A diagnosis of schizophrenia was made, and the patient responded to high doses of chlorpromazine and was discharged to his father's care in 1959.

Severe headaches over the vertex were first noted during a short readmission in June 1959. His psychotic symptoms and headaches were largely unchanged on each of two further readmissions (1964 and 1972); each time he improved sufficiently on medication to be discharged.

On his last admission, the patient for the first time manifested confusion, and a history of abuse of phenacetin-containing headache powders was obtained from his brother. Urinalysis revealed blood and protein in the urine. Blood tests were as follows: sodium, 141 mEq/liter; potassium, 5.6 mEq/liter; chlorine, 102 mEq/liter; HCO_3, 18.5 mEq/liter; urea, 505 mg/100 ml; and hemoglobin, 8.9 g/100 ml.

The patient died 4 days after his admission to the hospital. The main findings at autopsy were an analgesic nephropathy and cardiomegaly. Semi-serial section of the brain stem revealed an extensive encephalitic-type lesion (glial knots, neuronal loss, and perivascular infiltration) involving the left trigeminal nucleus and extending from the main sensory nucleus to the caudal part of the spinal nucleus.

This case illustrates not only the complexity of the factors to be considered but also the morbidity and mortality that may be associated with this syndrome.

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M. FISHMAN, M.B.
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Narcolepsy and Schizophrenia

SIR: I read with great interest the article by Bruce Shapiro, M.D., and Henry Spitz, M.D., "Problems in the Differential Diagnosis of Narcolepsy Versus Schizophrenia" (November 1976 issue). At present I am treating a patient who in my opinion has both schizophrenia and narcolepsy.

The patient is a 50-year-old man who approximately 10 years ago began experiencing the sudden onset of sleep while eating, driving, or working. In several instances the sudden onset of sleep resulted in moderate injury and on at least three occasions he miraculously avoided serious injury when he fell asleep at the wheel of his automobile.

The patient has been evaluated in the past by a number of neurologists and psychiatrists who entertained the possibility of concurrent narcolepsy and depression. However, EEGs were consistently normal; a 24-hour EEG was never performed. He was placed on a trial of methylphenidate and imipramine, with only mild improvement.

An in-depth history on this patient revealed that he experiences cataplexy, sleep paralysis, and hypnogogic hallucinations. He also acknowledges the presence of auditory hallucinations. His mental status examination was noteworthy in that his affect was flat, his mood was severely dysphoric, and he was fearful that people were out to harm him. His sensory examination revealed no signs of organicity.

Psychological testing revealed a profile that best fit the category of a schizo-affective disorder. Neurological referral substantiated the earlier impressions that narcolepsy was present, although once again an EEG done in the office was unremarkable.

The patient has responded well to a combination of 150 mg/day of amitriptyline and 30 mg/day of *d*-amphetamine after trials of up to 60 mg/day of methylphenidate failed to improve his narcolepsy. However, he continues to experience auditory hallucinations, although they are somewhat less disconcerting and occur less often. The patient's *d*-amphetamine dosage is still being readjusted and the addition of antipsychotic medication is being considered.

I present this case to remind clinicians that schizophrenia and narcolepsy need not be mutually exclusive and may be present simultaneously.

KENNETH C. ULLMAN, M.D.
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Drs. Spitz and Shapiro Reply

SIR: We thank Dr. Ullman for his interest in our article and the description of the patient he is presently treating. Dr. Ullman is clearly correct in stating that narcolepsy and schizophrenia may coexist. In fact, this was documented in the article by Sours (1) that was included in the bibliography of our paper. Sours stated that "eight patients displayed schizoid personality disturbances. Ten other patients developed frank schizophrenic psychoses which required prolonged hospitalization. Three such patients had been taking Dexedrine."

It is interesting to note, however, that Dr. Ullman's patient is described as having responded well to amitriptyline and *d*-amphetamine. Both of these drugs are well known to exacerbate schizophrenic psychosis; in view of a favorable response, one must wonder whether this patient truly has a schizophrenic illness.

Perhaps this only further highlights the point made in our paper regarding the need for accuracy in formulating diagnostic criteria when faced with clinical situations in which there is major overlap of symptoms.

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HENRY I. SPITZ, M.D.
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Betel Nut as a GABA Blocker

SIR: In a most interesting paper, "γ-Aminobutyric Acid (GABA) and the Dopamine Hypothesis of Schizophrenia" (February 1977 issue), Daniel van Kammen, M.D., pointed up the importance of examining ways of modulating the inhibitory gabergic control of the putatively overactive dopamine-containing cells of the substantia nigra in schizophrenia. The tardive dyskinesia problem makes it particularly urgent to develop new treatment methods to replace the phenothiazines and the butyrophenones. Dr. van Kammen stated that there are no known blockers of GABA uptake with sufficient specificity to warrant study. Any such agents might act in schizophrenia similarly to the way that tricyclic antidepressants work in depression by elevating the functional levels of GABA in the gabergic synaptic cleft.

Dr. van Kammen did not mention a recent paper (1) reporting just such an agent, arecaidine, the active ingredient of betel nut. Betel has been utilized in India for centuries as a "tranquilizer." For a long time it was thought that the pharmacologically active ingredient was arecoline, a muscarinic cholinergic agonist. However, the nut is chewed with lime, which hydrolyzes the arecoline to arecaidine. This is a potent blocker of GABA uptake. Thus the psychopharmacological effects of betel nut in normal people may be due to its raising effective brain GABA levels. This in turn suggests that arecaidine and related agents should be tested in schizophrenia. Betel nut chewing causes ulcers and eventually cancers of the lip and mouth, but this is almost certainly due to the excessive exposure to the lime and not to the arecaidine per se. Arecaidine itself may not cross the blood-brain barrier, but a metabolite may do so.

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JOHN R. SMYTHIES, M.D.
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On Diagnostic Classification

SIR: In "Mania and Schizo-Affective Disorder, Manic Type: A Comparison" (December 1976 issue) Richard Abrams, M.D., and Michael Alan Taylor, M.D., hypothesized that mania and schizo-affective disorder, manic type, were separable on the basis of the nature of the secondary

symptoms, as proposed *DSM-III* criteria suggest. They were unable to demonstrate such a distinction, probably because of marked confusion between basic signs and secondary symptoms in these criteria.

My clinical impression is that these two disorders are different psychoses that occur in different personality disorders—manic psychosis in cyclothymic personality and schizo-affective schizophrenia in hypomanic personality.

The signs of manic psychosis include: affect—absent and cynical; mood—absent and hyperenergetic; thinking—concrete and pressured; behavior—aimless and exhausting activity; defense mechanisms—wish fulfillment and hyperactivity; and judgment—impaired regarding creativity. The symptoms include: stereotyped hyperproductivity, clang associations, word salad, cyclical psychosis, stereotyped counterfeiting, and plagiarism.

The signs of schizo-affective schizophrenia include: affect—silliness, volatile swings; mood—swings from excitement to dullness; thinking—illogical and superficial; behavior—bizarre and intermittent hyperactivity; defense—global withdrawal of interest and conventionality; judgment—impaired regarding social protocol. The symptoms include bizarre cultishness, bizarre drug and alcohol abuse, bizarre imaginary companions, neologisms, and bizarre worship of youthful appearance.

I hope that if the authors reexamine their data they will find that these criteria differentiate the illnesses more successfully. They are to be lauded for investigating some of the hypotheses on which *DSM-III* will be based and demonstrating their inaccuracy. The basic definitions of signs and symptoms in *DSM-III* proposals have little resemblance to actual signs and symptoms in patients. The spokesmen for the profession would do well to bring a halt to these proposals before we are faced with a hopelessly confused statistical nightmare that bears little, if any, resemblance to psychiatric disorders as they exist clinically.

AUGUSTUS F. KINZEL, M.D.
New York, N.Y.

Drs. Abrams and Taylor Reply

SIR: Dr. Kinzel offers his clinical impression of possible differences between manic psychosis and schizo-affective schizophrenia based on a hypothesis that the conditions are derived from distinctly separate disorders of personality and manifest specific differences in patients' presentation of psychopathology.

Unfortunately, the validity of the personality disorders he describes has yet to be determined, and we are unaware of specific criteria that could be used to test his hypothesis. The individual signs and symptoms he describes are subject to the same criticism—how, for example, would one test for "bizarre cultishness," and what evidence is there for its association with schizo-affective schizophrenia?

With a little imagination, one could create any number of "syndromes" of the sort Dr. Kinzel presents; the point is to subject them to validity testing, and this we invite Dr. Kinzel to do.

The major thrust of our research findings has been the demonstration that once a patient satisfies criteria for affective disorder, the additional presence of first-rank symptoms or other "schizophrenic" features is not of critical importance. Current procedure for *DSM-III*, however, assigns preeminence to the presence of schizophrenic features, and it is this process we would like to see reversed. If our recommendation were followed, schizo-affective disorder would be included as a subgroup of affective disorder rather than schizophrenia.

Finally, Dr. Kinzel may mistakenly feel that because we have criticized one aspect of the proposed *DSM-III* criteria we are in opposition to the entire proposal. On the contrary, we feel *DSM-III* is a progressive document of major importance for American psychiatry, and we have taken the time to examine the validity of some of its assumptions in order to strengthen it.

RICHARD ABRAMS, M.D.
MICHAEL A. TAYLOR, M.D.
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On the Psychology of Military Incompetence, *by Norman Dixon. New York, N.Y., Basic Books, 1976, 439 pp., $12.50.*

Dr. Dixon has written a rich and fascinating account of the psychology of military command. He has examined various military leaders and commanders of the past and divided them into the successful and the failed. The book attempts to document the basic attributes and the psychological makeup of the contrasting types.

Dixon defines the characteristics of military incompetence as those which involve wastage of human resources, clinging to outworn tradition, the tendency to reject or ignore unpalatable information, the tendency to underestimate the enemy, indecisiveness, obstinate persistence at a fruitless task, failure to exploit favorable situations, failure to make adequate reconnaissance, a predilection for frontal assaults, belief in brute force, the failure to make use of surprise or deception, undue readiness to find scapegoats, suppression or distortion of news from the front, and belief in such mystical forces as fate or bad luck.

The first part of the book is devoted to a rousing discussion of prime examples of military incompetence on a vast scale. The author confines himself to examples from the British Armed Forces. As a former officer in the Royal Engineers he is most familiar with his own army's past. In any case, the British Army in the nineteenth and twentieth centuries furnished classical examples of stark incompetence. Dixon describes the Crimean War, the Boer War, warfare in India, the First World War, and the Second World War battles for Singapore and Arnhem.

Criticizing British leadership in those conflicts is, of course, shooting fish in a barrel. There could not be any more outrageous examples of total incompetence than the British command in the Crimean War. In Lord Lucan, Lord Ragland, and Lord Cardigan England managed to put together in leadership posts three men utterly unfitted for the conduct of any large enterprise, let alone one operating under the exigencies of war. These feckless aristocrats have furnished the inspiration for many books and films. It would be very difficult to find in other wars quite the careless failure to grasp wartime realities that the charge of the light brigade represented. As the French said at the time, "It is magnificent but it is not war." Nor was it successful. Lord Ragland, the British Commander-in-Chief, extravagantly exemplified most of the attributes of military incompetence mentioned above.

Later in the book Dixon deals with successful military leaders, such as Nelson, Wellington, Slim, and Allenby. They, of course, receive much less mention than do their incompetent brethren. The book, after all, is about the psychology of military incompetence rather than competence. No doubt bumblers are far more interesting subjects for psychological study than are the notoriously successful.

Dixon delineates a personality profile that he believes is characteristic of incompetent militarists. The personality type is one of anal obsessionalism, lack of self-confidence, and authoritarianism. These primary qualities are displayed as outward traits of rigidity, conformity, traditionalism, anti-intellectualism, indecisiveness, and stubbornness. Traditional military organizations reinforce these personal defenses against primary anxiety by their emphasis on ritual, dogma, codes of honor, and by what the English call "bull" and the Americans call "chicken." "Bull" is petty intrusiveness about such symbolic items as the shining of belt buckles and shoes, short haircuts, and the length of fingernails. Successful leaders tend to be those who possess creativity, flexibility, high intelligence, strong self-confidence, and a lack of authoritarianism.

The author makes a good case for categorization of commanders into these groupings; however, the case remains unproven in the scientific sense. The book shares the weakness of all psychohistory. The arguments are plausible enough in the manner of the law rather than as scientifically corroborated insights. The predominant use of secondary or tertiary sources further increases one's hesitancy about the scientific validity of the author's assertions. For example, Dixon states that Field Marshal Haig, the British Commander-in-Chief on the western front in World War I, possessed "well-marked obsessive, authoritarian traits " He also states that Haig suffered from "asthma" and alleges that he had one of his worst attacks before the ill-fated Battle of Loos in 1915. The author further asserts that asthma is a psychosomatic complaint that results from repressed aggression. All of this may be correct, or it may not be. One would have to examine Haig's medical records to know whether, in fact, he really had asthma or some other respiratory disorder that in the context of 1915 medicine might have been known by the generic name of asthma. Additionally, although few would deny the role of psychic factors in the etiology of asthmatic attacks, the belief that asthma comes on only as a result of psychogenic forces is not held by those who have studied the disease well. Finally, the author would have to demonstrate that the peculiar circumstances of that particular historical moment provoked an asthmatic attack that served as a "psychosomatic outlet for surplus aggression." This is only one of the innumerable points the author makes that would be open to challenge by someone who examined the situation in a detailed, skeptical way.

There is a disjunction between the two basic sections of the book: very lively and interesting historical and personality descriptive sketches versus an accurate description of such psychological configurations as the anal or the authoritarian personality. It is in the attempt to blend these two areas that the seams show. The blending is not smooth; the transitions are abrupt, making it seem as if there is an arbitrary quality to the juxtaposition of the historical and the psychological material. The psychohistorical genre is prone to this jagged quality of format.

Nevertheless, in spite of the feeling of final doubt about the proof of the author's main thesis, the book is a marvelous Christmas fruitcake filled with all kinds of gorgeous stories and descriptions of individuals who operated at a high level

of drama, however unworthy their own talents were to cope with the vast challenges of warfare.

All who have served in the military will recognize the types described by Dr. Dixon. They exist at lower levels as well as in exalted commands. However, anyone with further experience of life would know that such types are not, by any means, confined to the military. Civil government, the world of commerce, universities, and the various medical societies have their due share of the timid, the stubborn, the authoritarian, the indecisive, the opinionated, the dogmatic, and the incompetent. One is struck, looking at high command in any organization, by the unevenness of quality. Some individuals are supremely well-fitted for posts of major responsibility. Others have characterological and intellectual defects that make one wonder how any selection process could come up with such obvious misfits. It seems to be in the nature of human organizations that it is not always possible to choose competent leaders. In the case of the wartime military, this is particularly tragic because of the wastage of lives. A bad dean can make trouble for a school of medicine and a bad chairman of the board can run a company into bankruptcy; but a bad fleet admiral or field marshal can destroy thousands of lives unnecessarily.

RANSOM J. ARTHUR, M.D.
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Philosophical Dimensions of the Neuro-Medical Sciences, *edited by Stuart F. Spicker and H. Tristram Engelhardt, Jr. Lawrence, Mass., D. Reidel Publishing Co., 1976, 268 pp., $29.00.*

This is in many respects a remarkable book. It is, to begin with, a fine specimen of bookcraft, splendidly printed and well bound, making it a pleasure to handle and to read. Also, it is devoted to an esoteric theme, one that embraces and attempts to both bridge and reconcile the basic scientific assumptions on which the neuromedical sciences are founded with the philosophical issues that these assumptions engender in the "philosophically minded."

It is to be said forthwith that the attempt at the aforedescribed reconciliation is, in this instance, inspiring but not very definitive. Prof. Marjorie Grene, in her summating remarks, states, "The network of problems in which . . . we have been entangled . . . this nest of problems has certainly not produced a finished pattern of thought for neuroscientists and philosophers to follow." On the contrary, to some extent, she says, the result was "misunderstandings or just missing of minds." However, those who will trouble to read this book (and it does not offer easy reading) will also concur with Professor Grene's enthused comments: "Yet how varied, how full of content, how tantalizing are the horizons opened up by this closing session alone," and, I would add, the other sessions too.

The central and basic question at issue in this symposium was the relation of brain to mind. Is mind an "emergent of brain organization" or is mind, as Professor Grene argues, "extrinsic," while brain, as part of the body, is "intrinsic"?

In the pursuit of these issues the 17 symposium participants ranged far and wide, drawing on the motley history of neurology, phrenology, psychosurgery, hemispheric singularity and dominance, pain, and Freud. The philosophically minded brought into the fray include Descartes (both pro and con), Flourens, Fourier, Cabanis, Magendie, Hughlings Jackson, Kant, Lashley, Sherington, Kurt Goldstein, and Michael Polanyi, to name only the preeminent and at random.

As indicated in Professor Grene's cited comments, the symposium did not yield a consensus, but the published volume of its transactions offers stimulating, provocative, and in some elements distressing reading. One distressing element is the iron grip that Cartesianism, in its materialist, mechanist, and positivist commitments, exercises on the thinking of so many of the neuromedical scientists. These scientists' simplistic, naive concepts of causality are astonishing, and their dedication to the Cartesian *Méthode*, in the light of what we know of Gestalt and holism, is archaic and atavistic.

There is, pace Pythagoras, a science not only of things but of their relations. Hans Jonas argues this (and more beside) in a brilliant chapter titled "On the Power or Impotence of Subjectivity." Professor Grene, in an equally noteworthy chapter titled "Mind and Brain: The Embodied," succinctly affirms that the belief "that there *are* sensations, isolable, identifiable, single bits of consciousness out of which experience gets built is just plumb *wrong*. . . . Our everyday experience just is not made up of psychological atoms." Incidentally, Hans Jonas and Marjorie Grene are the only symposium participants who show any sympathetic understanding of Freud and of psychoanalysis. Psychiatry itself receives but scant notice, and in the main this is negative.

To summate, this is a fine, stimulating, and instructive book that deserves a place of prominence in one's reading list and library.

IAGO GALDSTON, M.D.
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The Post-Physician Era: Medicine in the Twenty-First Century, *by Jerrold S. Maxmen. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1976, 285 pp., no price listed.*

The primary objective of this startling book is to demonstrate the feasibility, inevitability, and desirability of a post-physician era. The author predicts that within the next 50 years the present physician-centered model of health care will shift first to a health team model and then to a "medic-computer" model in the twenty-first century. At that time the physician's head will be replaced by the computer, and his or her heart by a new breed of health professional called the "medic."

The present use of computers to assist in various clinical and nonclinical functions of the physician will increase in such a way that the physician will no longer be needed. History taking, physical examination, ancillary testing, diagnoses, and, with the exception of surgery, almost all treatment will be carried out entirely by the computer. The need for physicians in the twenty-first century, Maxmen argues, is therefore a myth. Logical medical decisions that require objectivity, probability concerns, and memory will be made more competently by computers than by physicians, who are described as "frequently overworked, preoccupied with the technical aspects of their jobs . . . and respected by their colleagues because of their knowledge of medicine rather than because of their sensitivity to patients" (p. 32). Indirect care functions, including research, education, and administration, will also be accomplished by computer. The medic will deal with the educational and emotional needs of patients.

The major organizational issue to be faced in establishing the medic-computer model concerns control over the health system, including priority setting, establishing standards of care, dealing with health costs, and attaining and evaluating continuity of care. Currently, the author suggests, physicians are seen as resentful of and resistant to outside qualified authorities setting standards of medical care. A solution he proposes is worth quoting because it also conveys some of the philosophy and flavor of the book:

> With the emergence of a post-physician era a government panel of experts, drawn from the nation's most qualified medical researchers, could establish high standards of clinical care, program them into the computer, and thereby minimize the problem. . . . [Then,] because an automated system of health care would collect all relevant clinical data, these government-appointed experts will possess more information to make more valid judgments than would their modern counterparts. (p. 98)

A comprehensive review of present advances in medical communication follows, including the use of interactive television, videophone, and wireless monitoring of patients' physiological processes.

In considering the emergence of the medic, the author foresees substantial changes in the education and careers of all the physician's colleagues in the present health care system. It now takes 8 years to train a medical doctor; the medic will become qualified in 18 months. The medic's training will emphasize the psychology of illness, the need for sensitivity to group processes, medical sociology, terminology, ethics, patient administration, and the technical aspects of supportive care. As the traditional medical school disappears, there will be established colleges of health science, medical management, public health, and medical investigation as well as a form of human services information center that would assist in the education of the public.

A chapter titled "Draconian Images and the New Biology" carefully reviews present and predicted issues in organ replacement, reproduction, genetic control, behavior regulations, and aging. The ethical, moral, and legal aspects of new developments in biology are clearly considered. The current complexities of these issues create a "present shock" in the reader.

There are two appendices. One considers the critical elements in future research. The other is a chronology of the future, a daring timetable that predicts interrelated developments, in parallel-step fashion, of communication and computer technology, biomedicine and therapeutics, education and occupations, and several related factors through the year 2025.

The author acknowledges in his introduction that it is a risky business to predict what the distant future can, will, or should be. The feasibility of the medic-computer model is viewed and at times extolled by the author as inevitable. The supporting facts are impressive, compelling, well written, and documented with an excellent bibliography. However, when the author reinforces the model as a desirable conclusion, I felt the uncertainty of the travelers from the yellow brick road when they confronted the wizard. The confusing advice given led them eventually to find that their own inner wisdom, love, courage, and caring were the things that mattered most in the relief of their suffering. In my opinion, Maxmen's medic-computer model does not deal enough with key human and emotional aspects of medical practice. Whether these factors can be programmed along with logic into the computer and whether the medic, as health leader, can be programmed to deal with them through a somewhat instant insight training program in 18 months are left unclear.

The computer age can help us to gain better management of direct and indirect services and to find new metaphors in psychiatry that are presently unseen. Careful attention to future programming will be required forever, lest bureaucratic red tape be merely turned into electronic pathways or practical problems in service delivery be lost in the deliberations of the government-appointed experts who would control the standards.

This book offers many points for criticism and in doing so succeeds in a most difficult task: that of reminding us that chance favors the prepared mind and that preplanning, positive action, and the creation of a constructive and more certain future is preferable to the passive denial of change beyond a 5-year period. As an important addition to the literature on change and the future it also reminds us that 1985 is, in fact, only 8 years ahead.

JAMES W. DYKENS, M.D.
Gardner, Mass.

Pitfalls in Human Research: Ten Pivotal Points, *by Theodore Xenophon Barber. New York, N.Y., Pergamon Press, 1976, 111 pp., $6.95.*

This book is a systematic, concise, and lucid analysis of major sources of fallibility in research carried out by, for, and with human beings. Bias and errors can and frequently do produce invalid conclusions. This fact is exemplified by the author's courageous and penetrating critiques of errors and invalid conclusions in recently published scientific papers. The implications for psychiatric research are profound and more than a bit sobering. The book is of great value to research psychiatrists but, most importantly, provides the practicing psychiatrist with the knowledge he or she needs to be more critical of research reports.

The first "pitfall" Barber describes is the usually implicit research paradigm—those general beliefs and methods shared by many scientists at any given time. The paradigm has a pervasive influence on all subsequent steps in the research process. It determines the choice of hypotheses, the research design, the kind of data to be collected, and how the data will be gathered, analyzed, interpreted, generalized, and published. Barber makes a convincing case for the investigator to be aware of his or her paradigm and to be more explicit about its influence on the conclusions.

The investigator's choice of research design (e.g., same-subjects design versus randomized-group design, the inclusion or exclusion of sex differences, and the choice of scales) can determine whether the investigator obtains positive, negative, or equivocal results. Failure to replicate a study can result from only a slight variation in the research design.

A third pitfall deals with the investigator's failure to write explicit step-by-step details of the procedure, which leaves latitude for experimenters to vary procedures and thus introduce bias and make replication difficult. A fourth problem is the bias introduced by the investigator's statistical procedures. One does not need to be a statistician to understand the logic of nine common ways to manipulate data to yield highly prized "significant findings." To me the chapter on the use of statistical procedures does most to improve the

practicing clinician's ability to recognize at least the more glaring deficiencies in current research reports. Barber is to be commended for his scientific critique of the data analysis procedures employed in two widely cited psychological studies. Had appropriate data analysis procedures been employed they would have produced insignificant differences rather than the "significant findings" that were reported. His recommendations, although primarily statistical, are delightfully logical and devoid of cynicism.

In chapter 5 Barber deals with the taboo subject of investigators "fudging," by which he means the *intentional* reporting of results that differ from the results obtained. This varies from downright fraud to minor but powerful enhancement of correlation values. References from past and current literature support the idea that some investigators actually fabricate or falsify their data. Barber attributes their motivations to the tremendous competition for recognition among scientists. He suggests that the motivation for honesty among scientists is roughly proportional to the probability of detection and public exposure of cheating. He concludes by suggesting that the canons of science are probably sufficient to prevent what he terms "grand scale" fraud but that in a few cases the countermotivation to fudge wins out.

The last five chapters of the book deal with possible sources of bias, error, and incorrect conclusions introduced by the experimenter. Throughout, Barber makes it clear that the investigator is responsible for the selection and conduct of experimenters and cannot ignore the possibility that they too may be fallible.

Chapter 6 convincingly documents that the experimenter's sex, age, prestige, ethnic identity, anxiety, warmth, dominance, friendliness, and hirsute adornment affect the subjects' responses on a wide variety of specific research tests. If the investigator can decide beforehand which personal attributes of the experimenter are relevant to the tests and to the subjects' responses, he or she could select experimenters who differ on these attributes. Conclusions from studies should state that results may be limited to the specific kind of experimenter(s) used in the study.

Chapter 7 deals with the failure of the experimenter to follow the research procedures. Barber reports studies which convincingly demonstrate that most experimenters using a well-constructed protocol have serious difficulties in following prescribed research procedures. Experimenters vary widely among themselves and from subject to subject, both consciously and unwittingly. It appears that careful supervision of experimenters may be necessary to reduce this serious source of error.

Chapter 8 discusses the fact that experimenters can systematically incorrectly record or sum up their subjects' responses. Misrecording is usually in the direction of the experimenter's or investigator's desired result, according to several studies.

In chapter 9 Barber discusses fudging by the experimenter, which is easier to detect than the fudging done in privacy by the investigator. He cites a variety of studies in which experimenters were found to cheat when they were observed or checked on without their being aware of it. A surprising number of experimenters altered their data to make better correlations and, in a more difficult interview procedure, actually made up data out of whole cloth. These findings are supported by post hoc statements made by a number of experimenters regarding their own fudging. Again, it is the investigator's responsibility to provide adequate supervision to minimize cheating.

In chapter 10 Barber puts to rest the popular assumption that the experimenter, through *unintentional* use of paralinguistic cues (e.g., tone of voice) and kinesthetic cues (e.g., changes in posture and facial expression), frequently influences the subjects' responses. He reviews 75 studies that attempted to demonstrate such unintentional effects. Forty percent of the studies demonstrated that experimenters' expectations did not affect the results. The results found in some studies were due to other kinds of bias. Only a few studies showed unintentional experimenter effect as the main effect or an effect in interaction with other variables.

The last chapter, "Overview and Recommendations," contains 13 recommendations that are both a challenge to scientists and a timely contribution to the scientific method. The postscript acknowledges the absence of optimism in Barber's findings but provides substantial advice for those who dare to trod the dangerous road of experimental research.

STANLEY H. ELDRED, M.D.
Belmont, Mass.

Modern Trends in Psychosomatic Medicine 3, *edited by Oscar Hill. Boston, Mass., Butterworths, 1976, 506 pp., $24.95.*

Psychosomatic medicine is alive and growing. It is becoming more visible as it emerges from the research area to the clinical. This process has been developing as the emphasis in the field has shifted from the psychodynamic-intrapsychic to the social-environmental-ecological. Also contributing to the new visibility of psychosomatic medicine are the developments in the field of electronic monitoring of physiological processes, i.e., biofeedback.

In the 1950s and 1960s investigators at the Chicago Psychoanalytic Institute, led by Franz Alexander, developed the concept that three sets of variables codetermined the development of "the seven psychosomatic diseases": a psychodynamic constellation formed early in life, the precipitating life event, and a constitutionally determined organ vulnerability.

In the 1970s such researchers as Roessler and Engel are saying that an individual's emotional and physiological responses to a given environmental stimulus are determined by the following set of variables: the nature and intensity of the stimulus; enduring characteristic, personality, and physiological traits of the patient; and the current state of the organism, i.e., the individual's physiological state at the time he or she receives the stimulus. Z.J. Lipowski opens this book with a fine overview of psychosomatic medicine, providing observations that set the stage for what is to follow.

The book covers 24 different topics contributed by an international group of authorities. The editor has tied the topics together effectively, making it possible to read one or more of the papers with a sense of satisfaction. There is something for everyone in this volume, and as I read it I began to feel that psychosomatic medicine is becoming too appealing to be left to the exclusive domain of psychiatrists.

There is a shift in emphasis in the health care field from crisis therapy to health maintenance. The individual, "the consumer," is expected to contribute more while demanding less of the physician. Dynamic psychiatry has always emphasized this. Now the emphasis is on a much broader scale. If the general physician reading this book sees it as a way of broadening his or her ability to manage a patient, he or she will also see its potential for helping the patient shift to a greater degree of preventive self-help. We cannot and will

not cope with the forces outside ourselves until we are convinced they are there. This book provides support for such awareness.

This book will have some appeal to just about every physician. The topics range from the opening chapter, "Psychosomatic Medicine: An Overview," to the closing chapter, "The Care of Bereaved Adults." I especially responded to the chapters on "Electrodermal Activity," "Psychoendocrine Relationships in Affective Disorders," Alexithymia: A View of the Psychosomatic Process," and "Hypnosis–Auto-Hypnosis in Treating Psychosomatic Illness."

Both the providing and the receiving physician in liaison psychiatry should find this book attractive. Have it on your bookshelf as a reference for your particular interests as well as for a broader awareness of the psychosomatic approach and its increasing possibilities in patient management and education of the patient in a more healthy self-management program.

PHILIP C. ROND, M.D.
Tallahassee, Fla.

The Powerholders, *by David Kipnis. Chicago, Ill., University of Chicago Press, 1976, 227 pp., $12.50.*

This small book deals with a subject that has occupied much of our interest since the Nixon administration made us realize how close we are to forgetting that ours should be a government of laws, not of men. If our leaders can, within the limits of our systems, manipulate and control us (the author calls us "target persons") it is high time we considered their motivations and the devices that can be used for this purpose.

Dr. Kipnis feels that motivation for the exercise of power may be irrational, derived from a sense of weakness, an absence of love, feelings of inferiority, or continual anxiety. On the other hand, it may be simply role behavior. This type of power motivation seems to be distinguished by the feeling of being a pawn in a situation with considerable distance from the target person and a feeling that one is only doing one's duty. This rationalization was prominent in the Nazis who conscientiously manned the ovens in the concentration camps; unfortunately, it is also a concept used by military personnel in our own country who say they were following orders and were therefore not personally responsible. The institution provides absolution for behavior that might seem unacceptable. A third type of power motivation is described as "instrumental behavior," the idea that "induction of behavior in others can be instrumental in obtaining rewards for oneself."

There is considerable discussion of the use of rewards and coercive devices, with some evidence that rewards can be elicited if there is a sufficient number of noncompliant colleagues and apple-polishing behavior thus stands out. Under these circumstances, one is tempted to wonder where the control is placed. In a slave-master relationship the slave often exerts as much "control" as the master; there is also a time-honored joke that laboratory rats feel, perhaps with some justification, that after many trials they have taught the experimenter to provide them with rewards.

It is disturbing to be reminded of Milgram's findings in an experimental situation that most persons carried out the experimenter's order to hurt another person. Kelman and Lawrence found that 67% of a cross-section of American people said that most people would follow orders and shoot "all inhabitants of a Vietnamese village . . . including old men, women and children." Further, a study by Zimbardo involving volunteers who alternated between acting as prisoners and guards revealed that the prison guards "acted in an increasingly brutal manner and . . . reported that they enjoyed the master-slave relationships that were evolving."

It is interesting to note that Kenneth B. Clark, in a presidential address to the American Psychological Association in 1972, suggested a "biochemical pacifier" that would be a required part of the diet of world leaders and "stabilize and make dominant [their] moral and ethical propensities. . . ." This is "making the world better through chemistry" in the best possible way.

The book presents an interesting array of philosophical and experimental approaches to the problem of understanding power in many of its attributes and describes an interesting gallery of power-wielding people, including Hitler, Machiavelli, and Iago.

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Santa Barbara, Calif.

Children's Rights and the Mental Health Professions, *edited by Gerald P. Koocher, Ph.D. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1976, 246 pp., $17.95.*

In a series of short essays, the majority of which are written by psychologists and a few by lawyers, this book deals with four facets of children's rights in the mental health field. It raises questions about individual professional responsibility toward child clients (i.e., telling children the results of clinical evaluations) and agency or institutional responsibility (confidentiality of children's health records). It deals with the responsibility of the mental health professions in broad social issues concerning children (the role of professionals in child-custody disputes) and, finally, discusses the legal rights of children who become involved in mental health treatment or evaluation. (Should hearings be required before children can be hospitalized for mental problems?)

The essays cover a wide terrain, ranging from the ethical problems of using children as research subjects and the dangers of overdrugging "hyperactive" children to misuse of culturally biased intelligence tests and the overintervention of state agencies into family autonomy under the rubric of neglect-and-abuse laws. They touch on virtually all of the major problems in the area of children's mental health that have surfaced over the past five years. The result is a useful overview for consciousness-raising and, I think, for training purposes.

The individual chapters are, however, uneven. The critiques and conclusions sometimes seem too pat for the complexities of the issues, and the viewpoints reflected are widely disparate: the traditional helping therapist viewing his or her own profession, the skeptical mental health worker looking at the results of legal efforts to establish children's due process rights, and the disenchanted attorney attacking the medical model underpinnings of the juvenile court. Any one of the 20-odd chapters merits a book by itself (books have indeed been written about most of these topics) to probe the subject matter adequately from an experimental point of view and to survey thoughtfully the options for doing better by children.

To me, the most provocative single issue (raised in different aspects by different authors) is the pervasiveness of institutionalized obstacles to what all mental health profes-

sionals and lawyers must accept as a common goal: sensitive, individualized, protective, nonstigmatizing treatment of troubled children and families. The following examples illustrate this point:

1. Children referred for mental evaluation or treatment are often only one piece of a pathological or dysfunctional family puzzle. There is no reason to single out the child for labeling as the "sick" one. However, private and public health insurers invariably insist that individual diagnoses be made of the child's psychiatric disorder before they will pay for the treatment; family problems are noncompensable. The no-fault principle of auto insurance surely deserves consideration in mental health insurance as well.

2. Training for mental health professionals needs revamping. Several authors point out that clinicians in training are generally exposed to children as cases, not as children, and, like sentencing judges, they rarely if ever get feedback on the results of their therapy; that according to one survey only 13% of medical researchers had even one training lecture on the ethics of research with human subjects; and that courses in pharmacology rarely take the different physiologies of children into account. Then there are the perennial conflicts of training institutions: client service must vie with training opportunities in admissions, treatment choices, and even in the content of patient records. Patients' interests are in the least amount of recorded data commensurate with treatment needs, but the training needs of students include practice in recording their observations and evaluations for supervisory reaction. One answer is two sets of records—the bareboned official one for necessary inspections by third-party payers or investigators and the therapist's private working papers, which, one hopes, would be unsubpoenable.

3. Too many of the mental health professionals who deal with children in agencies, schools, and juvenile courts engage in assessment only; they bear no responsibility for linking assessments to services. Children are thus too often labeled to no avail. Packaging evaluation and treating or at least service-brokering skills in the same people or job slots seems an overdue reform.

The path of a child advocate in such institutional arrangements is a treacherous one. On the other hand, lawyers who represent children as clients have an easy time; their single-minded loyalty to the child is officially sanctioned by their profession. Mental health professionals who opt for their perception of children's rights over their employer-institution's perceived needs may be courting personal career disaster. All of us have seen such cases.

Lawyers welcome tracts like this as an admonition to mental health professionals to protect their young clients. However, while they realize full well that enlightened and sensitive professionals are more likely than lawsuits to help more troubled youngsters in the long run, lawyers also recognize that until some of these basic institutional problems are solved their own abrasive, often exacerbating presence in the mental health field must continue.

PATRICIA M. WALD
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Marital and Family Therapy, *by Ira D. Glick, M.D., and David R. Kessler, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1974, 172 pp., $14.00.*

Drs. Glick and Kessler have presented a well-organized and very readable introductory text in a field that has grown so rapidly in the past decade that the beginner is often unable to know how to begin to understand the concepts and practices that have emerged. They start with a clear, concise definition of the field and include just enough of a historical perspective to enable the reader to follow the developments that have occurred. They continue with a discussion of family systems and the life cycle that elucidates concepts of family interaction and tasks with important emphases on marital and parenting coalitions and a consideration of generational boundaries. They then consider dysfunctional families.

The authors include a discussion of commonly accepted myths that the beginning therapist, as well as the family, must confront and dispel if effective therapeutic work is to be done. The sections on treatment raise many important questions, such as who should be included in family therapy sessions, what therapist combinations are advantageous and disadvantageous, and what general indications and contraindications exist. The authors present an excellent overview of specific treatment strategies and then briefly but effectively tackle the difficult area of family treatment when the identified patient has a specific psychiatric diagnosis. The appendix material on the family interview will be valuable for the beginning therapist and reinforcing for those who do only a small amount of work in this area.

Glick and Kessler have provided a clearly focused and well-balanced view of an unsettled field. They communicate with the reader, using themselves as educational facilitators in the same way that they encourage the therapist to use himself or herself in the therapeutic process. They see the family therapist as an active participant, as a role model, and as an educator to the family. This book should be required reading for all beginning family therapists.

CAROL C. NADELSON, M.D.
Boston, Mass.

The Frontier of Brief Psychotherapy: An Example of the Convergence of Research and Clinical Practice, *by David H. Malan, D.M. New York, N.Y., Plenum Medical Book Co., 1976, 358 pp., $19.95.*

The frontiers of brief psychotherapy are often invaded by psychological barbarians. Fortunately for the psychotherapeutic community, Dr. Malan is a stalwart settler in that territory. His work is a firm bulwark and constitutes a major step in the effective application of well-established psychoanalytic and psychodynamic principles to this still underdeveloped but vastly important area.

The book indicates the social necessity, the theoretical foundation, and the clinical feasibility of brief psychotherapy. The accomplishments of pioneers such as Franz Alexander, Thomas French, and Michael Balint are duly acknowledged; among more recent contributions, the work of Peter Sifneos is emphasized.

Psychodynamic formulation, focal conflict, motivation for treatment, and response to interpretation must be determined quickly and accurately in order to plan whether and what kind of brief therapy should be employed. A clear discussion of these considerations is followed by the presentation of a number of cases that provide a detailed picture of the actual therapeutic interventions and patient responses. The powerful emotional forces generated in brief therapy are stunningly evident, and the therapeutic benefits follow with impressive regularity. Dr. Malan, in his even-handed way, also heeds the dangerous potentials inherent in

this very rapid, fluid, and relentless process. Errors in diagnosis, assessment of suitability, and therapeutic technique are freely—and refreshingly—acknowledged. This renders all the more persuasive the author's strong conviction that brief psychotherapy has a firm conceptual basis that can be studied, taught, and learned. The appropriateness of brief psychotherapy for selected cases is made compellingly clear throughout the book. Brief psychotherapy should not be considered the stepchild of psychiatry, reserved for the stepchildren of society.

The book is replete with clinical wisdom and conceptual stimulation; therapists of varied backgrounds and experience can find new information pertinent to their specific needs. It could serve as an excellent text for courses in psychotherapy for psychiatric residents, clinical psychology trainees, and students of psychiatric social work. For seasoned analysts and therapists, the book, especially with its follow-up reports, will serve as a touching reminder of the many patients seen for a brief time or seen on a few occasions over a long period of time whose lives were greatly enriched by a brief contact with psychotherapy. Such cases often stand out as among the most memorable and mystifying for the therapist. The mystery is reduced by this book.

The problems of this book are few. The author, and the reader, have some difficulty discriminating between a 600-hour psychoanalysis and a 6-hour psychotherapy. At times Dr. Malan seems reluctant to acknowledge that psychoanalysis and brief psychotherapy are essentially different kinds of therapy. Another point of difficulty is the author's use of data from psychotherapy clinics with which he is probably not intimately acquainted. These deficiencies are minor and should not be seriously distracting.

Dr. Malan has made a major contribution to progress in psychodynamic psychiatry by helping to expand the range of systematic psychotherapy and by making the new territory inviting and understandable to his colleagues.

JOSEPH M. NATTERSON, M.D.
Los Angeles, Calif.

New Directions in Sex Research, *edited by Eli A. Rubinstein, Richard Green, and Edward Brecher. New York, N.Y., Plenum Press, 1976, 170 pp., $19.50.*

This volume consists of the proceedings of a conference titled Sex Research: Future Directions, held at the State University of New York at Stony Brook in June 1974. The conference was planned by Stanley F. Yolles, Paul Gebhard, Richard Green, and Eli Rubinstein. The proceedings were first published in the July 1975 issue of *Archives of Sexual Behavior. New Directions in Sex Research* is a hardcover edition of this issue of the journal.

After the opening address by Dr. Yolles, 9 papers were delivered and discussed. Discussants included invited experts. Of 10 authors, only 1 is a psychiatrist, 7 have Ph.D. degrees, 1 is an obstetrician-gynecologist, and 1 is a physiatrist. Topics covered include early sex differences in the human, research strategies in sexual identity, male-female differences in sexual arousal and behavior, evaluation of treatment procedures in heterosexual dysfunction, sexuality and physical disabilities, neuroendocrinology, animal models and problems of human sexuality, research in homosexuality, changing sex roles, and comprehensive sex research centers. In addition, there are discussions of ethical issues and recommendations for future research.

On the whole, the material in this book is well presented. The editing is of high quality, and the discussions that follow each chapter are lively. Psychiatrists not already familiar with the data put forth here will enjoy becoming so. Clinicians will be stimulated not only by the content of Goy and Goldfoot's chapter on the hormonal determinants of bisexuality in lower animals but also by the way these authors think about sexual behavior. All therapists will find the sensitive work of Dr. Theodore M. Cole on spinal cord injury and sexuality of interest.

Although the Stony Brook conference was multidisciplinary, it might have been more so. Little attention was paid to integrating the facts, hypotheses, and models presented with those derived from dynamic psychiatry. Although many aspects of sexual behavior were discussed in depth, the relationship between sexual behavior and unconscious motivation in humans was not. This limitation should not be construed as a fault, however, since all conferences must have boundaries for practical reasons. Perhaps this volume will lead to others in which the movement toward interdisciplinary communication will continue.

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From Group Dynamics to Group Psychoanalysis: Therapeutic Applications of Group Dynamic Understanding, *edited by Morton Kissen. Washington, D.C., Hemisphere Publishing Corp. (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1976, 352 pp., $21.95.*

Any book review reflects the subjective opinions of the reviewer; despite sincere attempts to be as unbiased and objective as possible, many personal elements are involved. This book has an exciting title, a splendidly detailed bibliography, a well-conceived format divided into six major areas, and a conclusion, but it left me with an empty feeling. Why?

In many texts with a cluster of different authors there are bound to be inconsistencies in presentation, style, and ability to present thoughts lucidly. However, in this work there is a remarkable uniformity in the presentations, characterized by two significant elements: an unbelievable use of jargon, which detracts from ideas being discussed (this use not only numbed my brain with such words as "succorance," "identificatory," "elementaristic," "powerfulness," and others but made me wonder why the authors had the need to obfuscate), and a plethora of typographical errors, suggesting a lack of not only appropriate scholarship but downright poor editing. It is my opinion that an author or editor who fails to do a careful job of editing demonstrates carelessness.

Several chapters, however, require additional comments in a more positive vein. "The Work of Wilfred Bion on Groups" by Margaret J. Rioch presents an interesting and fundamental exposition of Bion's theory of group development. This is a readable and succinct paper that clarifies Bion's major themes of basic assumption and work groups. I found "The Pleasure and Reality Principles in Group Process Teaching" by Stephen A. Applebaum in the section titled Freudian Concepts Related to Group Dynamics to be a particularly good review of the dynamics of the group. This chapter is unfortunately marred by typographical errors.

The thesis in George R. Bach's chapter, "Observations on Transference and Object Relations in the Light of Group Dynamics," has as its premise the idea that "the group does *more* to and for its members than the therapist could do for

his patients alone" and elaborates carefully and lucidly on this topic. Louis R. Ormont's contribution, "Group Resistance and the Therapeutic Contract," made me wonder why it took the analyst in this particular case eight months before intervening therapeutically. The patient "opened the session in a mood of deep despair. He was discouraged about his career and homosexual liaisons. He had reached a dead end and was thinking of leaving therapy. The analyst chose this moment to approach the role of the covertly resistant members by underlining how he had been used." (This took eight months.)

A final question is why the editor did not identify the disciplines with which the various authors are associated—psychiatry, psychology, or social work. Was this a not so subtle way of saying that all health professionals are the same? In all fairness to the reader as well as to the authors, I believe appropriate recognition of the authors' fields of practice would have been helpful as well as displaying honesty.

Although it appears my negative comments outweigh the positive ones, I feel this book could serve as a useful volume for individuals undertaking group psychoanalysis. The various carefully annotated bibliographies provide a wealth of additional material.

HOWARD A. WINKLER, M.D.
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The Leader in the Group, *edited by Zanvel A. Liff, Ph.D. New York, N.Y., Jason Aronson, 1975, 315 pp., $15.00.*

This is one of a series of books derived essentially from the Postgraduate Center for Mental Health in New York City. It is dedicated to Alexander Wolf, who was clearly a leader in that he applied psychoanalytic concepts to group psychotherapy before other therapists had ventured into that field.

The first of the book's four sections consists of excerpts from the Dr. Wolf's writings that give the essence of his perspective. These set a theme carried out rather well in the other sections of the book by giving a view of group therapy from inside the head of the therapist. The volume is refreshing in its avoidance of the cookbook-style how-to-do-it enumeration of techniques in favor of providing guiding principles and concepts that give any technique a meaning and use for therapy.

Later sections of the book include the perspectives of American and a few European group psychotherapists with a psychoanalytic orientation. The approach of these authors is often personal, subjective, and even autobiographical. S.H. Foulkes and Emanuel K. Schwartz write perhaps the most informal and personal contributions. For both the book is, most unfortunately, posthumous. Since Manny Schwartz worked so closely with Al Wolf, it is especially fitting that the last entry is a memorial article by Dr. Schwartz.

Theoretical material is presented quite well in a number of articles. Jerome Kosseff produces the longest chapter and deals with the use of object-relations theory in a well-formulated summary for group therapists.

By the end of the book one gets the impression that the title did not quite fit the spirit of the book. A number of the authors in this book and especially Al Wolf show discomfort in dealing with the everyday connotations of the word "leader." If there is a point in avoiding the word "therapist," most of the authors seem more comfortable with the paradigm of parent or even coworker.

For any group therapist seriously interested in the working concepts of his or her field, this is an excellent resource.

JAY W. FIDLER, M.D.
Plainfield, N.J.

Chronically Ill and Handicapped Children: Their Management and Rehabilitation, *by Thomas L. Creer and Walter P. Christian. Champaign, Ill., Research Press Co., 1976, 183 pp. $7.95 (paper).*

In their preface the authors indicate that chronically ill children require a considerable amount of time and attention by a large number of social agents, including physicians, teachers, psychologists, nurses, social workers, and child care workers. Both authors have worked at the National Asthma Center in Denver, Colo., and have done research on social learning principles to rehabilitate chronically ill children. They give case reports of their work with asthmatic children as well as with those suffering from cerebral palsy, mental retardation, and epilepsy. They also provide clinical examples of the various forms of therapy they have used, which include respondent conditioning, operant conditioning, and weakening and extinction of response.

In their many case examples the authors emphasize the need for therapists to work with parents, with the diverse disciplines that deal with chronically ill and handicapped children, and with boards of directors of institutions for these children. They describe their own success using behavior modification, self-monitoring, relaxation training, and systematic desensitization by reciprocal inhibition; they also include 197 references to other works.

This book is highly recommended for professionals dealing with chronically ill and handicapped children.

FRANK J. CURRAN, M.D.
New York, N.Y.

Alcohol Control Policies in Public Health Perspective. Finnish Foundation for Alcohol Studies Vol. 25, *by Kettil Bruun, Griffith Edwards, Martti Lumio, Klaus Mäkelä, Lynn Pan, Robert E. Popham, Robin Room, Wolfgang Schmidt, Ole-Jørgen Skog, Pekka Sulkunen, and Esa Österberg. Helsinki, Finland, Finnish Foundation for Alcohol Studies (New Brunswick, N.J., Rutgers University Center of Alcohol Studies, distributor), 1976, 106 pp., $8.00.*

In this brief, tightly constructed, well-referenced study of public health aspects of alcohol consumption in various societies of the industrial world (primarily Europe and the Americas), the authors propose and then set out to demonstrate the following proposition: "Changes in the *overall* consumption of alcohol beverages have a bearing on the health of the people in any society. Alcohol control measures can be used to limit consumption; thus, control of alcohol availability becomes a public health issue." The method used is primarily statistical; the authors restrict themselves to a presentation of long-term physical health damages. Mental health and sociological considerations are purposely excluded, and there is no emphasis on the clinical aspects of alcoholism as a disease process. The authors are careful to point out that these exclusions are not intended to negate the significance of these areas in considerations of individual health.

As chapter builds on chapter, an impressive array of statistics buttresses the authors' conclusions. Although I have a natural suspicion of conclusions drawn from statistical evidence alone, the data used are indeed impressive and will give anyone pause for thought, even the statistical skeptic. Some of the conclusions drawn from the data are as follows:

1. Heavy users of alcohol have a substantially increased risk of morbidity and premature death.
2. The prevalence of heavy users, however defined, is directly correlated with the per capita consumption of alcohol regardless of drinking customs or other variables, such as acceptance or nonacceptance of drunkenness in a society.
3. The overall trend in alcohol consumption throughout the industrial world has shown a remarkable increase since World War II, particularly during the 1960s.
4. The degree of overall availability of alcohol has a direct effect on the general level of alcohol consumption in a society. Therefore, alcohol control measures can be used to control consumption and, by inference, morbidity and mortality.

There is little in this book to interest the clinician who deals exclusively in the diagnosis and treatment of alcoholism. However, some of the clinician's traditional beliefs are challenged by the data presented in this book. For example, according to the data the total consumption of alcohol is distributed in a population in a manner that is fairly stable from country to country. Cultural differences and social norms do not seem to result in significant differences in this distribution. Therefore, the prevalence of heavy users in any society would be directly proportional to the per capita consumption of alcohol. This would mean that countries with the highest per capita consumption, namely the wine-drinking countries of France, Italy, Portugal, Spain, and Switzerland, will have the highest prevalence of heavy users; countries that are primarily spirit- or beer-drinking, such as the United States, Sweden, and Ireland (all of which are considered to have a high percentage of alcoholism), are lower on the list.

Although the authors disclaim that they advocate stricter control policies, their implicit suggestion is that such policies would be good from a public health point of view. They mention that such controls can involve considerable costs, which may outweigh their benefits, but it seems to me that insufficient attention has been directed to the wider social consequences that have always attended extreme control measures, namely, illicit production and trade of alcohol, criminality, and the use of other substances, which occurred during Prohibition in the United States. Of course, it was not the authors' intention to stray from their limited path to focus on these other consequences. They are aware that the area of alcohol control policies can be highly controversial; they issue caveats to potential reformers as well as to others to debate the evidence objectively.

A very useful section of the book deals with the amount of alcohol consumption that is hazardous to physical health. Although no definite conclusions can be drawn from the data, it seems that the lowest level of chronic alcohol consumption that constitutes a significant hazard to health is almost certainly less than 120 g (5 ounces) of absolute alcohol per day and possibly less than 60 g.

All in all, the book is readable, interesting, challenging to some of my myths, and provides a wealth of statistical data as well as 150 references.

JOHN H. SAYERS, M.D.
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The Scientific Study of Marihuana, *edited by Ernest L. Abel, Ph.D. Chicago, Ill., Nelson-Hall, 1976, 299 pp., $12.50.*

This volume is a collection of papers of considerable variability in content, purpose, and scientific quality and design. The 31 papers are divided into sections, each of which has an introductory comment by the editor. Except for the excerpts from "Les Paradis Artificiels" by Charles Baudelaire, written in the last century, the papers reprinted were all published between 1967 and 1972. The 4-page introduction gives an outline history of the knowledge about the use of marijuana and the recent spread of its use and effect, particularly on young individuals. In this introduction the editor states,

> The collection of scientific reports contained in this volume represents some of the most recent findings in the area of marijuana research. Hitherto, much of this material has been buried in scientific journals, generally inaccessible to the non-professional. The purpose of this book is to make some of this material available to all interested readers, laymen and professionals alike.

The introduction also states that "socially relevant questions" that cannot be feasibly or ethically studied in humans have been observed in animals while under the influence of cannabis or its derivatives. The editor recognizes that the dosage administered to subhuman species is often 100 or more times greater than that used by humans and that generalizations from such studies therefore become problematical. In concluding the introduction, the editor says,

> The topics and articles that are included here have been selected because to me, at least, they represent some of the more socially relevant approaches to cannabis research taken by various investigators. In the conduct of some of these studies, I have had a personal involvement. My selection of material may, of course, be regarded by some readers as a biased one. What is of interest or importance to one individual is not necessarily of interest or importance to another. Still I trust that my choice of material has been diverse enough so that anyone who is interested in knowing about the effects of cannabis will find at least one topic that piques his curiosity.

The topics that I found most interesting and/or most satisfactorily presented were the excerpts from Baudelaire; "Marijuana Intoxication: Common Experiences" by C.T. Tart; "Marijuana Effects: A Survey of Regular Users" by James A. Halikas, Donald W. Goodwin, and S.B. Guze; "Adverse Reactions to Marijuana" by M.H. Keeler; "Marijuana Use in Psychiatric Illness" by J.A. Halikas, Donald W. Goodwin, and S.B. Guze; and "Marijuana in Man: Three Years Later" by L.E. Hollister.

The editorial comments preceding each section summarize the effect of the reviewed articles on the editor. I did not think that the editorial comments and introduction together served adequately to synthesize a position statement regarding marijuana from a clinician's point of view. The material gathered leaves the reader with the same sense of inconclusiveness, indecision, and equivocation that has been produced by most of the recent marijuana studies, reports, and "anthologies."

Inclusion of bibliographical references with the reprinted original papers is a useful feature of this volume. In my opinion, the presentation of everyday clinical problems associat-

ed with marijuana effects is inadequate. The sophisticated student will find no new material presented in this volume. The unsophisticated lay person, however intelligent, would not derive a solid foundation of knowledge of the subject from this volume alone. This is not unusual with editorial collections of other people's work.

DOUGLAS GOLDMAN, M.D.
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Lithium in Psychiatry: A Synopsis, *edited by A. Villeneuve, M.D. Quebec, Canada, Les Presses de l'Université Laval, 1976, 205 pp., $10.00 (paper).*

This collection of papers represents the proceedings of the First Canadian International Symposium on Lithium, held in May 1974. In the main, the papers are clinically oriented. Those which discuss the biochemical and physiological properties of lithium do so with an eye toward clinical implications and clues to etiology of affective illness. The thread of clinical pragmatism is evident throughout the authors' interpretations and summaries of the most careful and relevant work in lithium therapy.

Since lithium is being used for conditions ranging from hangnails to hangovers, one welcomes Mogens Schou's authoritative evaluation of its use in a wide variety of seemingly unrelated psychiatric and nonpsychiatric illnesses. His assessment of the prophylactic uses of lithium is particularly helpful. Angst and Grof's summary of the findings of a cooperative international study of the course of unipolar depressions and bipolar psychoses also helps resolve the "who" and "how long" of prophylaxis. Papers describing the psychological problems of lithium use and the instruction of patients, nurses, and primary care physicians are timely. Next to poor patient selection, inadequate education of lithium patients and front-line personnel with subsequent failure of motivation and monitoring is probably the greatest cause of lithium failure.

The most interesting paper in my opinion is Goodwin, Wehr, and Sack's summary of the studies of the action of lithium in man. These authors take the reader through a brief but remarkably clear discussion of affective disorders, giving particular attention to the information gleaned from CSF-probenecid studies.

This book is a compilation of a number of good papers; some are already dated, but all are readable and instructive. With contributors coming from different centers and discussing the same topic with basically the same emphasis, there is an unavoidable amount of duplication and repetition. For the lithium neophyte this repetition is useful; for the more experienced this book warrants perusal for areas of special interest.

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Hyperactivity: Research, Theory, and Action, *by Dorothea M. Ross and Sheila A. Ross. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1976, 365 pp., $18.95.*

Only unquibbling praise is deserved by a book so complete and critical in its review of the literature on hyperactivity. The two courageous women who wrote the book are mythoclastic and iconoclastic in the extreme, as the status of the subject of hyperactivity requires. They discredit the viewpoints often held by psychiatrists, pediatricians, and behavioral scientists that tend to mystify the topic of the hyperactive child. They contend, on the basis of logic and empirical study, that hyperactivity is not synonymous with minimal brain damage (MBD), and they decry the sloppy formulations that have equated hyperactivity with MBD or with major and clinically demonstrable brain damage. Very few hyperactive children, they say (correctly, according to my own experience), suffer from any soft or hard CNS disorder. That hyperactivity is a typically neurological sign or symptom is one more myth to lay to waste. Only rarely does a hyperactive child suffer from MBD; the child who does needs a specially adapted and appropriate combination of treatment modalities that have proven helpful.

Another myth that the authors go after is that hyperactivity is mainly part of the experience of the child in middle childhood, or "latency." They show that hyperactivity is an affliction of fetuses in the last trimester of gestation, infants, preschool children, adolescents, and adults as well as elementary school children. They also show that untreated or improperly treated, a hyperactive infant often goes on into adult life with the picture of hyperactivity; there are added interactional effects that help to comprise the changing picture during different ages, but the core problems persist throughout the life span.

The Rosses also discard the myth that pemoline, methylphenidate, amphetamines, and other psychic energizers have a paradoxical effect on the hyperactive person. Further, they toss overboard the idea that these energizers improve all types of learning and lament that the more useful types of human learning are not enhanced by methylphenidate and its stimulating substitutes. They hold that intentional learning is retarded when a child is given stimulants. If incidental learning (which probably constitutes 80% of all learning by a child) is diminished, the child does not overhear valuable cues and is not aware of mannerisms, vocal nuances, and social amenities unless all of these are explicitly, formally laid out. It does not facilitate a child's gracefulness, peer acceptability, formation of friendships, or self-esteem if his or her intentional learning is improved at the expense of informal, incidental social learning. Drug therapy soothes parents and teachers into thinking that all causation resides within the child and that the adults need do nothing further.

The Rosses take a jab at another popular formulation, which views hyperactivity as principally an attention deficit. They specify that in reality the problem of the hyperactive child's attention decrement is not generic and total because in some aspects of attention and attentiveness the hyperactive child is not distinguishable from the normal child. Therefore, another researchable formulation seems rejectable.

With some gusto, I felt, the authors debunk the myth of the Strauss-Lehtinen-Kephart method of teaching hyperactive children; they point out flaws in design, method, data collection, and interpretation. Their conclusions cast serious doubt on the belief that distractibility is paramount in the problems felt and shown by the hyperactive child.

In a book that surveys and critiques research with hyperactive children it is rare to find so much attention given to the children's perspective, their own ideas about their dysfunctions and remedies. Particularly touching is the authors' repeated emphasis on the possible long-range effects, not all salutary, of the psychic energizers. Long-range drug effects haunt the Food and Drug Administration and physicians less

than they haunt the children, one suspects.

With all these good things to say about authors who make so many incisive and illuminating comments about conventional shibboleths and bandwagons, I hesitate to take exception, but I do in regard to their antimedical bias. I feel they have an outspoken and familiar antimedical agenda, e.g.:

> It assigns a coordinating role to the psychologist rather than to the pediatrician on the team; it focuses on prevention at both the professional *and* parent level; and it assigns much of the responsibility for teaching behavioral control principles to the school nurse. (p. 301)

That quotation is not out of context. The authors give no sign of recognition to psychiatry or child psychiatry, put down pediatrics, and vaunt psychology for coordination and nursing for teaching of behavioral control principles.

When they stick to research, Dorothea and Sheila Ross are brilliant. Their chapter on etiology is excellent; it is so thorough that it notes every conceivable etiological circumstance, whether sensible or nonsensical, whether pertaining to genes and temperament or to food additives and fluorescent lighting. When they turn to remedial and clinical work, however, the authors lack the luster that is otherwise very much in evidence. It is as a compendium of hyperactivity research that this volume is an indispensable and vital contribution to child psychiatry.

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Child Abuse and Neglect: The Family and the Community, *edited by Ray E. Helfer and C. Henry Kempe. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1976, 426 pp., $20.00.*

Two earlier volumes edited by Drs. Helfer and Kempe, *The Battered Child* (1) and *Helping the Battered Child and His Family* (2), are part of the basic literature in child abuse and neglect. The present volume will become part of that literature. The title cannot describe the diversity of important information in each of the six sections of the book.

In section 1, Dysfunction in Family Interaction, Steele writes in depth on "Violence Within the Family." He describes the complex biological, psychological, social, and cultural factors involved, concluding that violence breeds violence. Kennell, Voos, and Klaus present their studies in early parent-infant bonding and emphasize the significance of bonding from pregnancy on. Helfer, McKinney, and Ruth Kempe write about the arrest of developmental processes in childhood in pathological parent-child interactions. In "Family Pathology As Seen in England," Ounsted and Lynch illustrate 18 types of family pathology and the indicated therapy.

Section 2 contains four chapters on Evaluating the Family. The first two contributions discuss in detail how the child protection team functions and how family pathology is assessed. The second two chapters deal with sexual exploitation and incest. In "Humanistic Treatment of Father-Daughter Incest," Giarretto writes from experience with more than 250 families and emphasizes distorted family homeostasis. Section 3, Family Oriented Therapy, illustrates the need for a comprehensive multimodality interdisciplinary approach. The various authors elaborate on the details of the treatment process and indicate where the pitfalls in treatment can occur. Lynch and Ounsted again give their experience with families in residential therapy in England, and Jeffery describes in detail a behavior therapy approach used in Australia to modify parents' interaction with their children.

Section 4, The Community, consists of three chapters that are clear expositions on community organization in civilian and military life. Community organization, personnel, education, administration, financing, and many other aspects of programming receive attention. This section is valuable in that the authors, all of whom have considerable experience, illustrate the complexities of community organization and the need to persevere. Section 5, The Family and the Law, consists of contributions by authors with extensive experience with the legal aspects of abuse and neglect. Brian Fraser, an attorney, writes on "The Child and His Parents: A Delicate Balance of Rights," and Judge James Delaney addresses himself to "New Concepts of the Family Court." Section 6, Early Recognition and Prevention of Potential Problems in Family Interaction, has three chapters on assessment and prediction. The studies focus on mothers during the prenatal and perinatal periods. The methodology, protocols, and results are given, with emphasis on the fact that the research must be validated. There are extensive references for each chapter at the back of the book.

This volume presents a comprehensive view of what we currently know about child abuse and neglect. A practitioner familiar with the field would find this book to be a good review with additional studies in research areas. A psychiatrist or other professional with limited or no experience in abuse and neglect could do no better than to read this book with its wealth of basic information.

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2. Kempe CH, Helfer RE (eds): Helping the Battered Child and His Family. Philadelphia, JB Lippincott Co, 1972

JOSEPH FISCHHOFF, M.D.
Detroit, Mich.

Basic Child Psychiatry, 2nd ed., *by Philip Barker, M.B., D.P.M., D.C.H. Baltimore, Md., University Park Press, 1976, 268 pp., $14.50.*

Those familiar with the 1971 edition of Dr. Barker's primer of child psychiatry will see that he has not only brought the text up to date but also improved the presentation of most of the sections. The result is a book reflecting an even greater breadth and maturity of clinical judgment than the first edition. Dr. Barker's eclectic approach struck some reviewers in 1971 as rather bold in that he included such items as emphasis on limit setting and use of behavior modification techniques for enuresis. Clinical eclecticism has now become more acceptable in child psychiatry; Dr. Barker's balanced approach does not stand out from the accepted approaches as much today as it did in 1971.

A unique contribution in the new edition is a chapter on the legal aspects of child psychiatry, including a very nicely written description of how to examine and how to write reports on children and adolescents for the court. In this volume there is also a greater emphasis on family group therapy as well as a new chapter on mental handicaps (the new British term for the mental retardation syndromes). In the 1971

edition references to mental handicaps were scattered among several chapters.

Even though the book is for beginners it is sufficiently comprehensive to provide a certain amount of depth. The author provides some interesting and appropriate case histories as well as selected references that seem quite useful. A glossary of treatment terms is appended for those unfamiliar with medical or psychiatric terminology. A listing of selected books and journals in the field of child psychiatry that was included in the first edition has wisely been omitted because it is difficult to keep such a list up to date.

The good qualities of the first edition have been retained, including the sections on normal development, child psychiatric disorders, and methods of assessing children and their families. The language is clear; the sections on diagnostic categories are elementary. Although the sections on family therapy are an improvement over the previous edition, they present only a simplistic introduction to this area. The section on psychological testing briefly describes specific tests in both clinical and educational areas. The section on adolescence has been expanded.

The two concluding chapters on treatment and prevention of child psychiatric disorders will be most useful to the beginner in their descriptions of the many modalities of therapy. In contrast to other elementary textbooks, *Basic Child Psychiatry* is aimed at the neophyte therapist in some very fundamental ways, such as listing five principles of psychotherapy with young children (i.e., never criticizing a child, bearing in mind that children do not come for treatment of their own accord, not plunging into straight discussion of symptoms, trying to understand the child's feelings and world as he or she sees it, and, finally, remembering that limits have to be set). Similarly basic are the descriptions of casework help for the family, family group therapy, the brief section on behavior therapy, and the practical description of the use of the psychoactive drugs and anticonvulsants.

The author can be criticized for using in an elementary text the 1969 World Health Organization proposal of a "triaxial diagnostic system"; at least on this side of the Atlantic the system has not yet found wide acceptance. There are for the most part no other serious problems for the American reader who is willing to skip over specifics about the British statutes and service and delivery systems.

In general this is a worthy revision of a fundamental textbook that could be very useful to beginning medical students, social workers, pediatric residents, psychiatric residents, and beginning residents in child psychiatry.

PAUL N. GRAFFAGNINO, M.D.
Hartford, Conn.

Child Personality and Psychopathology: Current Topics, Vol. 3, *edited by Anthony Davids, Ph.D. New York, N.Y., John Wiley & Sons, 1976, 294 pp., $18.95.*

This is the third volume of papers by a number of authors on the most current material in various areas of child development. Part 1 is concerned with personality development, part 2 with sexual development, and part 3 with psychophysiological and behavior disorders.

Part 1 is composed of two interesting chapters on the changing patterns of child rearing in alternative life-styles, including divorced parents and communal living. It also has a chapter on imaginative play and pretending. In part 2 the authors discuss juvenile gender identity, masculinity and femininity during boyhood, and homosexual orientation and its antecedents. Part 3 contains a chapter on hyperactivity, a chapter on central and autonomic system function in the hyperactive syndrome with a discussion of treatment and research implications, and a chapter on the neuropsychological approach to causation, prevention, and treatment of delinquency.

Depending on the reader and how much depth he or she wishes to go into, this book varies from chapter to chapter quite markedly. The preface and introduction are written in straightforward, easily understood language, but many of the following chapters are esoteric, and one could say there are more "we don't knows" than there are proofs of what we do know. There is a good deal of neurophysiology and biochemistry and very little dynamic psychiatry; however, a great deal is being learned in these areas that we had not recognized before.

This is a book well worth reading; it can be recommended not only to those in child psychiatry but to those who treat adult patients as well. It would be a valuable source on many subjects not well known by the average psychiatrist in training.

STUART M. FINCH, M.D.
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Handbook of Small Group Research, 2nd ed., *by A. Paul Hare. New York, N.Y., Free Press (Macmillan Publishing Co.), 1976, 743 pp., $19.95.*

The author defines a small group as

> any number of persons engaged in interaction with each other in a single face to face meeting or series of meetings, in which each member receives some impression or perception of each other member distinct enough so that he can, either at the time or in later questioning, give some reaction to each of the others as an individual person, even though it be only to recall that the other person was present.

This is what the book is all about.

This second edition has the same goal as the first edition, published in 1962: to make available to students, researchers, and practitioners a catalogue of the field together with some suggestions of principles by which it may be organized. There are some differences between editions that add to the book, such as the addition of separate chapters on group development and social perception and suggestions for laboratory study of the history of small group research and sensitivity training. The 1,385 references to the literature from the 1962 edition are included along with an additional bibliography of relevant articles up to 1974, making a total of 6,037 references. This provides an essential resource for sociologists, psychologists, social workers, group workers, teachers, and researchers in general.

The exhaustive review of the literature is well organized in the three parts of the book. The first section considers essential tendencies of the interaction process and group structure. Here the elements of social interaction are introduced and later elaborated. This is followed by a review of the research on the process through which culture develops, persists, and changes. The next chapter considers the interaction process as group members solve problems through discussion. This emphasis is continued in chapter 4 with a

description of the phases a group passes through in its development during the course of a single meeting and over a series of meetings. The author completes his first section by outlining the details of social perception, the role system of the group, and, finally, the network of interpersonal choice.

In part 2 the author reviews some of the factors that can be varied to produce changes in the group norm, the interaction process, the role system, and the pattern of interpersonal choice. These factors are the personalities and social characteristics of members, group size, task, communication network, and leadership.

Part 3 is a summary of the literature from the point of view of productivity. The questions considered are When is a group more productive than an individual? and What are the characteristics of the most productive groups? The four appendices provide specialized information for persons involved in teaching, research, or application.

A further example of the author's thoroughness and thoughtfulness is the abundant use of diagrams and charts to help clarify and illustrate the context of this text. Hare illustrates his clear thinking and writing by neatly summarizing the essence of each chapter in a page or two. For those who wish to go farther, he provides additional references to texts and works that cover the major contributions to the chapter theme. I found that reading the summary and then the text of the chapter made the volume much easier to understand.

Although the book was not meant to be a text in group psychotherapy for the practitioner, it becomes clear from the first page on that this is a text on something as basic as anatomy, physiology, and chemistry are to the practice of medicine. Understanding the content is bound to enrich the reader because, unlike the authors of most reference texts, throughout his book Hare projects the feeling that he is dealing with people. He never loses sight of the fact that to understand the group one must understand the person. This book is a must for those involved in small group research or group therapy.

BARNEY M. DLIN, M.D.
Philadelphia, Pa.

Adolescent Coping, *by Alice E. Moriarty, Ph.D., and Povl W. Toussieng, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1976, 212 pp., $14.50.*

The midwest, unlike the east, is blessed with researchers on normal adolescent development. First there was Offer and now there are Moriarty and Toussieng, who report their findings in 54 adolescents who have been studied and reported on since early childhood as part of a project at the Menninger Clinic directed by Lois Barclay Murphy.

Each of the 54 subjects had a three-hour psychiatric interview and a series of psychological tests in his/her senior year of high school. The parents were not seen, and the researchers made no effort to press for data on sex.

The authors divide their subjects into two groups on the basis of whether the adolescent denied his or her sensory experience in forming a value system or used his or her sensory system to develop a value system. The former, who were in the minority of the total group, were called "censors"; these adolescents restricted their perceptions to support their value system. The latter, who were in the majority, were called "sensers"; they sought new perceptions to alter their values. These two groups differed in many ways, such as the sources of their self-respect and their object relations.

The authors caution against generalizations from their small sample, but they found that the censors tended to have controlling mothers and distant fathers, while the sensors had close relationships with their mothers and fathers, who were much involved in their upbringing (shades of Mahler's finding that fathers represent an important bridge to reality and individuation in the earlier separation-individuation phase of development).

Despite the fact that the parents raised these adolescents according to conservative standards, 70% of the adolescents adopted nontraditional views without the kind of rebellion seen in the 1960s—i.e., they changed themselves but did not find it necessary to try to change anyone or anything else. Their tolerance of differences made it unnecessary to rebel against adults.

The authors' style of writing is clear, and their many excellent quotations from the interviews really bring the adolescents and their struggles to life. The findings both complement and supplement those of Offer, who studied normal male adolescents. The limited sample—a white conservative middle-class farm community—makes generalizations difficult, however. For example, I cannot imagine an eastern urban adolescent being so uninfluenced by television as these rural subjects were. Further, it is unfortunate that the authors refrained from pressing for sexual data—is not sex the strongest force with which the adolescent must cope?

In the last chapter the authors spell out the implications of their findings for parents, teachers, and colleagues by revealing the new points of view they developed as a result of their findings. This is a surprising chapter in an otherwise sophisticated book because the authors report as new—at least to them—points of view that, to my knowledge, have been commonplace beliefs in developmental and clinical work with adolescents for at least 25 years. The following statements provide examples: "Wish for change must not automatically be viewed as being synonymous with rebellion or a wish for revolution." "Children can and will take responsibility for their own fulfillment, if given the chance." "Parents too will be better off if they seek the sources of their own fulfillment within themselves." "They respected parents and other adults who really lived up to what they believed."

How is one to explain the paradox of clinical sophistication combined with a naïve perspective? Could it be that the authors changed from censors to sensers as a result of their work? If so, it raises questions about their objectivity in using this particular axis for analysis.

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The Intensive Group Experience, *by Max Rosenbaum and Alvin Snadowsky. New York, N.Y., Free Press (Macmillan Publishing Co.), 1976, 205 pp., $13.95.*

From the title I expected this would be yet another textbook-sized volume about group psychotherapy. I was therefore both surprised and pleased to find a slim book consisting of five separate chapters in easily readable print. The contributions are on vastly different aspects of group dynamics, which is partially explained by the editors' stated intention of providing a "consumer's guide" to the group experience. In fact, only Rosenbaum, in the first chapter on group psychotherapy, provides the kind of professional judgment about the variety of group experiences that is promised by

the concept of a consumer guide. In general the contributions tend toward the descriptive. The authors give detailed descriptions of a type of group experience, its historical development, and specific examples from that group process but do little in the way of either critical evaluation or appraisal of how individual psychopathology is affected in each of the groups described. The reader is treated to a first-rate review of the history of the development of group psychotherapy in the first chapter. Unfortunately, this history is repeated by subsequent contributors, although in somewhat altered forms.

Although Rosenbaum's opening chapter has some of the characteristics of a guide for the potential group therapy patient or group experience consumer, the remaining chapters often take contradictory positions about various group therapies and therapists. For instance, Rosenbaum is quite skeptical about Perls and his Gestalt therapy. In contrast, Giges and Rosenfeld describe Perls as an extraordinarily gifted therapist whose contributions are at the center of the personal growth-encounter movement.

Rosenbaum ignores any consideration of the limitations of group therapy in dealing with neurotic illness. His preference for a meaningful group therapy experience based on regressive reconstructive lines ignores the ample reasons for doubting that psychoanalytically oriented psychotherapy can be conducted with any sophistication in a group setting. The recent report of Malan and associates on the results of group psychotherapy conducted along psychoanalytic lines confirms the clinical impression that such treatment may not be beneficial to any great number of patients (1).

Although the tone of this book would indicate that a large number of Americans yearn for an intensive group experience, this is certainly not confirmed by the experience of many directors of psychiatric outpatient clinics, where it is difficult to establish and maintain group programs. The resistance to entering group therapy is routinely greater than the resistance to individual treatment. This may be related to a number of factors, including a basic positive transference, which can be easily mobilized only in an individual relationship. Many patients are simply unable to conceive of groups in a positive sense or to substitute them for the transference figure of the individual therapist. Furthermore, the feeling of many patients that they require individual attention is justified by the complexity of their internal and external difficulties. One of the early rationales for group treatments, namely, the shortage of trained psychotherapists, has become a less compelling factor and may, in many locations, be a rationalization for a form of treatment that has gained its adherents rather than a reality.

The remaining four chapters of the book are concerned with groups as social phenomena rather than treatment efforts. They include material about so-called sensitivity encounters, self-awareness groups, human relations training, and communes. Rather than summarizing the contents of each of these chapters I would like to emphasize the underlying common theme that they are overly general and vague as to what functions these groups are supposed to perform. The meaning of such groups to psychiatrists involved with treatment of neuroses, borderline conditions, and psychoses is minimal. They often represent an attempt to add something, perhaps something nonessential, to an individual's life. They stand in definite contrast to individual and group treatments that attempt to recognize illness, increase the patient's awareness of its causality when possible, and encourage working through and change based on insight or modification and, when necessary, with the help of medication.

The following statement appears without any critical comment but with a note of definite approval:

> Ida Rolf developed structural integration now commonly known as rolfing in order to free habitual patterns deeply engrained in physical structure. Rolfing is a system of deep muscle manipulation and realignment. The nature of the manipulation is based on the way the rolfer reads the structure of a client's body and its relationship to the environment.

That such a basic misunderstanding of the connection between psyche and soma could be propagated, with positive comment, is distressing indeed.

The basic problem with this book is that it is at war with itself. The first chapter is a basically sound survey of various group psychotherapy techniques. The second through fifth chapters are concerned with "movements" that have more in common with religious belief systems than psychotherapy. Unfortunately, because of the chapter distribution the forces of sound clinical judgment appear to be losing during most of it. As psychiatrists, we are frequently faced with patients who are seduced into questionable group movements that promise magical changes in the form of rapid growth and flowering of extraordinary personal capacities. It is time that we recognized the distinct possibility that experiential groups may be antitherapeutic at their very core. An individual patient who seeks treatment because of personal suffering may attain such in either a group or individual setting. However, the possibility exists that individuals may be seduced away from active professional treatment by the false belief that something can be added to their personality that will relieve suffering or that the suffering itself can be cured by growth.

For the psychiatrist this book has little to offer. As a guide for the general public it leaves much to be desired. Consumers are left on their own amid an alarming number of promises from various types of group proponents. Perhaps this book should be read by professionals in the mental health field to increase their caution in recommending a group experience for those who depend on them for insight and healing.

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HENRY J. FRIEDMAN, M.D.
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Le Projet Psychothérapique: De l'Activation à l'Auto-Activation, *by Mario Berta and Jean Claude Benoit. Paris, France, Denoël, 1976, 205 pp., no price listed (paper).*

The modern psychotherapist is confronted with various, sometimes mutually opposing, schools of psychotherapeutic thought. Mario Berta and Jean Claude Benoit attempt to present in this volume a unified psychotherapeutic approach that aims at integrating biological, psychological, and social approaches to the treatment of mental illness. This attempt at integration is concretized in their formulation of a new type of psychotherapy, developed by Benoit and called "psychotherapeutic activation." Berta and Benoit show in

particular in this volume the application of this approach in individuals and in the institutional setting.

The authors briefly present the personality theory underlying their approach in the introduction. This attempt at integration is certainly very commendable, but it appears to have led the authors to a rather superficial and little elaborated mix of various points of view. The basis of their theory rests on some aspects of existential psychology, with an emphasis on the style with which each individual perceives the world. The authors differentiate several dimensions of psychological perceptions that present themselves always in a bipolar way; e.g., the time dimension ranges from the past to the future. An individual becomes neurotic when he or she uses rigidly and persistently only one pole of these various bipolar dimensions. Other theoretical viewpoints included in the authors' formulation stem from systems theory and learning theory; unconscious dynamic factors and transference issues are given a minor, almost nonexistent place in their theoretical construct.

The cornerstone of the therapeutic approach derived from this theory is a continuous emphasis on the mutually stimulating activity of therapist and patient: "The therapist-patient relationship has to be an activating one, rather than like the one between an omnipotent master and his passive apprentice." The authors therefore suggest that the patient should be invited from the first session on to accept more responsibility for his or her behavior and that his or her active engagement in therapy should be requested. They advocate the further step of gradually increasing the patient's anxiety by confronting him or her in a measured way with the anxiety-creating stimulus. The ensuing reactions will break the patient's rigid, ineffective defensive systems, and alternative modes of behavior can be introduced.

There is a continuous emphasis in this book on teaching the patient more appropriate and more effective modes of behavior. Insight is deemphasized, and understanding of the patient's rigid and faulty "style" of perception is emphasized. The patient's potential to learn new behavior is to be actively stimulated to help him or her creatively find new ways of adaptation. The final goal of the authors' therapy is not a cure but the self-activation of the patient by which he or she can pursue autonomous growth with what he or she has learned during therapy.

The authors are very well aware that their psychotherapeutic approach is a departure from the traditional psychoanalytic one and that they are attempting to present an eclectic and flexible approach based on a pragmatic-pluralistic use of various active interventions. Their approach also appears to be a formulation of a brief psychotherapy and reflects the more eclectic approach of European schools of psychotherapeutic thought. In terms of relating their approach to other newer methods of psychotherapy, one could place it near Frankl's existential logotherapy or Ellis' rational emotive therapy.

In the second part of the volume Berta presents the application of the authors' approach in the individual private practice setting. He emphasizes early and complete history taking, particularly along the lines of the individual patient's psychological reactions to past traumatic events. Berta gives a central position in the diagnostic evaluation to a "test of wishes": patients are asked to imagine themselves as nonhuman objects—one they would like to be and one they would not. The symbols obtained in this fashion are used not only diagnostically but also therapeutically by confrontation with the patient. A pictorial activation is used to expose the patient in a gradual and progressive way to the aversive stimulus represented by the negative wish symbol. Diagrams, active planning of alternative modes of behavior, and various relaxation techniques are used. Another unorthodox and thought-provoking technique is what Berta calls "kinetic objectivation." This symbolic confrontation, based on karate, is a sort of shadow boxing with an imaginary enemy and usually embodies the undesirable personality trait the patient wants to get rid of. This is integrated with an appropriate psychological preparation and exploration of the patient.

In the third section of this book Benoit presents the application of the authors' approach in the institutional setting. This section is mainly an up-to-date description of modern integrated hospital and day-hospital treatment of severe mental illness, with emphasis on the multidisciplinary approach and the integration of individual psychotherapy with a therapeutic milieu. The reader will find nothing new here; one is not quite sure why this section has been included in the book.

In all, I think that this particular method of psychotherapy adds relatively little in terms of theoretical thinking to the presently used nonanalytic body of theories. Its attempt at theoretical integration is positive but not convincing. It also leaves aside important contributions of dynamic psychology. Its very positive aspects are the emphasis on the therapist's active involvement; the clarification of maladaptive, unidimensional styles of perception; and the contribution of a number of interesting specific techniques that may be used in particular situations with specific patients. These techniques could be valuable for the eclectic therapist who is interested in an active, short psychotherapy model.

JEAN-PIERRE LINDENMAYER, M.D.
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Modern Perspectives in the Psychiatric Aspects of Surgery, *edited by John G. Howells, M.D., D.P.M. New York, N.Y., Brunner/Mazel, 1976, 729 pp., $25.00.*

This book contains 36 chapters by 48 authors from 10 different countries. As one might expect, it is sporadic in quality and continuity. The purposes of the book, as stated in the editor's preface, are to provide "a contribution towards the birth of surgeon psychiatrist" and "a unique international encyclopedia in psychiatry." The first concept, the surgeon psychiatrist, is further defined as "encompassing psychic as well as surgical diagnoses, perceptive of the psychic significance of operative surgery and with an holistic approach to management procedures." The volume also seeks a greater interaction between surgical and psychiatric departments in hospitals. However, this objective is hardly likely to be accomplished by this book.

The first chapter, "The Ethical Objectives of Surgery," is openly critical of surgery and surgeons. To ask whether surgery is really necessary, to suggest that "with any luck surgery would be obsolete in 50 or 100 years," and to admonish that surgeons "must guard against using patients to prove a theory or to advance their careers" in the opening chapter can hardly be expected to whet the surgeon's appetite for what follows. It is also regrettable that of the 48 authors only 6 are surgeons. It would appear that in a book whose authors span the globe it would have been possible to find more psychologically minded surgeons.

The book's second purpose, to provide an encyclopedia of psychological problems related to surgery, is somewhat more adequately, if sporadically, fulfilled. It should be noted

at this point that this is not a book to be read from cover to cover. The repetitive exhortations to consideration and understanding in approaching the patient are well enough said, but they become tedious when encountered in chapter after chapter. On specific topics some chapters are adequately referenced reviews while others are primarily the author's experience with a particular problem.

Among those topics which I found most useful were the following: head injury (chapter 8), accident proneness (chapter 4) and accident neurosis (chapter 35), hypnosis (chapter 28), renal transplantation (chapter 18), and surgical delirium (chapter 30). Other chapters may, of course, appeal more to other readers with particular interests. By and large the depth and comprehensiveness of individual chapters are more likely to appeal to the student than to the expert in the field. A search for particular information is aided by a good index. In such a comprehensive book one shirks from suggesting additions, but chapters on the adult burned patient and on bypass surgery for obesity would have been timely.

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Physiology of Aggression and Implications for Control: An Anthology of Readings, *edited by Kenneth Evan Moyer. New York, N.Y., Raven Press, 1976, 320 pp., $15.00 (paper).*

This book is a compilation of 26 articles from the periodical literature. They have been grouped in six sections: Neurosubstrates of Aggression: Some Evidence from Animals; Endocrine Substrates of Aggression: Some Evidence from Animals; Miscellaneous Studies on Animals; Neurosubstrates of Aggression: Some Evidence from Humans; Endocrine Substrates of Aggression: Some Evidence from Humans; and Physiological Control of Aggression. Each section is preceded by a brief introduction by the editor. Approximately half of the articles deal with the physiological correlates of aggression in animals and half with studies of humans.

Most of these articles were originally published after 1965; the earliest was first published in 1961. The editor notes in the general introduction that only 4 of more than 90 books published on aggression have focused primarily on physiological variables and that all 4 of these have been published since 1970. These dates reflect the relatively recent surge of social concern and scientific interest in aggression.

Of the 6 experimental studies in section 1, 4 deal with aggression in cats and 2 with aggression in rhesus monkeys. Of the 5 articles in section 2, 4 deal with aggression in rats and mice and 1 with aggression in rhesus monkeys. Of the 3 articles in section 3, 1 deals with mice, 1 with squirrel monkeys, and the third is a review article focused largely on aggression in rats and mice. The editor notes that one cannot generalize the findings on animals to man but then asserts that "although there is a wide gap between even the highest animals and man, similar biological mechanisms influence tendencies to aggression in both" (p. xiv). The key word in this quotation is "similar." Although the neural and hormonal substrates of aggression in humans may be similar to those in lower species in a very general sense, the psychiatrist reader is not likely to find much information in the first half of this book that is germane to the diagnosis and treatment of aggressive disorders of behavior in humans.

Section 4 contains 2 articles dealing with the relationship of CNS activity to aggression. The first of these, "Behavioral and Affective Responses to Brain Stimulation in Man," reports the subjective and behavioral results of the electrical stimulation of discrete regions of the subcortex, including the thalamus and limbic system. These studies were conducted with patients undergoing neurosurgery for intractable pain. The results are reported in a manner the authors themselves characterize as "anecdotal." The most striking fact about the inclusion of this article in this volume is that aggressive behavior was reported in only one of the many patients described, a patient who was stimulated in the amygdala. Later stimulation of the amygdala in the same patient did not evoke aggressive behavior. Moreover, none of the other patients stimulated in the amygdala or any other subcortical areas exhibited aggressive behavior. It is therefore difficult to understand why this article was included in this volume.

The second article in section 4 reports the results obtained when the clinical EEGs of habitually aggressive prisoners were compared with those of prisoners who had committed a single violent crime. The author concludes that 65% of the EEGs in the first group were abnormal, compared with 24% in the second group, a significant difference. He argues that this difference supports the interpretation that single acts of aggression are provoked primarily by "life situations," while repeated aggression is "endogenous" and a consequence of CNS pathology. Psychiatrists familiar with EEG interpretation may question the author's criteria for abnormality. Most important, so many types of abnormality in so many cortical leads are included in the statistical comparison that no conclusion can be drawn regarding the specific loci for the mediation of aggression in the CNS.

Section 5 reports that positive correlations exist between male sex hormones and the degree of aggressive behavior, that "a dysfunction of either the adrenal or thyroid gland results in increased irritability," that hypoglycemia is associated with a state of hyperirritability, and that stilbestrol has provided dramatic control of both "hypersexuality and irritable aggression." Although most physicians, including psychiatrists, would agree that aggressive behavior is sometimes associated with such hormonal changes, they also agree that the presence of such changes is not a sufficient condition to induce aggression. In fact, this is essentially the conclusion reached by Kreuz and Rose in an article in this volume.

Section 6 deals with efforts to treat aggressive disorders, including neurosurgical, psychotropic, and hormonal approaches. Psychiatrists with even minimal familiarity with the literature in this area will find little new in this section.

There has been a considerable amount of psychophysiological research on psychopathic and other impulsive people. This area is not represented in this volume; this is regrettable because the research of such investigators as Hare and Lykken is more rigorous methodologically than are most of the articles that have been included.

In summary, this volume contains a number of articles authored by investigators from diverse disciplines. It is difficult to identify a coherent theme even within disciplines and impossible to do so across disciplines. Psychiatrists who are entirely unfamiliar with the physiology of aggression may learn something from this heterogeneous group of articles, but if they are seriously interested in gaining information relevant to their clinical concerns they would do well to conduct their own literature search.

ROBERT ROESSLER, M.D.
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Treatment of Schizophrenia: Progress and Prospects, *edited by Louis Jolyon West, M.D., and Don E. Flinn, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1976, 284 pp., $22.50.*

This volume originates from a 1972 conference on the treatment of schizophrenia at the University of California, Los Angeles. Many of the contributors are internationally recognized authorities in the field. Although most of the papers are new or updated, the literature from 1974 to the present tends to be underrepresented in the references.

There are six sections. In part 1, Models of Schizophrenia, Zubin explores the concept of vulnerability to schizophrenia in an overlong, extremely complex chapter. Kety gives a brief overview of psychobiological factors, and Mandell describes "Adaptive Macromolecular Regulation of Neurotransmission in Brain." Part 2, Psychopharmacological Approaches, fails to live up to the editors' claim that it "constitute[s] a current guide to psychopharmacological treatment of schizophrenia." May's chapter is characteristically pithy and clearly written but uncharacteristically choppy and fragmented in its approach. The chapter by Jarvik and associates provides a perfunctory view of psychopharmacological treatment, including a table that provides extremely low daily dose ranges for antipsychotic drugs for psychotic inpatients (e.g., 4–15 mg a day for haloperidol [Haldol]) (p. 87). Lipton effectively debunks megavitamin therapy in his chapter, which emphasizes the therapy's total lack of scientific validation.

In part 3, Individual Psychotherapy, Arieti and Grotstein explore psychoanalytical approaches to schizophrenia but provide no scientific or statistical data to balance those found in other sections. In part 4, Small Group Approaches, Ludwig describes his fascinating work with the "buddy treatment model" and other group treatment modalities. Mosher discusses family therapy, but his most recent reference other than his own work is from 1972.

In part 5, Milieu and Social Approaches, Liberman gives an excellent overview of behavior therapy, especially token economy systems. Lofgren describes success with "small, leaderless, process-oriented" therapy for schizophrenia. Van Putten and May discuss milieu therapy, emphasizing that it is "not an adequate substitute for treatment with antipsychotic drugs, although it may add to their effects." They also discuss the toxic overstimulation of some schizophrenic patients in some lively treatment milieus with "role diffusion, parties, loud music, inability to distinguish staff from patients." They conclude that current methods of hospital milieu therapy "add little to the treatment of the ordinary schizophrenic patient once gross neglect is corrected and adequate chemotherapy is used." Bellak convincingly presents his view that community psychiatric treatment has not been a panacea for chronic schizophrenic patients.

In part 6, titled Overview, Flynn, May, and West review the literature, especially May's brilliant 1968 book (1). However, little is done to integrate the previous parts of this volume into a coherent and consistent approach. In conclusion, this book has several excellent chapters but does not provide a comprehensive overview of the treatment of schizophrenia. It may be useful as a backup and reference work, but certainly not as a substitute for such basic textbooks as the second edition of the *Comprehensive Textbook of Psychiatry* (2).

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Origins of Concepts in Human Behavior: Social and Cultural Factors, *by Mark D. Altschule. Washington, D.C., Hemisphere Publishing Corp. (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1977, 200 pp., no price listed.*

Consent Handbook, *by the American Association on Mental Deficiency Legislative and Social Issues Committee Task Force. Washington, D.C., AAMD, 1977, 80 pp., no price listed (paper).*

Medical Socioeconomic Research Sources, Vol. 6, Annual Cumulation 1976, *by the American Medical Association. Germantown, Md., Aspen Systems Corp., 1976, 369 pp., no price listed.*

Raising Kids O.K.: Transactional Analysis in Human Growth and Development, *by Dorothy E. Babcock and Terry D. Keepers. New York, N.Y., Avon Books, 1977, 295 pp., $1.95 (paper).*

Human Sexuality in Four Perspectives, *edited by Frank A. Beach. Baltimore, Md., Johns Hopkins University Press, 1977, 317 pp., $15.00.*

Dictionary of Psychology. English-German, *by Alfred H. Berger. New York, N.Y., Frederick Ungar Publishing Co., 1977, 199 pp., $12.50.*

Working with People Called Patients, *by Milton M. Berger, M.D. New York, N.Y., Brunner/Mazel, 1977, 150 pp., $7.95 (paper).*

Therapeutic Psychology: Fundamentals of Counseling and Psychotherapy, 3rd ed., *by Lawrence M. Brammer and Everett L. Shostrom. Englewood Cliffs, N.J., Prentice-Hall, 1977, 466 pp., $13.95.*

The Social Worker as Sex Educator, *by Diane B. Brashear, Ph.D., ACSW. New York, N.Y., Sex Information and Education Council of the U.S. (SIECUS) (Human Sciences Press, distributor), 1976, 27 pp., $2.25 (paper).*

Current Issues and Strategies in Organization Development, *edited by W. Warner Burke, Ph.D. New York, N.Y., Human Sciences Press, 1977, 444 pp., $24.95.*

SIECUS Film Resources for Sex Education, 1976 ed., *compiled and edited by Derek L. Burleson, Ed.D., and Gary Barbash. New York, N.Y., Sex Information and Education Council of the U.S. (SIECUS) (Human Sciences Press, distributor), 1976, 52 pp., $4.95 (paper).*

Behavior and Adaptation in Late Life, 2nd ed., *edited by Ewald W. Busse, M.D., D.Sc., and Eric Pfeiffer, M.D. Boston, Mass., Little, Brown and Co., 1977, 366 pp., no price listed (paper).*

Sex Therapy: A Behavioral Approach, *by William Caird, Ph.D., and John P. Wincze, Ph.D. Hagerstown, Md., Harper & Row Medical Department, 1977, 197 pp., $12.95.*

Long-Term Treatments of Psychotic States, *by Colette Chiland, M.D., Ph.D., with editorial assistance from Paul Bequart. New York, N.Y., Human Sciences Press, 1977, 688 pp., $37.95.*

To Be an Invalid: The Illness of Charles Darwin, *by Ralph Colp, Jr., M.D. Chicago, Ill., University of Chicago Press, 1977, 276 pp., $17.50.*

Behavioral Neurochemistry, *edited by J.M.R. Delgado, M.D., and F.V. DeFeudis, Ph.D. New York, N.Y., Spectrum Publications (Halsted Press, John Wiley & Sons, distributor), 1977, 249 pp., $20.00.*

Crisis Intervention and How It Works, *by Romaine V. Edwards, M.S.W. Springfield, Ill., Charles C Thomas, 1977, 73 pp., $8.00.*

Clinical Interviewing Skills: A Programmed Manual for Data Gathering, Evaluation, and Patient Management, 3rd ed., *by Robert E. Froelich, M.D., and F. Marian Bishop, Ph.D., M.S.P.H. St. Louis, Mo., C.V. Mosby Co., 1977, 201 pp., $7.50 (paper).*

Assert Yourself! How To Be Your Own Person, *by Merna Dee Galassi and John P. Galassi. New York, N.Y., Human Sciences Press, 1977, 237 pp., $7.95 (paper).*

Coping and Defending: Processes of Self-Environment Organization, *by Norma Haan. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1977, 342 pp., $17.50.*

Psychology: The Basic Principles, *by John F. Hahn and Sanford Lopater. Totawa, N.J., Littlefield, Adams & Co., 1977, 208 pp., $3.50 (paper).*

Communication in Medicine: A Challenge to the Profession, *by Ole K. Harlem. New York, N.Y., S. Karger, 1977, 98 pp., $15.25 (paper).*

Going Sane: An Introduction to Feeling Therapy, *by Joseph Hart, Ph.D., Richard Corriere, Ph.D., and Jerry Binder, M.A. New York, N.Y., Jason Aronson, 1975, 464 pp., $9.95.*

Progress in Behavior Modification, Vol. 4, *edited by Michel Hersen, Richard M. Eisler, and Peter M. Miller. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1977, 376 pp., $19.50.*

Child Sense: A Guide to Loving, Level-Headed Parenthood, new and expanded ed., *by William E. Homan, M.D. New York, N.Y., Basic Books, 1977, 295 pp., $10.95.*

The Hutt Adaptation of the Bender-Gestalt Test, 3rd ed., *by Max L. Hutt. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1977, 268 pp., $14.50.*

Social Interaction, *by Thomas M. Kando, Ph.D. St. Louis, Mo., C.V. Mosby Co., 1977, 331 pp., $9.25 (paper).*

The Guidance Counselor as Sex Educator, *by Gary F. Kelly. New York, N.Y., Sex Information and Education Council of the U.S. (SIECUS) (Human Sciences Press, distributor), 1976, 26 pp., $2.25 (paper).*

Schizophrenie und Gesellschaft: Zum Menschenbild in Psychiatrie und Soziologie, *by Marianne Krüll. Munich, Germany, Verlag C.H. Beck, 1977, 211 pp., 14,80 Deutsche marks (paper).*

Is There Life After Group? *by Lester Libo. Garden City, N.Y., Anchor Books (Anchor Press/Doubleday), 1977, 144 pp., $1.95 (paper).*

Piagetian Theory and Its Implications for the Helping Professions: The Handicapped Child, *edited by James F. Magary, Marie K. Poulsen, Philip J. Levinson, and Priscilla A. Taylor. Los Angeles, Calif., University of Southern California Special Order Department and Bookstore, 1977, 461 pp., $10.00 (paper).*

Psychiatric Treatment of the Child, *edited by John F. McDermott, Jr., M.D., and Saul I. Harrison, M.D. New York, N.Y., Jason Aronson, 1977, 826 pp., $25.00.*

Challenges in Mental Retardation: Progressive Ideology and Services, *by Frank J. Menolascino, M.D. New York, N.Y., Human Sciences Press, 1977, 350 pp., $16.95.*

Coping with Physical Illness, *edited by Rudolf H. Moos in collaboration with Vivien Davis Tsu. New York, N.Y., Plenum Medical Book Co., 1977, 433 pp., no price listed.*

The Sexual Self, *by Avodah K. Offit, M.D. Philadelphia, Pa., J.B. Lippincott Co., 1977, 292 pp., $10.00.*

Cultures in Collision, *edited by Issy Pilowsky. Adelaide, Australia, Australian National Association for Mental Health, 1975, 427 pp., $17.80 (Australian dollars; includes postage) (paper).*

Not Dying, *by F. Robert Rodman. New York, N.Y., Random House, 1977, 216 pp., $7.95.*

Work, Aging, and Social Change: Professionals and the One Life-One Career Imperative, *by Seymour B. Sarason. New York, N.Y., Free Press (Macmillan Publishing Co.), 1977, 293 pp., $14.95.*

Currents in Alcoholism, Vol. I, *edited by Frank A. Seixas, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1977, 480 pp., $19.50.*

Currents in Alcoholism, Vol. II, *edited by Frank A. Seixas, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1977, 534 pp., $19.50 ($35.00 for volumes I and II).*

Child and Adolescent Psychiatry: Continuing Education Review, *edited by G. Pirooz Sholevar, M.D. Flushing, N.Y., Medical Examination Publishing Co., 1977, 230 pp., $10.00 (spiral-bound).*

A School for Fools, *by Sasha Sokolov; translated by Carl R. Proffer. Ann Arbor, Mich., Ardis, 1977, 228 pp., $10.00; $3.00 (paper).*

Psychodrama: Rehearsal for Living. Illustrated Therapeutic Techniques, *by Adaline Starr. Chicago, Ill., Nelson-Hall, 1977, 363 pp., $18.00; $9.95 (paper).*

Assessment of Persons, *by Norman D. Sundberg. Englewood Cliffs, N.J., Prentice-Hall, 1977, 344 pp., no price listed.*

The Rights of Young People: The Basic ACLU Guide to a Young Person's Rights, *by Alan N. Sussman. New York, N.Y., Avon Books, 1977, 251 pp., $1.50 (paper).*

Temperament and Development, *by Alexander Thomas, M.D., and Stella Chess, M.D. New York, N.Y., Brunner/Mazel, 1977, 265 pp., $13.50.*

Depression: Clinical, Biological and Psychological Perspectives, *edited by Gene Usdin, M.D. New York, N.Y., Brunner/Mazel, 1977, 338 pp., $15.00.*

Barbiturates: Their Use, Misuse, and Abuse, *by Donald R. Wesson, M.D., and David E. Smith, M.D. New York, N.Y., Human Sciences Press, 1977, 138 pp., $4.95 (paper).*

Highlights of the 130th Annual Meeting

The 130th annual meeting of the American Psychiatric Association, which focused on the theme "Professional Responsibility and Public Trust," was held in Toronto, Ont., Canada, May 2–6, 1977. The total registration was 10,612, including 4,940 members, 3,380 nonmembers (including program participants), 668 scientific exhibitors, 132 members of the press, and 1,492 spouses and families of members.

The opening session, on Monday morning, May 2, was called to order by Robert W. Gibson, M.D., 105th President of the Association. The invocation was given by Rabbi Gunther Plaut of the Holy Blossom Temple, Toronto. Official greetings were extended by John Evans, M.D., President of the University of Toronto. Dr. Gibson read special greetings from Paul V. Godfrey, Chairman of the Metropolitan Toronto Council, and from David Crombie, Mayor of the City of Toronto. Dr. Gibson then introduced Dr. S.E. Greben, Chairman of the Board of Directors, Dr. André Coté, Secretary, and Dr. John S. Pratten, President, Canadian Psychiatric Association. Dr. Pratten read a message from Prime Minister Pierre Trudeau and extended greetings on behalf of the Canadian Psychiatric Association.

Dr. Gibson introduced the distinguished guests, who included the following: Dr. Tsung-yi Lin, President of the World Federation for Mental Health; Dr. Gerald J. Sarwer-Foner, President of the American Academy of Psychiatry and the Law; Dr. John Wolfe, President of the National Council of Community Mental Health Centers; Dr. Irving N. Berlin, President of the American Academy of Child Psychiatry; Dr. Paul Chodoff, President of the American Academy of Psychoanalysis; Dr. John L. Carleton, President of the American Association for Social Psychiatry; Dr. Marc Hollender, Vice-President and representative of the American Board of Psychiatry and Neurology; Dr. George L. Lipton, the Honorable Federal Secretary and National Representative of the Australian and New Zealand College of Psychiatrists; Dr. John H. Budd, President-Elect and representative of the American Medical Association; Dr. Edward O. Harper, President of the American College of Psychiatrists; Dr. Léon Chertok, Secretary of the Société de Médecine Psychosomatique, Paris; Dr. John Donnelly, President of the National Association of Private Psychiatric Hospitals; Dr. Henry Laughlin, President of the American College of Psychoanalysts; Dr. Howard P. Rome, President of the World Psychiatric Association; Dr. Wilbert J. McKeachie, Past-President and representative of the American Psychological Association; Ms. Martha L. Mitchell, Chairperson of the American Nurses' Association's Division on Psychiatric and Mental Health Nursing Practice, representing the American Nurses' Association; Dr. Jane W. Kessler, President of the American Orthopsychiatric Association; Dr. Kenneth T. Calder, President of the American Psychoanalytic Association; Dr. Joe W. King, President-Elect and representative of the American Society for Adolescent Psychiatry; Chaplain Emanuel L. Lifschutz, President of the Association of Mental Health Clergy; Dr. Rubin L. Siegel, President of the American Society of Physician Analysts; and Dr. Walter Shervington, Chairman, Section on Neurology and Psychiatry and representative of the National Medical Association.

Dr. Gibson then called on Dr. Alfred M. Freedman, Chairperson of the Task Force To Plan a Joint Meeting with the French, who reported on plans for the meeting, to be held in Paris following the 1978 annual meeting in Atlanta. Dr. Shervert W. Frazier, Chairperson of the VI World Congress of Psychiatry Organizing Committee, commented on plans for the Congress, which will be held in Honolulu, Hawaii, August 28–Sept. 3, 1977. Dr. Tsung-yi Lin, President of the World Federation of Mental Health, extended an invitation to attend the Federation's World Congress, which will be held in Vancouver, B.C., Canada, August 21–26, 1977. The theme will be "Today's Priorities in Mental Health."

Dr. Gibson thanked Dr. Peter Martin and the Program Committee for their excellent work. Dr. Martin commented on changes in the program and noted some of its highlights. Dr. Gibson then introduced and thanked Dr. Quentin A.F. Rae-Grant, Chairperson of the Task Force on Arrangements. Following this, Dr. Frederick H. Lowy introduced a slide show prepared by the National Film Board of Canada titled "Between Friends—Entre Amis."

Vice-President Daniel X. Freedman, M.D., presented a proper accolade of Dr. Gibson, who gave an eloquent Presidential Address on "A Profession Worthy of the Public Trust." Vice-President Alan A. Stone, M.D., similarly introduced Jack Weinberg, M.D., who gave the response of the President-Elect. (The texts of these addresses are published elsewhere in this issue of the *Journal*.)

The Reverend G. Freemesser, M.D., APA Fellow and member of the Order of Basilian Fathers, St. Basil's College, Toronto, delivered the benediction. The opening session closed with organ music by John E. Fryer, M.D.

SCIENTIFIC SESSIONS

The scientific sessions began Monday afternoon, May 2. There were 429 papers presented in regular sessions, as well as 27 papers on new research presented in several sessions throughout the week. There were 150 morning and evening panels, 7 videotaped sessions, 62 courses, and 7 film sessions.

The Solomon Carter Fuller Lecture, "Black Leaders'

Handling of Their Own Anger," was given by Allison Davis, Ph.D., John Dewey Distinguished Service Professor, Department of Education, University of Chicago. David Hamburg, M.D., President of the Institute of Medicine, National Academy of Sciences, Washington, D.C., presented the Seymour Vestermark Memorial Lecture, "Reflections on Psychiatry and Medical Education in the Remainder of this Century." Professor Doctor Thure von Uexküll, Emeritus, University of Ulm, West Germany, gave the Adolph Meyer Lecture, "The Psychosomatic Approach: The Problem of a Theory of General Medicine." The Simon Bolivar Lecture, "Science and the Americas," was given by Humberto Fernandez-Moran, M.D., Professor of Biophysics, Research Institute of the University of Chicago. Daniel Aaron, Professor of American Civilization, Harvard University, Boston, Mass., was this year's Benjamin Rush Lecturer, on the subject of "The Etiquette of Grief: A Literary Generation's Response to Death."

There were 3 special lectures: "The Psychiatrist and Public Policy," by Peter G. Bourne, M.D., Special Assistant to the President for Mental Health and Drug Abuse, Washington, D.C.; "Long-Range Trends in Urban Mental Health: Old Theories and New Evidence from the Midtown Manhattan Restudy," by Leo Srole, Ph.D., Professor of Social Sciences, College of Physicians and Surgeons, Columbia University, New York, N.Y.; and "Group Cohesion and Dissidence," by J.O. Wisdom, Ph.D., University Professor of Philosophy and Social Sciences, York University, Toronto, Ont., Canada.

CONVOCATION

Dr. Gibson presided at the 21st Convocation of Fellows, held in the Grand Ballroom of the Sheraton Centre on Monday, May 2. An invocation was given by the Right Reverend H. Stiff, Dean of St. James Cathedral, Toronto. Jack Weinberg, M.D., President-Elect, then inducted Life Fellows and Fellows.

Dr. Gibson introduced Manfred Bleuler, M.D., Zurich, Switzerland, Jean Paul Delay, M.D., Paris, France, and Pierre Georges Deniker, M.D., Paris, France, who were designated Distinguished Fellows of the Association. The Reverend George C. Anderson, D.D.†, Swarthmore, Pa., and Mr. Edward F.X. Lawlor, Jr., Ambler, Pa., were designated Honorary Fellows.

Dr. Gibson introduced Life Fellows and life members who have been in the Association for 50 years (1927–1977). They are: Katherine Grace Brockman, M.D., Lacey, Wash.; Walter L. Bruetsch, M.D.†, Santa Barbara, Calif.; George Eaton Daniels, M.D., New York, N.Y.; Thomas Morton French, M.D.†, Chicago, Ill.; Daniel Patrick Griffin, M.D., Bridgeport, Conn.; Henry Harper Hart, M.D., Southbury, Conn.; Livingston Spraker Hinckley, M.D., Miami, Fla.; George Stephen Johnson, M.D., San Francisco, Calif.; Leo Kanner, M.D., Baltimore, Md.; J.H. Fernand Longpre, M.D., Florence, Mass.; William Malamud, M.D., Bloomfield, N.J.; Clyde Slocomb Marshall, M.D., Halifax, Nova Scotia, Canada; Hosea Webster McAdoo, M.D., North Little Rock, Ark.; Veronica Murphy Pennington, M.D.†, Jackson, Miss.; George Salvadore Stevenson, M.D., Red Bank, N.J.; Kenneth James Tillotson, M.D., Belmont, Mass.; and Alphonse Ralph Vonderahe, M.D., Cincinnati, Ohio.

†Deceased.

The following were designated Corresponding Fellows: Dr. Romano Antonelli, Rome, Italy; Dr. Rose Chacko, Vellore, South India; Dr. Hari Dass Chopra, Parkville, Victoria, Australia; Dr. Hanfried Helmchen, Berlin, Germany; Dr. Jean Jacques Meisch, Ettelbruck, Grand Duchy of Luxembourg; Dr. M. Sarada Menon, Madras, India; Dr. Esra Seraja Petursson, Reykjavik, Iceland.

The presentation of awards followed. Dr. Gibson presented the Distinguished Service Award—given to APA members "whose distinguished careers have ennobled the profession of psychiatry"–to Jack R. Ewalt, M.D., Director, Mental Health and Behavioral Sciences Service, Veterans Administration, Washington, D.C., and Past-President of the Association (1963–1964).

The Lester N. Hofheimer Prize for Research was presented by Joel J. Elkes, M.D., to Eric R. Kandel, M.D., Division of Neurobiology and Behavior, Department of Physiology, College of Physicians and Surgeons, Columbia University, New York, N.Y., for his distinguished contribution "to an understanding of the biological basis of behavior, with special reference to problems of neuronal plasticity and the biological correlates of learning." The Hofheimer Prize Board also extended special recognition to Donald Cohen, M.D., for his "outstanding contribution to the study of biological and psychosocial aspects of human development, with special reference to the genesis of competence, and the antecedents of psychosomatic illness," and to Robert N. Emde, M.D., Theodore J. Gaensbauer, M.D., and Robert J. Harmon, M.D., for their "distinguished contribution to the study of emotional expression in infancy, with special reference to the timing and organization of behavioral shifts."

Joseph Noshpitz, M.D., presented the Agnes Purcell McGavin Award to Viola Bernard, M.D., Chairperson, APA Council on Children, Adolescents, and Their Families, and Clinical Professor Emeritus, College of Physicians and Surgeons, Columbia University, New York, N.Y., for "outstanding contributions to the prevention of mental disorders in children."

The APA Founders Award, established in 1976 to honor members "who have made outstanding contributions as author, spokesperson and advocate in the service of the mentally ill and disabled and to the art and science of helping them," was presented by Shervert H. Frazier, M.D., to Karl A. Menninger, M.D., who was characterized as an "elder statesman of our field, foremost humanist and champion of the mentally ill and troubled, striking communicator to people in all walks of life, compassionate physician and social reformer *par excellence*."

The Robert T. Morse Writers Award, which honors "popular writers who have made major contributions to the public understanding of psychiatry over an extended period of time," was then accorded to Abigail Van Buren, "columnist, writer, and lecturer of national and international impact."

The Manfred S. Guttmacher Award was presented by Carl P. Malmquist, M.D., to: Frank W. Miller, James Carr Professor of Criminal Jurisprudence, Washington University, St. Louis, Mo.; George E. Dix, Professor of Law, University of Texas at Austin; Robert O. Dawson, Wright C. Morrow Professor of Criminal Law, University of Texas at Austin; and Raymond I. Parnas, Professor of Law, University of California at Davis, for their book, *The Mental Health Process*, published by the Foundation Press, Mineola, N.Y.

The Seymour C. Vestermark Prize Award for "outstanding contributions to undergraduate and postgraduate medical education, to continuing education, and to the edu-

cation of behavioral scientists for research" was presented by L. Douglas Lenkoski, M.D., to David A. Hamburg, M.D., President, the Institute of Medicine, National Academy of Sciences, Washington, D.C.

The District Branch Newsletter of the Year Award was presented by Robert J. Campbell III, M.D., to *The Maryland Psychiatrist*, Lino Covi, M.D., Editor. This award recognizes "the overall excellence of outstanding newsletters published by the District Branches of the Association from the standpoint of editorial content, attractiveness of format, and usefulness in furthering communication within the profession."

The Isaac Ray Award for "psychiatrists, attorneys, and judges who have made outstanding contributions to legal psychiatry" was given to Bruno Comier, M.D., Professor, Department of Psychiatry, and Director, Clinic in Forensic Psychiatry, McGill University, Montreal, Que., Canada, by Naomi Goldstein, M.D.

Dr. Gibson presented a special plaque to Henry S. Sager, M.D., for his "long-term commitment to improving relations between psychiatrists in the United States and Canada."

Following the ceremonies, Dr. Gibson introduced Dr. Anne R. Somers, Professor, Departments of Community Medicine and Family Medicine, College of Medicine and Dentistry of New Jersey-Rutgers Medical School, and Research Associate, Industrial Relations Section, Princeton University. Dr. Somers gave the William C. Menninger Memorial Convocation Lecture, "Accountability, Public Policy, and Psychiatry."

The Convocation then recessed.

BUSINESS SESSION

The business session was convened as a meeting of the Assembly of District Branches acting for the membership in receiving reports of the officers and actions of the Board of Trustees. The meeting was held Tuesday afternoon, May 3, in the Grand Ballroom of the Sheraton Centre.

Dr. Gibson asked for a moment of silence in memory of deceased Fellows and members. John D. Griffin, M.D., then read a memorial to George Herbert Stevenson, M.D., Past President of the Association (1940–1941).

Robert J. Campbell, M.D., Recorder of the Assembly, called the roll of Assembly delegates and reported that a quorum was present.

Louis F. Rittelmeyer, M.D., Chairperson, Committee of Tellers, reported the results of the election of officers and trustees and of the vote on the proposed amendments to the Constitution and By-Laws. Jules H. Masserman, M.D., was certified as President-Elect; Donald G. Langsley, M.D., as Vice-President; H. Keith H. Brodie, M.D., as Secretary; Mary Ann Bartusis, M.D., as Trustee-at-Large; Abram M. Hostetter, M.D., as Area III Trustee; and Robert A. Moore, M.D., as Area VI Trustee. The Committee of Tellers also certified the approval of amendments to the following sections of the Constitution and By-Laws: Article II; Article III; Chapter One, Section 4; Chapter 2, Section 1; Chapter 2, Section 2; Chapter 3, Section 3; Chapter 7, Section 6; and Chapter 8, Section 6. The report of the Committee was accepted.

The reports of the officers to the membership followed. Jules H. Masserman, M.D., Secretary, summarized his 33-page report, which covered principal actions of the Board of Trustees and Executive Committee from April 1976 to April 1977. Charles B. Wilkinson, M.D., followed with the Treasurer's report, Irwin N. Perr, M.D., J.D., with the report of the Speaker of the Assembly of District Branches, and Daniel A. Grabski, M.D., with the report of the Speaker-Elect. Melvin Sabshin, M.D., presented the report of the Medical Director; Dr. Gibson thanked Dr. Sabshin and the Central Office staff for their work during the year. Walter H. Wellborn, M.D., Chairperson of the Committee on Constitution and By-Laws, commented on the committee's work and reviewed the procedure for submission of petitions to amend the Constitution and By-Laws, and John R. Elpers, M.D., reported for the Membership Committee. The Assembly accepted all reports as read.

Dr. Louis Jolyon West, Chairperson of the Council on Research and Development, sensed that the Council felt that its role was changing and urged that APA undertake more research of its own along the lines of the work performed by the Task Force on ECT.

Dr. Gibson then adjourned the business session and called the annual forum to order. Topics discussed included the efforts of APA to improve the image of psychiatry with other mental health disciplines, the increase in the number of members-in-training, and problems in acquiring and transferring District Branch membership. Dr. Gibson then adjourned the annual forum.

MEETINGS OF THE BOARD OF TRUSTEES

The Board of Trustees met in regular sessions on Sunday, May 1, and Thursday, May 5. At the Thursday meeting Dr. Gibson noted that the new President, Dr. Jack Weinberg, had had to return to Chicago for health reasons. He therefore was unable to preside at the session.

ALLIED MEETINGS

The following professional associations also held meetings in Toronto during the week of May 2–6: The American Association for Social Psychiatry, the American Academy of Child Psychiatry, the American Academy of Psychiatry and the Law, the American Association of Chairmen of Departments of Psychiatry, the American College of Psychiatrists, the American College of Psychoanalysts, the American Society for Adolescent Psychiatry, the American Society for Phenomenological Psychotherapy, the Association for the Advancement of Psychotherapy, the Association of Mental Health Clergy, the Behavior Therapy and Research Society, the Eastern Psychoanalytic Association, the Navy Psychiatric Seminar, and the Society of Biological Psychiatry.

LEISURE-TIME ACTIVITIES

The Task Force on Arrangements, Co-Chaired by Quentin Rae-Grant, M.D., and May Kay Lowy, arranged interesting activities in and around Toronto, including excursions to Niagara Falls, walking tours of shopping areas and Chinatown, and visits to Toronto art galleries. Other events were a lunch in honor of the families of APA officers and tours of the Ontario Science Center. Opportunities for outdoor activities comprised tennis, fishing trips, and a golf tournament.

JULES H. MASSERMAN, M.D.
Secretary, American Psychiatric Association

The American Board of Psychiatry and Neurology

The following successfully completed the Board examination given in Seattle, Wash., Feb. 7–8, 1977.

PSYCHIATRY

Addario, Dominick, M.D., San Diego, Calif.
Ali, Shaffideen Z., M.D., Hopkinsville, Ky.
Andres, Valentino W. Jr., M.D., Yuba City, Calif.
Angell, Richard H., M.D., Aurora, Ore.
Anoshiravani, A., M.D., Philadelphia, Pa.
Arevian, Sarkis T., M.D., Redondo Beach, Calif.
Armstrong, Ralph H., M.D., Burbank, Calif.
Arnold, Alan J., M.D., Las Vegas, Nev.
Asnis, Martin, M.D., New York, N.Y.

Baker, John H. Jr., M.D., Orinda, Calif.
Barthel, Richard P., M.D., San Francisco, Calif.
Baumbacher, Gordon, M.D., San Francisco, Calif.
Blase, Marvin, M.D., Atlanta, Ga.
Blaustein, Melvin, M.D., San Francisco, Calif.
Blum, Alexander, M.D., Palo Alto, Calif.
Brandes, Peter Jr., M.D., Berkeley, Calif.
Brenner, Martin, M.D., Balboa Island, Calif.
Bridge, Thomas P., M.D., Washington, D.C.
Brown, Craig A., M.D., Del Mar, Calif.
Brust, James S., M.D., Rancho Palos Verdes, Calif.
Brzozowski, Thomas H., M.D., Santa Rosa, Calif.
Buccigross, Richard L., M.D., La Jolla, Calif.
Burnap, Donald W., M.D., Rapid City, S.D.
Burnham, John R., M.D., S. Laguna, Calif.

Canada, Catherine F., M.D., La Jolla, Calif.
Carter, Dorothy L., M.D., Topeka, Kan.
Caster, David U., M.D., Colorado Springs, Colo.
Chappell, Ann L., M.D., San Francisco, Calif.
Chavez, Charles R., M.D., San Diego, Calif.
Cheah, Keong-Chye, M.D., North Little Rock, Ark.
Chisholm, Dugald D., M.D., Atascadero, Calif.
Chrisman, Allan K., M.D., Port Orchard, Wash.
Cohen, Seymour I., M.D., San Francisco, Calif.

DiGaudio, Paul T., M.D., Orinda, Calif.
Dondershine, Harvey E., M.D., Palo Alto, Calif.
Dy, Antonio J., M.D., Honolulu, Hawaii

Egerman, Lynn K., M.D., Beverly Hills, Calif.
Ehrlich, Theodore D., M.D., Los Angeles, Calif.
Engelberg, Steven J., M.D., Seattle, Wash.
Epstein, Robert A., M.D., San Francisco, Calif.
Evans, Dennis U., M.D., Pacific Grove, Calif.

Feigin, Ronald A., M.D., Indian, Ark.
Felthous, Alan R., M.D., Alameda, Calif.
Ferguson, Yvonne B., M.D., Los Angeles, Calif.
Fik, Edward J., M.D., Sepulveda, Calif.
Freedberg, Leonard E., M.D., Kensington, Calif.

Gale, Leonard A., M.D., Hermosa Beach, Calif.
Garrison, Penelope K., M.D., Portland, Ore.
Gerner, Robert H., M.D., Los Angeles, Calif.
Getto, Carl J., M.D., Denver, Colo.
Gislason, I. Lee, M.D., Vancouver, B.C., Canada
Godofsky, Irvin D., M.D., Hermosa Beach, Calif.
Goenjian, Armen K., M.D., Santa Monica, Calif.
Goldman, Herbert P., M.D., Los Angeles, Calif.
Green, Douglas O., M.D., Spokane, Wash.
Greene, Joseph B., M.D., Oakland, Calif.
Griswold, Robert L., M.D., Stockton, Calif.
Gunn, Michela F., M.D., Los Angeles, Calif.
Guthrie, Michael B., M.D., Colorado Springs, Colo.

Hague, William H., M.D., Mercer Island, Wash.
Halpert, E. Stephen, M.D., Tampa, Fla.
Hansen, Stanley F., M.D., Arcadia, Calif.
Harris, Robert F., M.D., Palo Alto, Calif.
Hart, Kent B., M.D., Davis, Calif.
Hartley, Vernon L., M.D., Las Vegas, Nev.
Henderson, Lawrence W., M.D., San Francisco, Calif.
Herman, Robert M., M.D., Los Gatos, Calif.
Hoffman, Robert S., M.D., Los Angeles, Calif.
Hoffman, Theodore E. Jr., M.D., Travis AFB, Calif.

Idzorek, Thomas S., M.D., Scottsdale, Ariz.
Imani, Pouran A., M.D., Chicago, Ill.

Joffe, Joy R., M.D., Baltimore, Md.
Johnson, Martin A., M.D., Sacramento, Calif.
Jorgensen, Judith A., M.D., La Jolla, Calif.

Kallen, Lowell H., M.D., San Francisco, Calif.
Karols, Kenneth E., M.D., La Jolla, Calif.
Kaufman, Herschel J., M.D., Santa Barbara, Calif.
Kirstein, Larry S., M.D., Davis, Calif.
Klevens, Stephen S., M.D., Los Angeles, Calif.
Knapp, Robert S., M.D., Piedmont, Calif.
Kopolow, Louis E., M.D., Rockville, Md.
Korpell, Herbert S., M.D., Berkeley, Calif.
Krasnow, Robert W., M.D., Los Angeles, Calif.

Larson, John W., M.D., Stockton, Calif.
Leibel, Elizabeth A., M.D., Millbrae, Calif.
Leigh, Fiona, M.D., Los Angeles, Calif.
Leritz, Arthur F., M.D., Spokane, Wash.
Lieberman, Barry S., M.D., Beverly Hills, Calif.

Mahakian, Charles G., M.D., Walnut Creek, Calif.
Manasse, Gabriel O., M.D., Topanga, Calif.
Marsh, Charles F., M.D., Carmichael, Calif.
Melson, Stephen J., M.D., Seattle, Wash.
Merrin, Edward L., M.D., San Francisco, Calif.

Miller, Barry, M.D., La Jolla, Calif.
Morong, Eugene, M.D., Lancaster, Calif.
Morrison, Andrew L., M.D., Indianapolis, Ind.

Naifeh, Sam C., M.D., San Mateo, Calif.
Neborsky, Robert J., M.D., Del Mar, Calif.
Nell-Badra, Rocio, M.D., Norristown, Pa.
Nelson, Gerald E., M.D., Del Mar, Calif.
Newton, William L., M.D., Marina Del Rey, Calif.
Nissanka, Manage D., M.D., Vernon, Conn.

Pack, Allen T., M.D., Venice, Calif.
Pariewski, Marta E., M.D., Los Angeles, Calif.
Peters, John A., M.D., Littleton, Colo.
Potkin, Steven G., M.D., Washington, D.C.
Pranger, Margaret A., M.D., Napa, Calif.
Prentice, Glenn D., M.D., Evergreen, Colo.
Purchatzke, Gerald, M.D., Denver, Colo.

Reid, William H., M.D., Santa Fe, N.M.
Ricaurte, Eduardo A., M.D., Rock Island, Ill.
Rice, Matilda, M.D., Old Westbury, N.Y.
Rigolin, Enio A., M.D., Glencoe, Ill.
Riopelle, Robert J., M.D., San Francisco, Calif.
Robinson, Richard S., M.D., Menlo Park, Calif.
Roesler, Thomas A., M.D., Seattle, Wash.
Roth, Janet H., M.D., Berkeley, Calif.
Rothbard, Richard D., M.D., Los Angeles, Calif.
Ruben, Irwin, M.D., Los Angeles, Calif.
Ruggles, James A., M.D., Stockton, Calif.
Rutt, Carl N., M.D., Sioux Falls, S.D.

Sabaratnam, S.K.R.N., M.D., Los Angeles, Calif.
Sarabia, Fermin, M.D., San Antonio, Tex.
Schiff, Matthew M., M.D., W. Long Branch, N.J.
Schneller, Sylvia J., M.D., Metairie, La.
Shah, Manhula A., M.D., Shaker Heights, Ohio
Sherman, Daniel A., M.D., Des Moines, Wash.
Silver, Richard, M.D., Moraga, Calif.
Silverman, David L., M.D., Las Vegas, Nev.
Silverstein, Ronald T., M.D., Long Beach, Calif.
Soghor, David N., M.D., Beverly Hills, Calif.
Soghor, Doris S., M.D., Beverly Hills, Calif.
Solomon, Maurice C., M.D., Colorado Springs, Colo.
Stein, Diane C., M.D., Seattle, Wash.
Straprans, Vija, M.D., Palo Alto, Calif.
Stulberg, Michael V., M.D., Santa Barbara, Calif.
Swartz, Herbert M., M.D., San Francisco, Calif.

Van Natta, John W. Jr., M.D., Palo Alto, Calif.
Venn-Watson, Patricia, M.D., San Diego, Calif.

Waldron, Patricia G., M.D., Hidden Hills, Calif.
Walker, Jeri A., M.D., Los Gatos, Calif.
Weisenfreund, Jochanan M., M.D., New York, N.Y.
Westmen, Lincoln H., M.D., Phoenix, Ariz.
Whyman, Andrew D., M.D., San Francisco, Calif.
Williams, David H., M.D., San Francisco, Calif.
Wiswesser, George A., M.D., West Reading, Pa.

Yano, Brian S., M.D., Honolulu, Hawaii
Yudofsky, Stuart C., M.D., New York, N.Y.

NEUROLOGY

Al-Wardi, Dhia A., M.D., Pasadena, Calif.
Baraff, Robert, M.D., Riverside, Calif.
Beckley, Dennis J., M.D., Benicia, Calif.
Bergman, Sander E., M.D., Walla Walla, Wash.
Bissell, John A., M.D., Sacramento, Calif.
Burcar, Patricia J., M.D., White Ridge, Colo.
Davison, William C., M.D., APO Seattle, Wash.
Diamond, Ivan, M.D., San Francisco, Calif.
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Ginsberg, Arthur H., M.D., Seattle, Wash.
Gollub, Marshall, M.D., Sacramento, Calif.
Gorbunoff, William, M.D., Rancho Palos Verde, Calif.
Hasan, Malik M., M.D., Pueblo, Colo.
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Mallison, Robert A., M.D., San Jose, Calif.
Mladinich, E. Kenneth, M.D., Albuquerque, N.M.
Newton, Fredric H., M.D., San Francisco, Calif.
Nichols, Buddy R., M.D., Presidio of San Francisco, Calif.
Paramesh, Kannen, M.D., Woodland Hills, Calif.
Paruchabutr, Ronakorn, M.D., New York, N.Y.
Robinson, Lewis, M.D., Billings, Mont.
Ruffman, Joel D., M.D., Los Angeles, Calif.
Sax, Richard M., M.D., Huntington Beach, Calif.
Schostal, Clifford J., M.D., North Bend, Ore.
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Spire, Jean-Paul C., M.D., Chicago, Ill.
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Stent, Raymond M., M.D., Torrance, Calif.
Stevens, E. T., M.D., Chicago, Ill.
Thompson, David A., M.D., Los Angeles, Calif.
Tosches, William A., M.D., Colorado Springs, Colo.
Traviesa, Daniel C., M.D., San Francisco, Calif.
Wasterlain, Claude G., M.D., Granada Hills, Calif.
Wilson, Malcolm E., M.D., Concord, Calif.
Woods, Roger K., M.D., Santa Monica, Calif.

CHILD NEUROLOGY

Carnay, Laurence D., M.D., Paramount, Calif.
Clay, Susan A., M.D., Glendale, Calif.
Goldstein, Michael L., M.D., Salt Lake City, Utah
Halverson, Dea, M.D., San Jose, Calif.
Holtzman, S. David, M.D., Stanford, Calif.
Skoglund, R. R., M.D., San Diego, Calif.

The following successfully completed the Board examination given by the Committee on Certification in Child Psychiatry in Chicago, Ill., March 14–15, 1977.

CHILD PSYCHIATRY

Alfie, Salomon, M.D., Merriam, Kan.
Alpern, Robert J., M.D., Smyrna, Ga.
Andrulonis, Paul A., M.D., West Hartford, Conn.
Atwood, Sue Culbertson, M.D., Memphis, Tenn.
Ball, Leonard D. III, M.D., Gulfport, Miss.
Barr, Dojna Galich, M.D., Joliet, Ill.
Barry, Ronald Makepiece, M.D., Baltimore, Md.
Bauermeister, Martin, M.D., Boston, Mass.
Belfer, Myron Lowell, M.D., Brookline, Mass.
Berkowitz, Robert, M.D., Lakewood, N.J.

Bird, Hector Ramon, M.D., New York, N.Y.
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Briscoe, William Cole, M.D., McLean, Va.
Brooks, Joae Graham, M.D., Brookline, Mass.
Brunt, Charles Hal, M.D., Memphis, Tenn.

Colligan, Joseph F., M.D., Columbia, S.C.

Dank, Gerald M., M.D., Boulder, Colo.
Demski, Robert Stanley, M.D., San Antonio, Tex.
Desai, Rajesh B., M.D., Flushing, N.Y.
Dogan, Kutas Tavlan, M.D., Carrboro, N.C.

Eichten, John Gregory, M.D., Minneapolis, Minn.
Etezady, M. Hossein, M.D., Gulph Mills, Pa.

Ferguson, Barbara Bess, M.D., Pacific Palisades, Calif.
Fernandopulle, B. Gregory C., M.D., Towson, Md.
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Frank, Sheldon M., M.D., Scarsdale, N.Y.

Garfinkel, Marc Edward, M.D., Pittsburgh, Pa.
Goldfine, Peter Elliot, M.D., Cape Elizabeth, Me.
Gomez, Humberto, M.D., Richmond, Va.
Gonzalez, Victor Rodolfo, M.D., Atlanta, Ga.
Grad, Gary Jordan, M.D., New York, N.Y.

Harding, Richard Kent, M.D., Bonita, Calif.
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Husain, Syed Arshad, M.D., Columbia, Mo.

Ibay, Manuel Sebastian, M.D., Searington, N.Y.

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Kim, Seungtai Peter, M.D., Bronxville, N.Y.
Kolansky, S. Kalman, M.D., Alexandria, Va.
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Lees, James M., M.D., Pittsburgh, Pa.
Levenson, Bert S., M.D., San Francisco, Calif.
Lewin, Charles Jay, M.D., Denver, Colo.
Liebman, Ronald, M.D., Wynnewood, Pa.
Looker, Andrew N., M.D., New York, N.Y.
Looney, John G., M.D., Dallas, Tex.
Loutsch, Erica Maria Kimelman, M.D., Pelham, N.Y.
Lunianski, Irwin L., M.D., Santa Barbara, Calif.
Lymberis, Maria Triantaphyllou, M.D., Santa Monica, Calif.

MacVicar, Katherine, M.D., Berkley, Calif.
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Quiroga, Nydia Mayolo, M.D., Jackson, Miss.

Rigamer, Elmore F. Jr., M.D., New Orleans, La.
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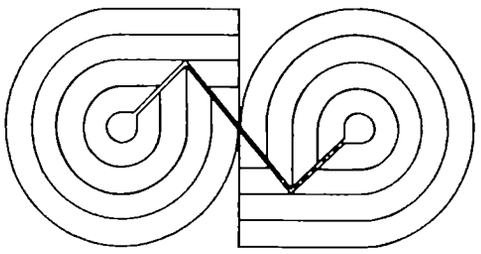
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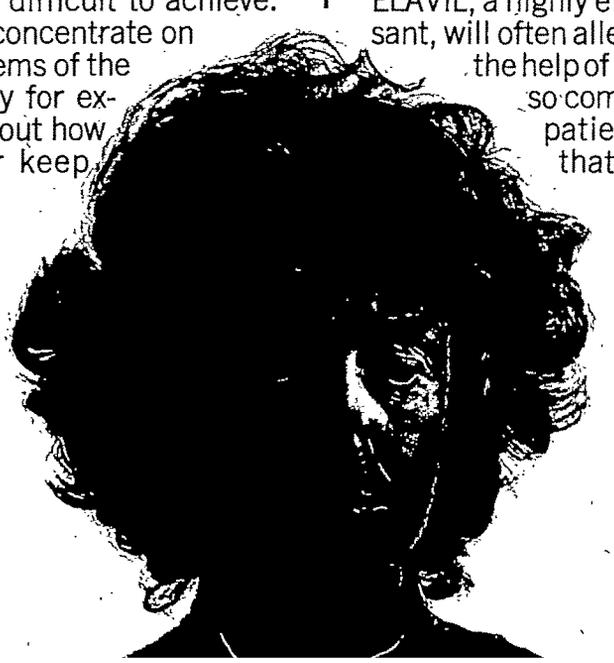
penetrating the symptom barrier to productive psychotherapy

symptoms often interfere with therapy

In clinically significant depression, the patient often becomes barricaded behind his own symptoms during psychotherapy. Because these symptoms can be so sharply defined and somatic in expression, he may reject the fact that they are psychic in origin. Sleep disturbance, gastrointestinal complaints, anorexia, chronic fatigue, and other psychosomatic symptoms may so preoccupy—or overwhelm—the depressed patient that truly productive therapeutic sessions are difficult to achieve. Instead of being able to concentrate on the core emotional problems of the depression itself, he may for example go on at length about how broken his sleep is or keep reiterating (while the therapeutic hour slips by) how physically miserable he feels.

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to help penetrate the symptom barrier in depression requiring medication

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well as other "barrier symptoms" may be lessened to the point where they no longer come between you and the patient.

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ELAVIL should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks; or in children under 12. Patients with cardiovascular disorders should be watched closely. Safe use during pregnancy and lactation has not been established. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Hospitalize as soon as possible any patient suspected of having taken an overdose.

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For a brief summary of prescribing information, please see following page.

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addendum

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Tablets

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ONCE-DAILY DOSAGE SCHEDULE FOR ADULT OUTPATIENTS:

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Usual starting dosage



A single 100-mg tablet
Maximum starting dosage

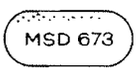


A single 50-mg tablet
Minimum starting dosage



Dosage may be increased by 25 or 50 mg as necessary until a total of 150 mg per day is reached.

A single 150-mg tablet
Maximum daily dosage



ELAVIL MAY ALSO BE PRESCRIBED IN DIVIDED DAILY DOSES

The 25-mg tablet



This strength may prove useful when therapy is initiated with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime dose.

The 10-mg tablet



This strength may prove useful for patients who require lower doses, e.g., adolescent and elderly patients. For these patients who can not tolerate higher doses, 10 mg three times a day with 20 mg at bedtime may be satisfactory.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.
When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

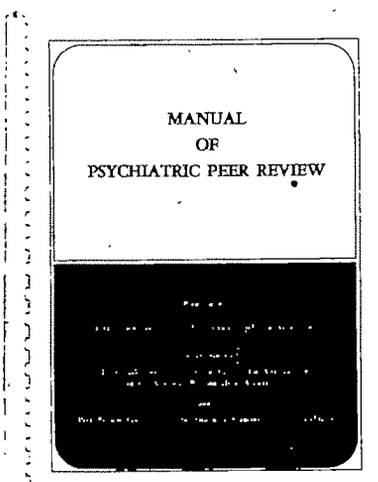
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"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

Robert W. Gibson, M.D.
President

Part I titled PSYCHIATRIC PEER REVIEW contains sections on Basic Relationships and Responsibilities, Appointment of Peer Review Committees, Funding, Operations and Procedures, Medical Care Evaluation Studies, Appeals Processes, Special Problems, Confidentiality, Educational Aspects, Medical Ethics, Advocacy, Interdisciplinary Aspects, with Appendices on Model Screening Format for Inpatient Treatment for Alcoholism, Model Screening Format for Outpatient Treatment, Sample Form for Requesting Extension of Office Treatment, Claims Review Guidelines Suggestions, APA Position Papers on Peer Review, Bibliography.

Part II titled PSYCHOANALYTIC PEER REVIEW has sections on Steps in Peer Review for Psychoanalysis, Guidelines for Model Criteria Sets, Pilot Application of Guidelines for Model Criteria Sets, Sample Peer Review Form, Sample data from Pilot Applications of Guidelines, and Glossary of Terms Used.

Part III titled PEER REVIEW AND CHILD PSYCHIATRY contains sections of Special Considerations, Legal Rights of Children, Issue of Confidentiality, Conclusions, References, and Appendices on Model Screen Criteria Format for Intermediate Care, for Acute Care in a Short Stay Facilities, and for Partial Hospitalization, Outpatient Diagnostic Evaluation Procedures, and Outpatient Treatment for Children and Adolescents.

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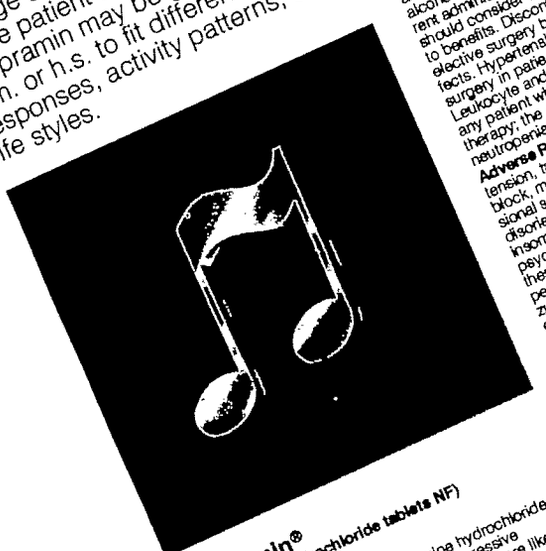
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Indications: Norpramin (desipramine hydrochloride tablets NF) is indicated for the relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Desipramine hydrochloride should not be given within two weeks of treatment with a monoamine oxidase inhibitor. Contraindications include the acute recovery period following myocardial infarction and hypersensitivity to the drug. Cross sensitivity with other dibenzazepines is a possibility.

Warnings: 1. Extreme caution should be used in patients: (a) with cardiovascular disease, (b) with a history of urinary retention or glaucoma, (c) with thyroid disease or those on thyroid medication, (d) with a history of seizure disorder. 2. This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. 3. *Use in Pregnancy:* Safe use during pregnancy and lactation has not been established. 4. *Use in Children:* Norpramin is not recommended for use in children. 5. This drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. Therefore, the patient should be cautioned accordingly.

Precautions: This drug should be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with drugs of this class. If possible, dispense in child-resistant containers. It should be kept out of reach of children.–Reduce dosage, or alter treatment, if serious adverse effects occur. Norpramin therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates and may cause exacerbation of psychosis in schizophrenic patients. Use cautiously with anticholinergic or sympathomimetic drugs. Response to alcoholic beverages may be exaggerated. In the concurrent administration of ECT and antidepressant drugs one should consider the possibility of increased risk relative to benefits. Discontinue as soon as possible prior to elective surgery because of possible cardiovascular effects. Hyperpyrexia has been observed during surgery in patients on desipramine hydrochloride. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is neutropenia.

Adverse Reactions: *Cardiovascular:* hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke. *Psychiatric:* confusional states (especially in the elderly); hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis. *Neurological:* numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus. *Anticholinergic:* dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, hypotonic bladder. *Allergic:* skin rash, petechiae, urticaria, itching, photosensitization, edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs. *Hematologic:* bone marrow depressions including agranulocytosis, thrombocytopenia, purpura. *Gastrointestinal:* anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, eosinophilia, black tongue. *Endocrine:* gynecomastia, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels. *Other:* jaundice (simulating obstructive), altered liver function; weight gain or loss; perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia. *Withdrawal Symptoms:* Though not indicative of addiction, abrupt cessation after prolonged therapy may produce nausea, headache and malaise.

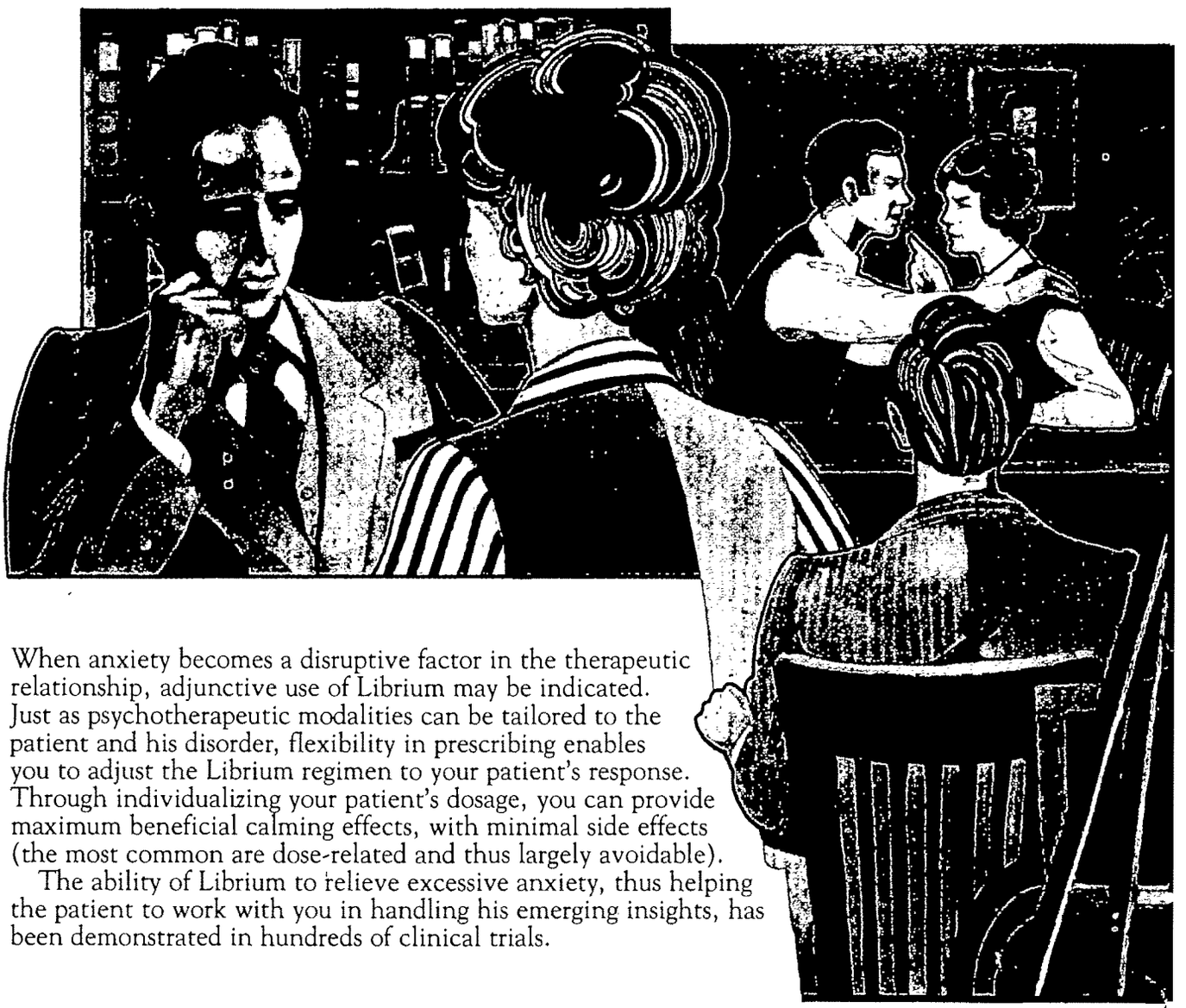
Dosage and Administration: *Usual adult dose:* 100 mg. to 200 mg. per day. Dosages above 200 mg. per day are not recommended. *Initial therapy:* Should be administered at a low level and increased according to tolerance and response. It may be administered in divided doses or a single daily dose. *Maintenance:* Lower adequate dose once daily to maintain remission. *Adolescent and geriatric patient dose:* 25 mg. to 100 mg. per day in single or divided doses. Dosages above 100 mg. are not recommended.

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LIBRIUM® (chlordiazepoxide HCl)
ADAPTABLE TO MANY MODALITIES



When anxiety becomes a disruptive factor in the therapeutic relationship, adjunctive use of Librium may be indicated. Just as psychotherapeutic modalities can be tailored to the patient and his disorder, flexibility in prescribing enables you to adjust the Librium regimen to your patient's response. Through individualizing your patient's dosage, you can provide maximum beneficial calming effects, with minimal side effects (the most common are dose-related and thus largely avoidable).

The ability of Librium to relieve excessive anxiety, thus helping the patient to work with you in handling his emerging insights, has been demonstrated in hundreds of clinical trials.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may oc-

WHEN ANXIETY BLOCKS PROGRESS



adjunctive
LIBRIUM® IV
chlordiazepoxide HCl/Roche
THE ANXIETY-SPECIFIC

cur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

Supplied: Librium® Capsules containing 5 mg, 10 mg or 25 mg chlordiazepoxide HCl. Libritabs® Tablets containing 5 mg, 10 mg or 25 mg chlordiazepoxide.

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Roche announces a new psychiatric series:
"Eclecticism: The New Flexibility In Psychotherapy."

Look for it in your mail.



Roche has prepared a new series on how therapists are combining several modalities to handle different disorders. The first in this series, "A Conversation with Dr. Helen Singer Kaplan on The Eclectic Approach to Treating Sexual Dysfunction," is being mailed to psychiatrists.

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for institutions and agencies concerned with the care of the mentally disabled

Enrollment in the Hospital & Community Psychiatry Service brings multiple copies of *Hospital & Community Psychiatry* into member agencies every month, keeping staff up to date on developments and issues in the mental health field, offering new ideas and fresh perspectives, and serving as a useful resource in staff development and training programs.

Hospital & Community Psychiatry is just one of the benefits of membership in the H&CP Service. Others include a film library containing more than a hundred films specially chosen for their usefulness in staff development and community education programs; supplementary mailings of important books, reports, articles, or other material of special interest to administrators or clinicians; reduced registration fees at the annual fall Institute on Hospital & Community Psychiatry; and, on request, information and consultation from the professional staff of the American Psychiatric Association. The H&CP Service also sponsors the annual Achievement Awards competition, which gives special recognition to outstanding programs for the mentally ill and mentally retarded.

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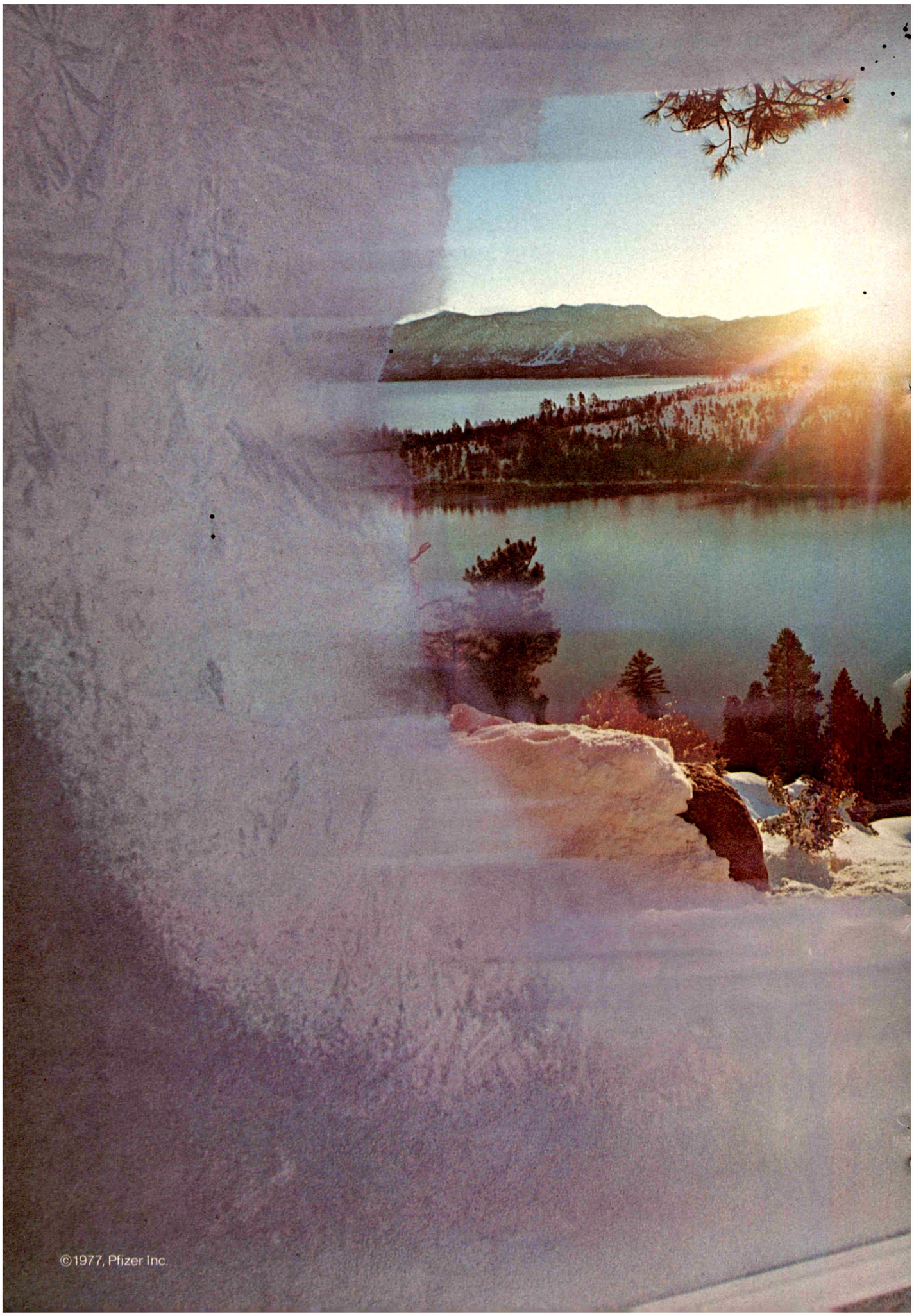
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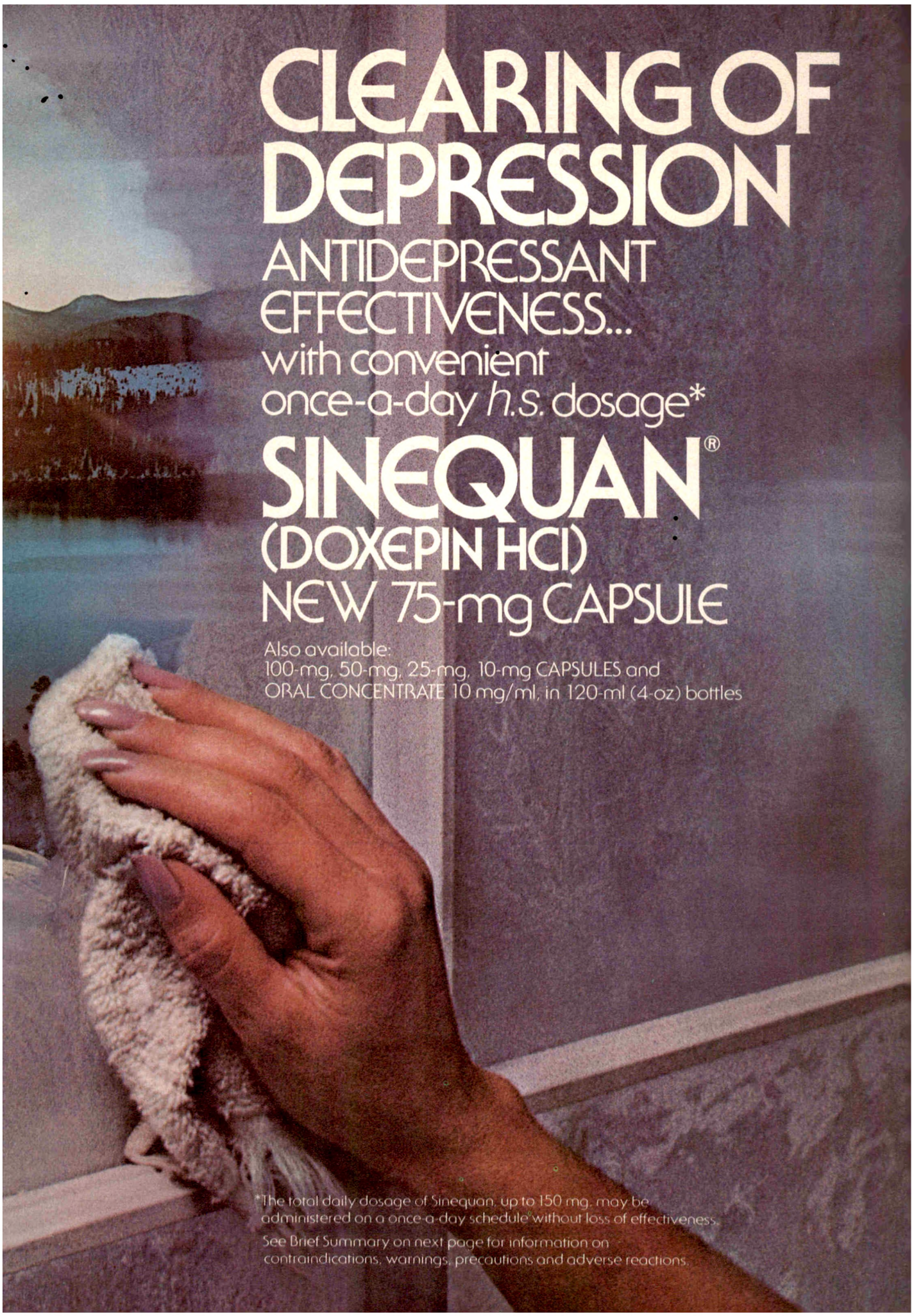
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INTRODUCING
NEW
SINEQUAN® 75 mg
(DOXEPIN HCl) CAPSULE



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CLEARING OF DEPRESSION
ANTIDEPRESSANT EFFECTIVENESS...
with convenient once-a-day h.s. dosage*
SINEQUAN®
(DOXEPIN HCl)
NEW 75-mg CAPSULE
Also available:
100-mg, 50-mg, 25-mg, 10-mg CAPSULES and
ORAL CONCENTRATE 10 mg/ml, in 120-ml (4-oz) bottles
*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.
See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

IN THE TREATMENT OF CLINICAL DEPRESSION—

NEW SINEQUAN® 75 mg (DOXEPIN HCl) CAPSULE

EXTENDS THE RANGE OF DOSAGE STRENGTHS

PROVIDING ANTIDEPRESSANT EFFECTIVENESS

IN ADDITION TO OTHER, OFTEN BENEFICIAL, PROPERTIES...:

CONVENIENT ONCE-A-DAY *h.s.* DOSAGE

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY GOOD TOLERATION

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have occasionally been reported. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug. Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting dose of 25 mg t.i.d. is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients an initial dose of 50 mg t.i.d. may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

As an alternative regimen, the total daily dosage, up to 150 mg, may be given on a once-a-day schedule without loss of effectiveness. This dose may be given at bedtime.

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. Available as capsules containing doxepin HCl equivalent to 10 mg, 25 mg, 50 mg, 75 mg, and 100 mg doxepin in bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). SINEQUAN 25 mg and 50 mg are also available in bottles of 5000. SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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Just Published . . .

The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

Paperback edition—$5.00 each, may be ordered from the AMERICAN PSYCHIATRIC ASSOCIATION, Publication Sales, 1700 18th St., N.W., Washington, D.C. 20009.

Hardback edition—$7.95 may be ordered from Basic Books, Inc., 10 East 53rd Street, New York, New York 10022.

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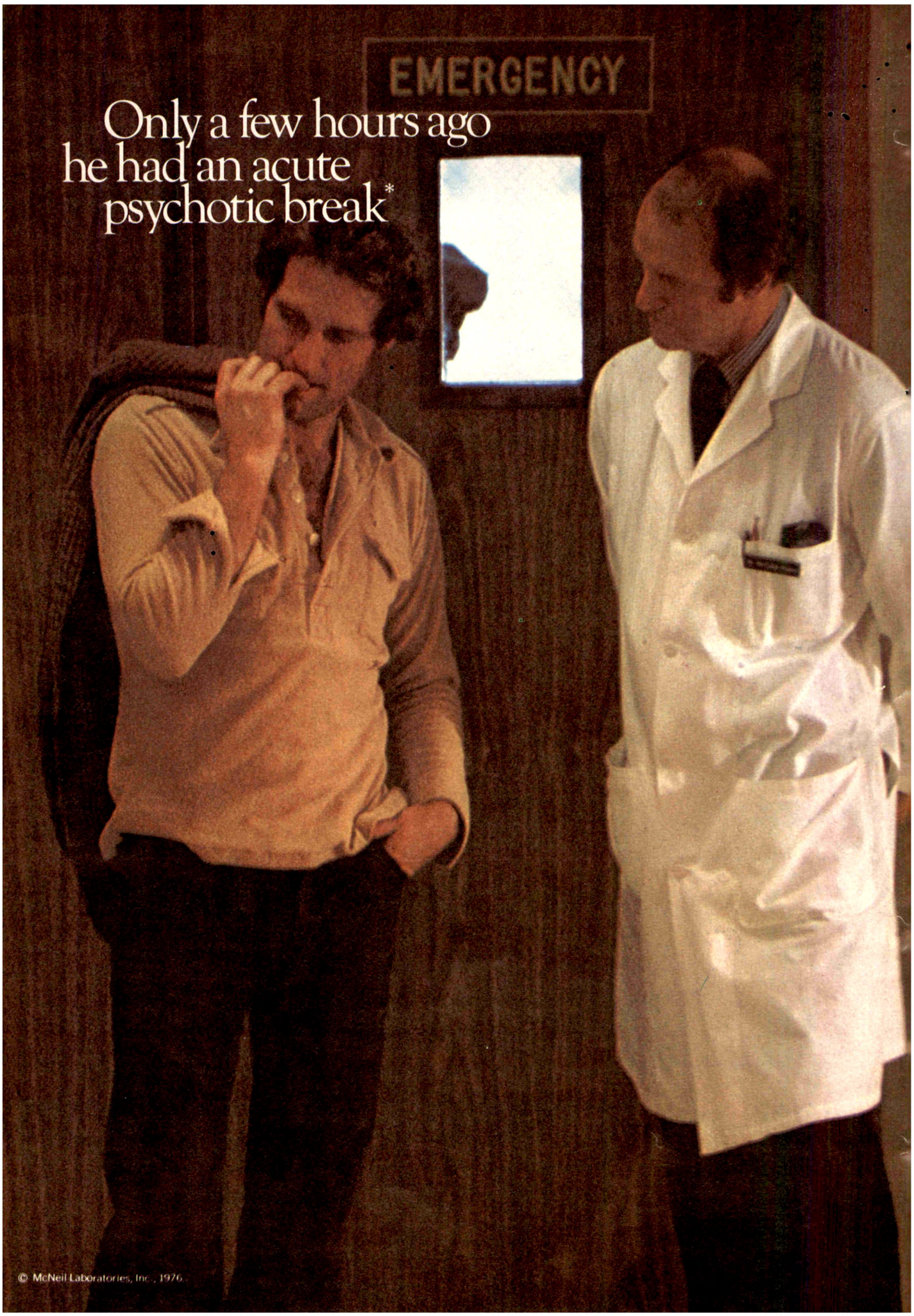
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Haldol®
(haloperidol)
injection

For rapid control with minimal risk of hypotensive crises

Rapidly controls acute psychotic symptomatology[1-4] associated with: functional disorders such as schizophrenia, mania and paranoid delusional states; alcoholic withdrawal; drug abuse or withdrawal. With close patient monitoring and adequate dosage levels, initial control of symptoms has often been obtained with IM dosage in as little as one to two hours, permitting early transfer to the oral form.

Often enables you to avoid hospitalization.[5-7]
Although some instances of drowsiness have been reported, marked sedation is rare,[1,4-6] often leaving the patient ambulatory, alert and more accessible to your efforts to establish rapport.

Common side effects easily controlled.[3,5]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

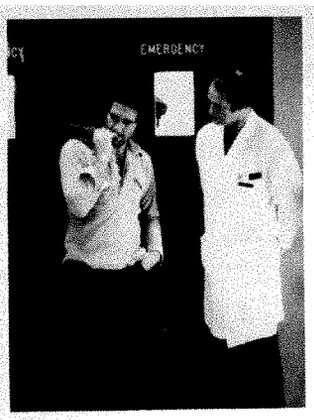
Reduces risk of certain troublesome reactions.[1-6,8,9]
Transient hypotension occurs rarely; severe orthostatic hypotension has not been reported. Also, HALDOL haloperidol is unlikely to interact adversely with commonly abused drugs.

Can be administered at any usual I.M. site
—rarely causing additional patient discomfort[2,8] or agitation due to local irritation or stinging.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Please turn page for information relating to Indications, Contraindications, Warnings, Precautions and Adverse Reactions.

Important: Full directions for use should be read before HALDOL haloperidol is administered or prescribed.



Haldol® (haloperidol)

tablets/concentrate/injection

For rapid control in psychiatric emergencies with minimal risk of hypotensive crises

A Dosage Form for Every Need:

5 tablet strengths for convenience in individualizing dosage: ½ mg., 1 mg., 2 mg., 5 mg. and 10 mg.

A tasteless, odorless, colorless liquid concentrate for better patient acceptability: 2 mg. per ml.

A rapid-acting injection for psychiatric emergencies: 5 mg. per ml., with 1.8 mg. methylparaben and 0.2 mg. propylparaben per ml., and lactic acid for pH adjustment to 3.4±0.2.

Indications: HALDOL haloperidol is indicated for use in the management of manifestations of psychotic disorders.

It is also indicated for the control of tics and vocal utterances of Gilles de la Tourette's syndrome.

Contraindications: HALDOL haloperidol is contraindicated in patients who are severely depressed, comatose, have CNS depression due to alcohol or other centrally-acting depressants, have Parkinson's disease or are hypersensitive to this drug.

Warnings: ***Usage in Pregnancy:*** Safe use of HALDOL haloperidol in pregnancy and lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the possible benefits of the drug be weighed against the potential hazards. A case of phocomelia in an infant whose mother received haloperidol along with a number of other medications during the first trimester of pregnancy has been reported (a causal relationship was not established in this case). Animals receiving 2 to 20 times the maximum human dose of haloperidol orally and/or parenterally showed increased incidence of resorption, reduced fertility, delayed delivery, dose-related pup mortality (presumably due to lack of maternal care reflecting CNS depression).

Usage in Children: Safety and effectiveness in children have not been established; therefore, this drug is not recommended for use in the pediatric age group.

General: Cases of bronchopneumonia, some fatal, have followed the use of major tranquilizers, including haloperidol. It has been postulated that lethargy and decreased sensation of thirst may lead to dehydration, hemoconcentration and reduced pulmonary ventilation. If these signs and symptoms appear, especially in the elderly, the physician should institute remedial therapy promptly. Although not reported with HALDOL haloperidol, decreased serum cholesterol and/or cutaneous and ocular changes have been reported in patients receiving chemically-related drugs. HALDOL haloperidol may impair the mental and/or physical abilities required for the performance of hazardous tasks such as operating machinery or driving a motor vehicle. The ambulatory patient should be warned accordingly. The use of alcohol should be avoided due to possible additive effects and hypotension.

Precautions: HALDOL haloperidol should be administered cautiously to patients: (1)—with severe cardiovascular disorders, because of the possibility of transient hypotension and/or precipitation of anginal pain. Should hypotension occur and a vasopressor be required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur. (2)—receiving anticonvulsant medication, because HALDOL haloperidol may lower the convulsive threshold. Adequate anticonvulsant therapy should be maintained concomitantly. (3)—with known allergies, or with a history of allergic reactions to drugs. (4)—receiving anticoagulants, since an isolated instance of interference occurred with the effects of one anticoagulant (phenindione).

If concomitant antiparkinson medication is required, it may have to be continued after HALDOL haloperidol is discontinued because of the difference in excretion rates. If both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson agents, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used to control mania in cyclic disorders there may be a rapid mood swing to depression.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions*—Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which usually were mild to moderately severe and reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Administration of antiparkinson drugs may be required for control of such reactions. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases. *Persistent Tardive Dyskinesia*—Tardive dyskinesia may appear during long-term therapy or after therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear irreversible. There is no known effective treatment. All antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent. *Other CNS Effects*—Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms including hallucinations. ***Cardiovascular Effects:*** Tachycardia and hypotension. ***Hematologic Effects:*** Reports have appeared of mild and usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis. Agranulocytosis has rarely been reported and then only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice have been reported, although a causal relationship has not been established. ***Dermatologic Reactions:*** Maculopapular and acneiform skin reactions and isolated cases of photosensitivity and loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

Complete dosage information available in insert which accompanies each package (or on request).

The use of the injectable form is intended for the acutely agitated psychotic patient with moderately severe to very severe symptoms.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed. 9/74

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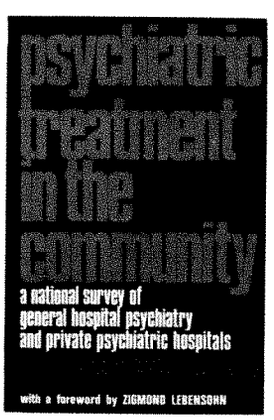
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Phillip H.* was an engineer with a problem... but now he's solving problems for industry

Patient name	Phillip H.*	Sex: Male	Age: 29
Marital status S. (M.) W. D.		Occupation:	Mechanical engineer

History: Studious, bright, only son; much older sister. Father an invalid; mother, a successful business woman. A "loner" with multiple psychosomatic illnesses, he started dating late and married first girl friend, several years older

Onset of illness: Upset at wife's pregnancy, expressed strong guilt feelings after her miscarriage; said people accused him of "murder." Unable to eat or sleep properly, felt he was "losing his mind." Unable to work, obtained leave of absence and was referred to psychiatrist

Diagnosis: Acute schizophrenia

Treatment: LOXITANE® Loxapine Succinate 25 mg b.i.d. increased to 50 mg t.i.d. over 2 weeks. Supportive psychotherapy three times a week

Therapeutic course: First week, eating and sleeping better. Guilt feelings expressed less often. Mild akathisia responded to appropriate antiparkinson medication

Second week, communicating better. Feared that "fatherhood would develop into rivalry," feared loss of wife's love

Third week, no delusions or hallucinations, relating better, but still to only one person at a time

Affect still blunted at one month, otherwise asymptomatic. LOXITANE decreased to 50 mg b.i.d.; antiparkinson medication continued

Follow-up: 10 weeks; at work for one week, seems to be adjusting well. Antiparkinson medication discontinued; psychotherapy weekly, LOXITANE 25 mg t.i.d.

*Not an actual patient. The collection of clinical data upon which this simulation is built illustrates the action of LOXITANE® Loxapine Succinate as reported in clinical studies.



He's adjusting well in his job, and with psychotherapy and medication, he also can be helped to feel more secure, less threatened in his relationships with others.

Loxitane®
LOXAPINE SUCCINATE
Lederle

For the symptoms that interrupt careers

See LOXITANE prescribing information on following page for indications, warnings and precautions and for more detailed information concerning side effects.

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For the symptoms that interrupt careers

Recommended Daily Dosage

Initial Dosage	MILD 10 mg b.i.d.	MODERATE 10 mg t.i.d. or q.i.d.	SEVERE 25 mg b.i.d.
First 7 to 10 Days	Increase dosage until psychotic symptoms are controlled. Dosage should not exceed 250 mg/day. Usual dosage during titration: 50 to 150 mg/day.		
Maintenance Dosage	Adjust to lowest effective level. Usual maintenance dosage: 60 to 100 mg/day. Many patients are controlled with dosages as low as 20 to 60 mg/day.		



Brief Summary

LOXITANE® Loxapine Succinate Capsules
OR
LOXITANE® C Loxapine Hydrochloride Oral Concentrate

Description: A dibenzoxazepine compound, representing a new subclass of tricyclic antipsychotic agent, chemically distinct from the thioxanthenes, butyrophenones, and phenothiazines.

Indications: For the manifestations of schizophrenia. Loxapine has not been evaluated for the management of behavioral complications in mental retardation, and therefore cannot be recommended.

Contraindications: In comatose or severe, drug-induced depressed states (alcohol, barbiturates, narcotics, etc.); and in individuals with known hypersensitivity to the drug.

Warnings: In Pregnancy: Safe use during pregnancy or lactation has not been established; in pregnancy, nursing mothers, or women of child-bearing potential, weigh potential benefits against possible hazards to mother and child. No embryotoxicity or teratology was observed in studies in rats, rabbits or dogs, although with the exception of one rabbit study, the highest dosage was only two times the maximum recommended human dose and in some studies they were below this dose. Perinatal studies have shown renal papillary abnormalities in offspring of rats treated from mid-pregnancy with doses of 0.6 and 1.8 mg./kg., doses which approximate the usual human dose but which are considerably below the maximum recommended human dose. **In Children:** Studies have not been performed in children; therefore this drug is not for use below the age of 16.

May impair mental and/or physical abilities, especially during the first few days of therapy; warn ambulatory patients about activities requiring alertness (e.g., operating vehicles or machinery), and about concomitant use of alcohol and other CNS depressants.

Precautions: Use with extreme caution in patients with history of convulsive disorders; seizures have been reported in epileptics receiving this drug at antipsychotic dose levels, and may occur even with maintenance of routine anticonvulsant drug therapy. Has an antiemetic effect in animals; this effect in man may mask signs of overdosage of toxic drugs and obscure conditions such as intestinal obstruction and brain tumor. Use with caution in patients with cardiovascular disease. Increased pulse rates in the majority of patients receiving antipsychotic doses and transient hypotension have been reported. In hypotension requiring vasopressor therapy, preferred drugs may be norepinephrine or angiotensin. Usual doses of epinephrine may be ineffective because of inhibition of its vasopressor effect by loxapine. Possibility of ocular toxicity from loxapine cannot be excluded at this time, therefore observe carefully for pigmentary retinopathy and lenticular pigmentation (observed in some patients receiving certain other antipsychotic drugs for prolonged periods). Because of possible anticholinergic action, use with caution in patients with glaucoma or a tendency to urinary retention, particularly with concomitant administration of anticholinergic-type antiparkinson medication.

Adverse Reactions: CNS: Adverse effects other than extrapyramidal, infrequent. Drowsiness, usually mild, may occur at beginning of therapy or when dosage is increased, usually subsiding with continued therapy. Incidence of sedation is less than that of certain aliphatic phenothiazines and slightly more than piperazine phenothiazines. Dizziness, faintness, staggering gait, muscle twitching, weakness, and confusional states have been reported. Extrapyramidal reactions during use of this drug have been reported frequently, often during the first few days of treatment. In most patients, these involve Parkinson-like symptoms such as tremor, rigidity, excessive salivation, masked facies, akathisia, usually not severe and controlled by dosage reduction or use of antiparkinson drugs in usual dosage. *Less frequent, but more severe:* Dystonias, including spasms of muscles of neck and face, tongue protrusion, oculogyric movement; dyskinesia in the form of choreo-athetoid movements. These sometimes require reduction or temporary withdrawal of drug dosage in addition to appropriate counteractive drugs. *Persistent Tardive Dyskinesia:* May appear in some patients on long-term therapy, or after therapy has been discontinued; the risk greater in the elderly, especially females, on high dosage. These symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements, sometimes accompanied by involuntary movements of extremities). No known effective treatment; discontinue all antipsychotic agents if these symptoms appear. The necessity to reinstitute treatment or increase dosage, or switch to a different antipsychotic agent, may mask syndrome. If medication is stopped when fine vermicular movements of the tongue occur, the syndrome may not develop. *Cardiovascular:* Tachycardia, hypotension, hypertension, lightheadedness, syncope. A few cases of EKG changes similar to those seen with phenothiazines have been reported, not known to be related to loxapine use. *Skin:* Dermatitis, edema (puffiness of face), pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of uncertain etiology seen in a few patients during hot summer months. *Anticholinergic:* Dry mouth, nasal congestion, constipation, blurred vision—more likely to occur with concomitant use of antiparkinson agents. *Other:* Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of uncertain etiology.

Dosage and Administration: Administered orally, usually in 2 to 4 divided doses a day. Adjust dosage to patient's need relative to severity of symptoms and history of response to antipsychotic drugs. Recommended initial dosage is 10 mg. b.i.d.; in severely disturbed patients up to 50 mg. daily. Increase dosage during first 7 to 10 days until psychotic symptoms are controlled. Usual therapeutic and maintenance range is 60 mg.-100 mg. daily. More than 250 mg. daily is not recommended. Maintenance dosage should be at lowest level to control symptoms; many patients have been maintained on 20 mg. to 60 mg. daily.

LOXITANE C Oral Concentrate should be mixed with orange and grapefruit juice shortly before administration. Use only enclosed calibrated dropper.

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Coverage and Utilization of Care for Mental Conditions Under Health Insurance —Various Studies, 1973–74

By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

This book reports six studies that add to the growing body of data demonstrating the feasibility of covering mental illness under health insurance. Although in some ways it may be considered a supplement to APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (Reed, Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

- Utilization of mental benefits under the Blue Cross and Blue Shield plan for federal employees, documenting that a "plateau" in the use of these benefits was reached in 1973–74.
- Comparison of benefits for mental and other illnesses under selected employee health benefit plans.
- Coverage of mental illness under collective bargaining agreements of selected unions.
- Utilization of care for mental conditions under the Canadian health insurance program, which gives the same coverage for mental as for other conditions.
- Updating of information on Blue Cross benefits for hospital care of mental illness.
- Data from selected Blue Cross and Blue Shield plans on coverage and utilization of mental conditions, with emphasis on major medical coverage.

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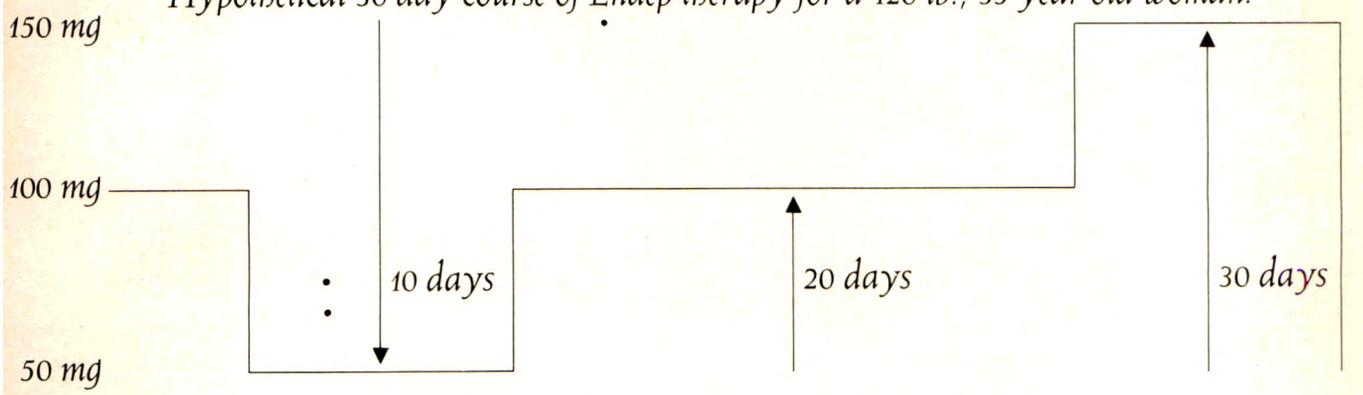
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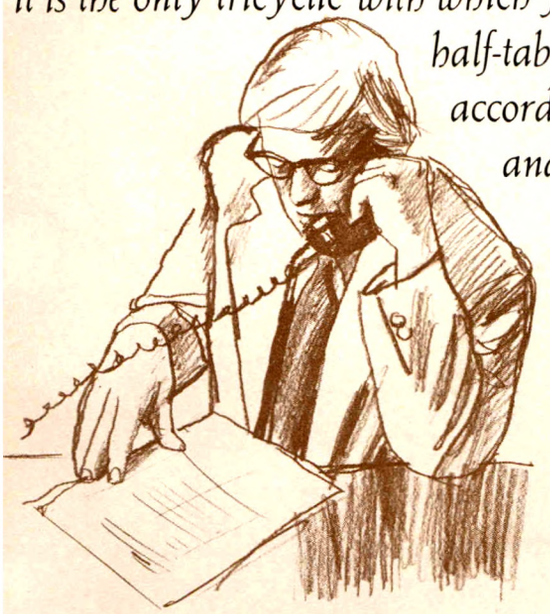
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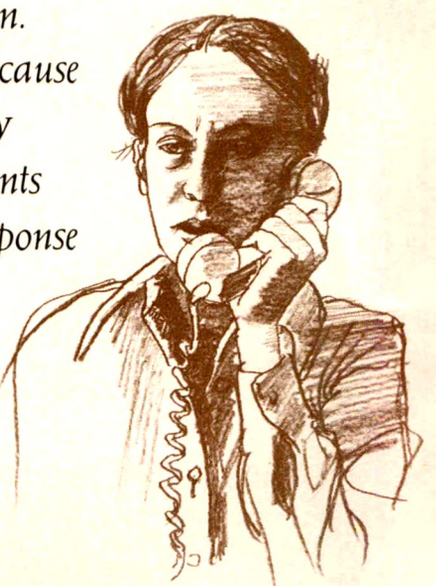
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Half-dose adjustments h.s., depending on progress or side effects.

Because therapeutic levels of amitriptyline vary greatly from patient to patient, reaching the optimal dose requires careful titration. Endep (amitriptyline HCl) greatly facilitates this process because it is the only tricyclic with which you can adjust dosage by half-tablet increments or decrements according to the therapeutic response and/or side effects.





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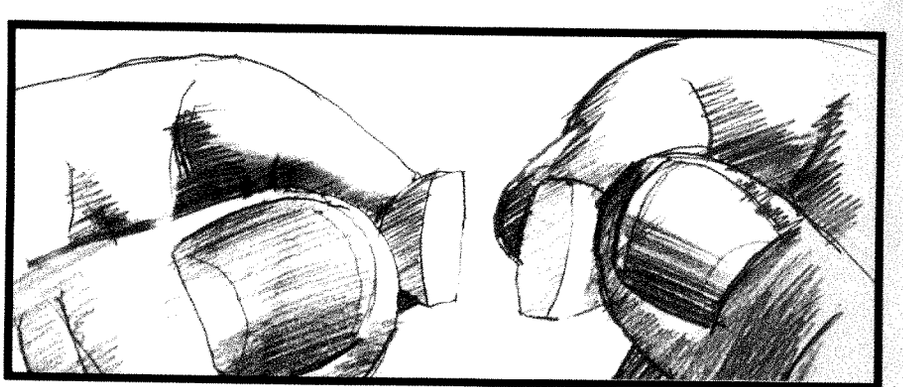
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The half-strength advantage

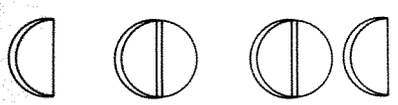
Before prescribing, please consult complete product information, a summary of which appears on the following page.

Practicality is a fundamental fact about Endep®

amitriptyline HCl/Roche

Every tablet of Endep (amitriptyline HCl) is scored for precise, convenient adjustment of dosage.

Half-tablet adjustment of dosage does not require a new Rx.



Single daily *h.s.* dosage promotes compliance and economy.

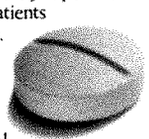
Compliance with practical regimen promotes optimal response.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

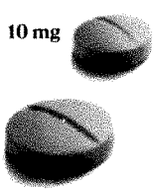
Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.



100 mg

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels.



10 mg

50 mg

25 mg

75 mg

150 mg

Other: Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels, increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier.

Initial Adult Dosage: Outpatients—25 mg *t.i.d.*; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate—50 to 100 mg *h.s.*, gradually increasing *h.s.* dose up to 150 mg/day. Hospitalized Patients—Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day.

Adolescent and Elderly Patients: In general, 10 mg *t.i.d.* with 20 mg *h.s.* may be satisfactory for those who do not tolerate higher doses.

Maintenance Dosage: With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg *b.i.d.* to *q.i.d.*, or 10 mg *q.i.d.* Continue maintenance therapy 3 months or longer to avoid relapse.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg, 75-mg, 100-mg and 150-mg scored tablets—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg, 100 mg and 150 mg), available singly and in trays of 10.

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$\frac{4}{5} \div \frac{7}{8} = \frac{32}{35}$
$\frac{4}{7} + \frac{3}{7} = \frac{7}{7}$

The vulnerable ages

The first epileptic seizure is most likely to occur during early childhood and at the onset of puberty

About 9 out of 10 epileptics experience their first seizure before the age of 20—with the highest incidence between 5 and 7, when children start school, and at the onset of puberty, a time of physiological and psychic turmoil.[1] The most common type, grand mal, occurs in approximately 75% of epileptic children,[1] and more than 50% of patients who suffer initially from petit mal develop grand mal seizures before they reach the age of 16.[2]

Mysoline (primidone) for control of grand mal, psychomotor and focal epilepsy

At the onset and afterwards— used alone or as concomitant therapy, MYSOLINE may reduce the frequency and severity of major motor seizures—or even eliminate them. *Excellent* for control of grand mal. Valuable for control of psychomotor[1,3,4] and focal epilepsy as well.[5]

Add Mysoline when control with other anticonvulsants is inadequate—As concomitant therapy, MYSOLINE can improve seizure control in grand mal and psychomotor epilepsy. The combined use of phenobarbital, diphenylhydantoin, and MYSOLINE may have additive anticonvulsant effects without additive side effects.[6]

Change to Mysoline when other anticonvulsants fail— A changeover to MYSOLINE is frequently warranted when other anticonvulsants must be discontinued because of important side effects, or when grand mal seizures are refractory to phenobarbital, with or without diphenylhydantoin.[7]

Ayerst®

Mysoline® (primidone)

Tablets 250 mg.
50 mg.
Suspension 250 mg./5 cc.

May be the start of a better life for the epileptic

See following page of advertisement for prescribing information.

7538

Mysoline® (primidone)

may be the start of a better life for the epileptic

initial and maintenance therapy for
grand mal, psychomotor and focal epilepsy

BRIEF SUMMARY
(For full prescribing information, see package circular.)

MYSOLINE® Brand of PRIMIDONE
Anticonvulsant

ACTIONS: MYSOLINE acts on the central nervous system to raise seizure threshold or alter seizure pattern. The mechanism(s) of action of anticonvulsant drugs is not known.

Primidone has anticonvulsant activity *per se*. In addition, its two metabolites possess anticonvulsant qualities. The major metabolite is phenylethylmalonamide (PEMA); the other is phenobarbital. In addition to its own anticonvulsant potential, PEMA potentiates phenobarbital.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several days before it can be assessed.

Use in pregnancy: Recent reports strongly suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Reference has been made to primidone in several cases in which it was used *in combination* with other anticonvulsants; but its teratogenicity has not been conclusively demonstrated. The possibility exists that other factors, *e.g.*, genetic factors or the epileptic condition, may contribute to the higher incidence of birth defects. The data also indicate that the great majority of mothers receiving anticonvulsant medication deliver normal infants.

Anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and risk to both mother and the unborn child.

When the nature, frequency, and severity of the seizures do not pose a clear threat to the patient, good medical practice requires that the physician weigh the expected therapeutic benefit of anticonvulsant therapy against possible risk on an individual basis.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

The physician should weigh all of the foregoing considerations when treating and counseling epileptic women of childbearing potential.

PRECAUTIONS: The total daily dosage should not exceed 2 Gm. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid, 15 mg. daily, without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 Gm. per day. The initial dose is 250 mg. Increments of 250 mg. are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 Gm. A typical dosage schedule for the introduction of MYSOLINE (primidone) is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg. daily at bedtime	*2nd Week* 250 mg. b.i.d.
3rd Week 250 mg. t.i.d.	*4th Week* 250 mg. q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg., with gradual weekly increases of 125 mg. a day, to a daily total usually between 500 mg. and 750 mg.

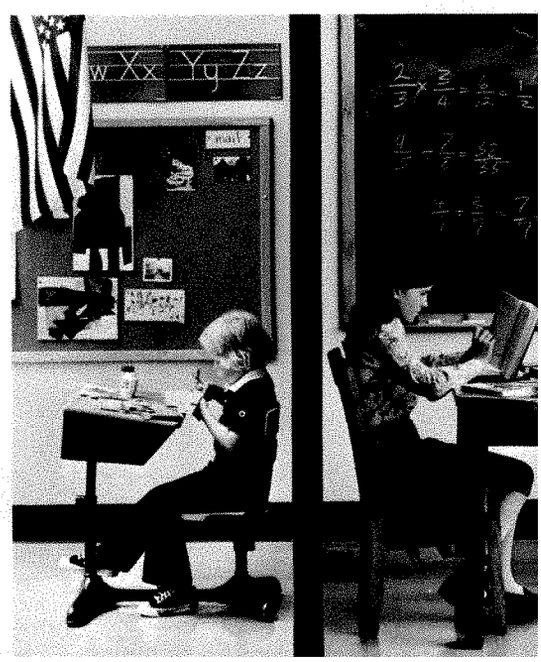
In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg. Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances:

- for initiation of combination therapy
- during "transfer" therapy
- for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.)

HOW SUPPLIED: MYSOLINE *Tablets*—No. 430—Each tablet contains 250 mg. of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg. of primidone (scored), in bottles of 100 and 500. MYSOLINE *Suspension*—No. 3850—Each 5 cc. (teaspoonful) contains 250 mg. of primidone, in bottles of 8 fluid-ounces.

References: 1. Livingston, S.: Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence, Springfield, Ill., Charles C Thomas, 1972, pp. 6, 7, 584. 2. Grossman, H.J.: Ill. Med. J. *135*:260 (Mar.) 1969. 3. Scholl, M.L., in Conn, H.F.: Current Therapy 1973, Philadelphia, Saunders, 1973, pp. 675-7. 4. Metrick, S.: C.M.D. *37*:49 (Jan.) 1970. 5. Forster, F.M.: Med. Clin. North Am. *47*:1579 (Nov.) 1970. 6. White, P.T.: Wis. Med. J. *68*:178 (Apr.) 1969. 7. Millichap, J.G.: Drug Ther. *1*:15 (Oct.) 1971.



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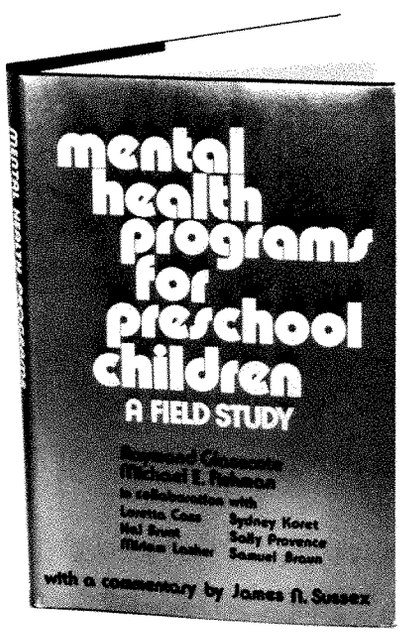
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ACTIONS—Pharmacologically, TRANXENE (clorazepate dipotassium) has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine.

INDICATIONS—TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE for long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS—TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS—TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS—In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS—The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION—

For symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg TRANXENE initially, followed by 30 to 60 mg in divided doses; 2nd 24 hours, 45 to 90 mg in divided doses; 3rd 24 hours, 22.5 to 45 mg in divided doses; 4th day, 15 to 30 mg in divided doses. Thereafter gradually reduce to 7.5 to 15 mg daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS—If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE (clorazepate dipotassium) is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE—Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed, since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY—Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption–Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED—TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

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Psychotherapies: An Overview
By Toksoz B. Karasu

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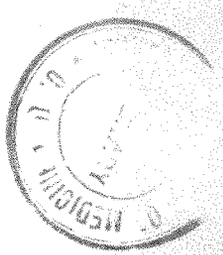
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'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.
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Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.
Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.
Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).
Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions; peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26
2. Schildkraut JJ: Tranylcypromine: effects on norepinephrine metabolism in rat brain. Am J Psychiatry 126:925–931, 1970
3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968, pp 1–12

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Actions. Navane is a psychotropic agent of the thioxanthene series. Navane possesses certain chemical and pharmacological similarities to the piperazine phenothiazines and differences from the aliphatic group of phenothiazines. Navane's mode of action has not been clearly established.

Indications. Navane is effective in the management of manifestations of psychotic disorders.

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy* – Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children–The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration – As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate–In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg. of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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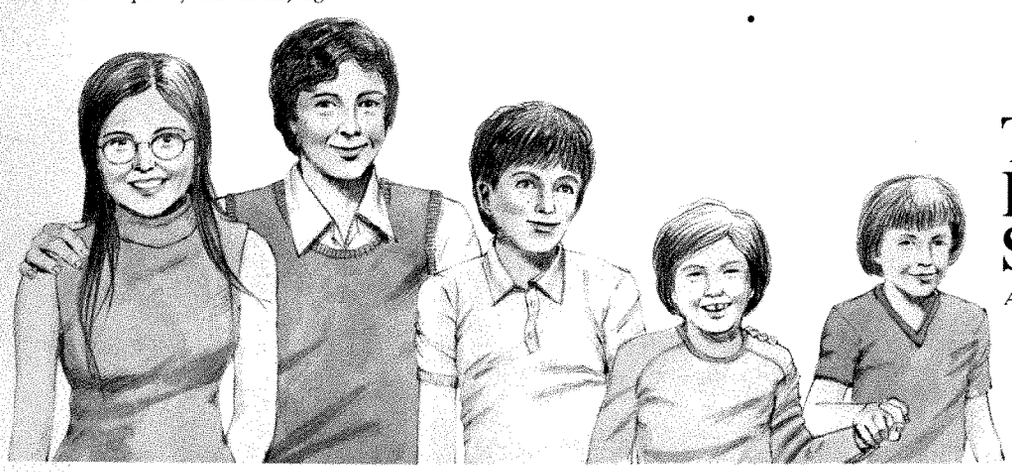
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Indications

Based on a review of this drug by the National Academy of Sciences – National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings:

Caution is recommended when 'Thorazine' is administered for the treatment of vomiting in children.

Antiemetics are not recommended to treat uncomplicated short-term vomiting in children or vomiting of unknown etiology.

The possibility of extrapyramidal reactions from 'Thorazine' may confuse the diagnosis of Reye's syndrome or other encephalopathy. Although unconfirmed, some suspicion exists that centrally-acting antiemetics may contribute to or adversely affect the course of Reye's syndrome, and should be avoided in suspected cases.

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Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

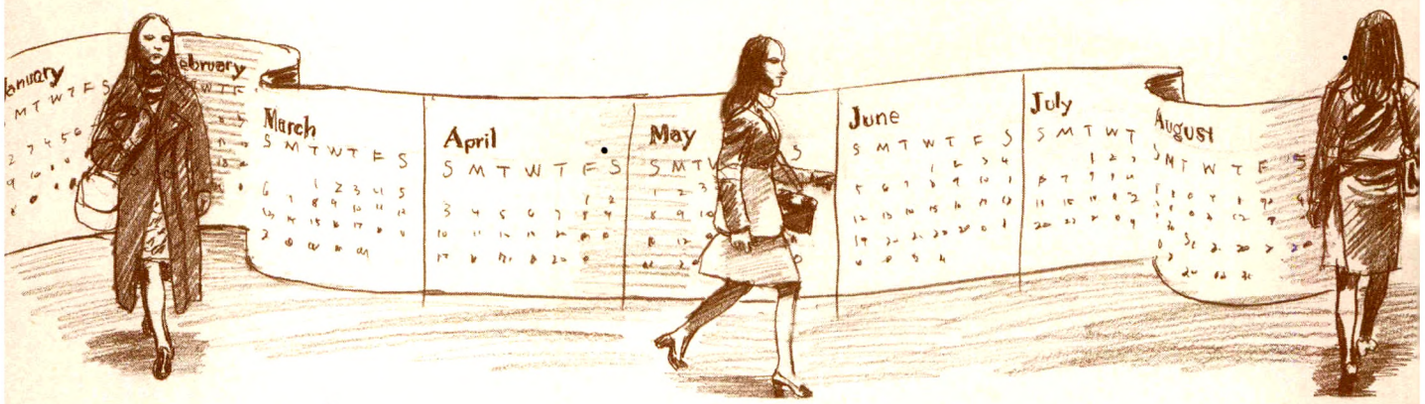
Supplied: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg., in bottles of 100; Single Unit Packages of 100 (intended for institutional use only). Spansule® capsules, 30 mg., 75 mg., 150 mg., 200 mg. and 300 mg., in bottles of 50; in Single Unit Packages of 100 (intended for institutional use only). Injection, 25 mg./ml.: Syrup, 10 mg./5 ml.: Suppositories, 25 mg. and 100 mg. Concentrate (intended for institutional use only), 30 mg./ml. and 100 mg./ml.

Smith Kline &French Laboratories
Philadelphia, Pa.

SK&F
a SmithKline company

Helps Return Patients to Reality

Why Endep® makes
with amitriptyline HC



Scored tablets h.s. enhance compliance and tolerability

Once you select the appropriate maintenance dose, you can prescribe a single tablet of Endep h.s. secure in the knowledge that you can adjust the dose by a half tablet without requiring the patient to obtain a new Rx. The simplicity of this h.s. schedule not only encourages compliance, but lessens the likelihood of anticholinergic and sedative side effects during daytime.

Scored tablets h.s. reduce confusion

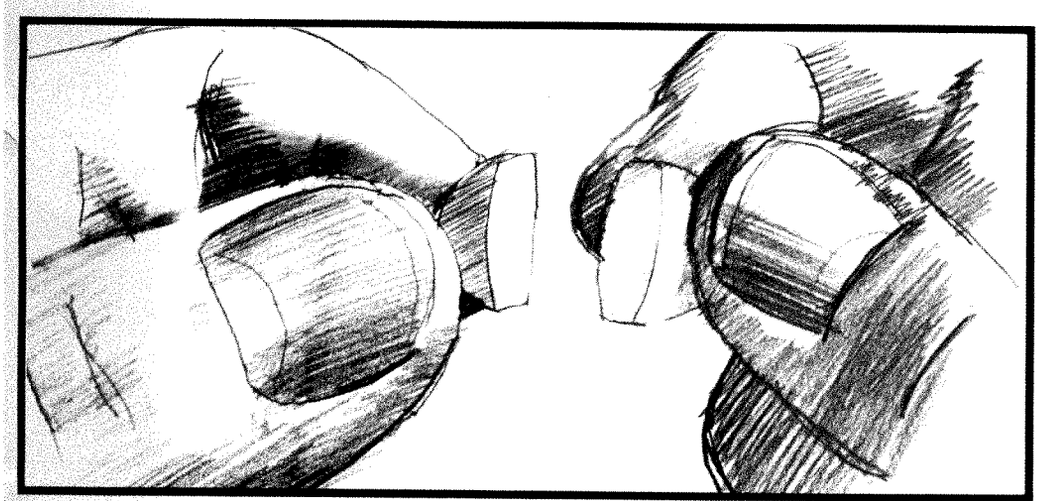
Bedtime dosage means that the patient has only one dose of Endep to remember, thus minimizing possible confusion with other medications taken concomitantly. The built-in flexibility of the scored tablet permits half-strength dosage adjustments without confusing the patient with a new color or size of medication.

ROCHE

long-term maintenance more practical:

Scored tablets h.s. promote economy

The economy of Endep (amitriptyline HCl) is particularly evident during long-term maintenance therapy, when your patient is purchasing refills of the single strength you have prescribed. The h.s. schedule enables you to prescribe a single strength in place of a more expensive multiple-dose prescription. The scored tablet permits you to adjust the regimen without putting the patient through the expense of a new prescription fee, without wasting tablets and without necessitating the purchase of more costly multiple-dose refills.



Endep®

amitriptyline HCl / Roche

The half-strength advantage

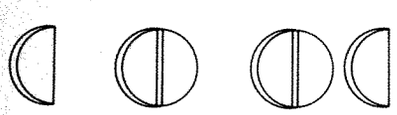
Before prescribing, please see following page for a summary of product information.

Practicality is a fundamental fact about Endep®

amitriptyline HCl/Roche

Every tablet of Endep (amitriptyline HCl) is scored for precise, convenient adjustment of dosage.

Half-tablet adjustment of dosage does not require a new Rx.



Single daily *h.s.* dosage promotes compliance and economy.

Compliance with practical regimen promotes optimal response.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

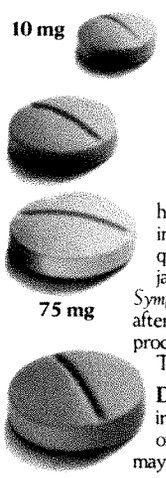
Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.



100 mg

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels.



10 mg

50 mg

25 mg

75 mg

150 mg

Other: Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels, increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier.

Initial Adult Dosage: Outpatients—25 mg *t.i.d.*; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate—50 to 100 mg *h.s.*, gradually increasing *h.s.* dose up to 150 mg/day. Hospitalized Patients—Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day.

Adolescent and Elderly Patients: In general, 10 mg *t.i.d.* with 20 mg *h.s.* may be satisfactory for those who do not tolerate higher doses.

Maintenance Dosage: With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg *b.i.d.* to *q.i.d.*, or 10 mg *q.i.d.* Continue maintenance therapy 3 months or longer to avoid relapse.

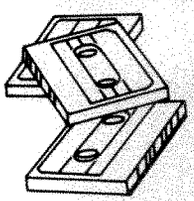
Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg, 75-mg, 100-mg and 150-mg scored tablets—bottles of 100 and 500, Tel-E-Dose® packages of 100; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg, 100 mg and 150 mg), available singly and in trays of 10.

ROCHE

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OF THE 130th ANNUAL MEETING
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*These materials meet the criteria of Category V continuing education activities of the APA Continuing Medical Education Requirements and the AMA Physician Recognition Award.

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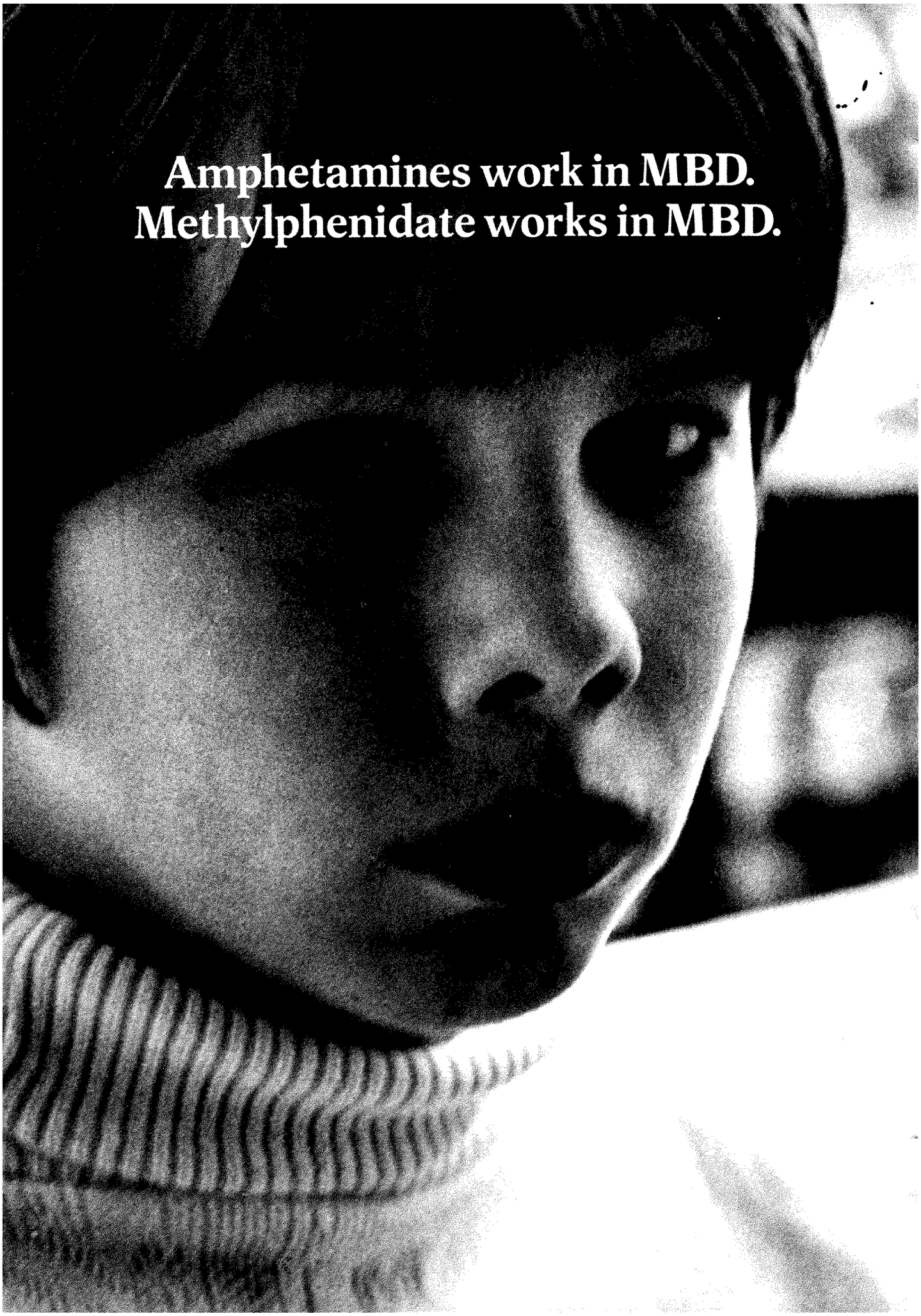
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Amphetamines work in MBD.
Methylphenidate works in MBD.

Then why Cylert® for MBD?

(pemoline)

Because Cylert offers a lot:

- Once-a-day dosage at home
- Elimination of mid-day school dose and need for school personnel supervision
- Avoids ups and downs of drug action brought about by multiple daily dosage
- Control of medication by the parent
- A chewable dosage form
- Less physician paper work (Cylert is in schedule IV)
- Safety and efficacy proven in extensive clinical studies*

*Copy of the Cylert Monograph available on written request.

Dosage and administration

Cylert is administered as a single oral dose each morning.

The recommended starting dose is 37.5 mg/day. This daily dose should be gradually increased by 18.75 mg at one week intervals until the desired clinical response is obtained. The effective daily dose for most patients will range from 56.25 to 75 mg. The maximum recommended daily dose of pemoline is 112.5 mg.

Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally (once or twice a year) to determine if behavioral symptoms are sufficient to require continued therapy.

When not to use medication

Cylert should not be used for (and will not be effective in) simple cases of overactivity in school age children.

Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

The physician should rely on a complete history of the child and a thorough description of symptoms from both parents and teacher before postulating a diagnosis of MBD.

ABBOTT

Please see next page for Prescribing Information.

708 3202

Cylert and Cylert Chewable Tablets

(pemoline)

Prescribing Information

Description: Cylert (pemoline) is a central nervous system stimulant. Pemoline is structurally dissimilar to the amphetamines and methylphenidate.

It is an oxazolidine compound and is chemically identified as 2-amino-5-phenyl-2-oxazolin-4-one.

Pemoline is a white, tasteless, odorless powder, relatively insoluble (less than 1 mg./ml.) in water, chloroform, ether, acetone, and benzene; its solubility in 95% ethyl alcohol is 2.2 mg./ml.

Cylert (pemoline) is supplied as tablets for oral administration.

Clinical Pharmacology: Cylert (pemoline) has a pharmacological activity similar to that of other known central nervous system stimulants; however, it has minimal sympathomimetic effects. Although studies indicate that pemoline may act in animals through dopaminergic mechanisms, the exact mechanism and site of action of the drug in man is not known.

The serum half-life of pemoline is approximately 12 hours. Peak serum levels of the drug occur within 2 to 4 hours after ingestion of a single dose. Multiple dose studies in adults at several dose levels indicate that steady state is reached in approximately 2 to 3 days.

Metabolites of pemoline include pemoline conjugate, pemoline dione, mandelic acid, and unidentified polar compounds. Cylert is excreted primarily by the kidneys; approximately 75% of an oral dose is recovered in the urine within 24 hours. Approximately 43% of pemoline is excreted unchanged.

Cylert (pemoline) has a gradual onset of action in children with minimal brain dysfunction. Using the recommended schedule of dosage titration, significant clinical benefit may not be evident until the third or fourth week of drug administration.

Indications: *MINIMAL BRAIN DYSFUNCTION IN CHILDREN*—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: The cause of minimal brain dysfunction (MBD) is unknown. Diagnosis of MBD involves the use of medical, psychological, educational, and social tools, since no single diagnostic test is adequate.

MBD is characterized by chronic moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindications: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See ADVERSE REACTIONS.)

Warnings: Cylert is not recommended for children less than 6 years of age since its safety and efficacy in this age group have not been established.

Sufficient data on the safety and the efficacy of the long-term use of Cylert in children with minimal brain dysfunction are not yet available.

A temporary suppression of the predicted growth rate (weight and/or height gain) has been reported for children receiving long-term stimulant therapy. A definite causal relationship between stimulant drugs and this finding has not been established.

Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans. Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates. However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline. Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) for use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laboratory animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on the fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannabilization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conception.

Adverse Reactions: Insomnia is the most frequently reported side effect of Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear to be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

Overdosage: Signs and symptoms of acute Cylert overdosage may include agitation, restlessness, hallucinations, tachycardia. The treatment for an acute overdosage of pemoline is essentially the same as that for an overdosage of any CNS stimulant. Management is primarily symptomatic and may include induction of emesis or gastric lavage, sedation, and other appropriate supportive measures.

Results of preliminary studies in dogs indicate that extracorporeal hemodialysis may be useful in the management of Cylert overdosage; forced diuresis and peritoneal dialysis appear to be of little value.

Dosage and Administration: Cylert (pemoline) is administered as a single oral dose each morning.

The recommended starting dose is 37.5 mg./day. This daily dose should be gradually increased by 18.75 mg. at one week intervals until the desired clinical response is obtained. The effective daily dose for most patients will range from 56.25 to 75 mg. The maximum recommended daily dose of pemoline is 112.5 mg.

Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally (once or twice a year) to determine if behavioral symptoms are sufficient to require continued therapy.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

18.75 mg. tablets (yellow-colored) in bottles of 100 (NDC 0074-6025-13)

37.5 mg. tablets (orange-colored) in bottles of 100 (NDC 0074-6057-13)

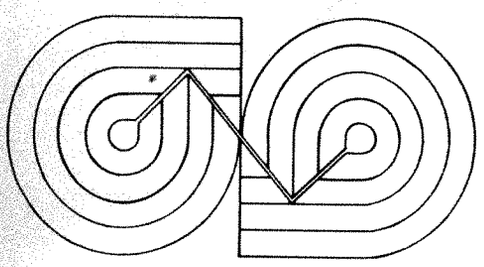
75 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-6073-13)

Cylert Chewable is supplied as monogrammed, grooved tablets in one dosage strength:

37.5 mg. tablets (orange-colored) in bottles of 100 (NDC 0074-6088-13)

ABBOTT

708 3202



PKSAP-III

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From the Sleep Research Laboratory:

Dalmane® (flurazepam HCl) proved the most effective hypnotic over 14 nights...[1]

For those patients who need it, Dalmane (flurazepam HCl) alone provides continued effectiveness[2,3]

Since insomnia is often transient and intermittent, the prolonged administration of a hypnotic is generally not necessary or recommended. But for those patients whose insomnia is a chronic problem, the continued effectiveness of Dalmane is a decided benefit. Even at the end of a 28-night medication period, patients with chronic insomnia were falling asleep faster, spending less time awake during the night, and sleeping substantially longer on Dalmane 30 mg *h.s.* than on baseline placebo nights (average results). This effectiveness was proved[2] and confirmed[3] in two independent sleep research studies using a new 47-night protocol. When similarly evaluated, pentobarbital was ineffective at the end of two weeks.[2]

TOTAL SLEEP TIME (MEAN)[3]
(5 patients with insomnia)

*Significantly increased over baseline placebo ($p < .05$)

14 consecutive nights:
3 in sleep laboratory, 8 at home,
3 in sleep laboratory



...and now, the only one proved effective over 28 nights.[2,3]

In previous studies,[1] chloral hydrate and glutethimide lost most or all effectiveness

In fact, continued effectiveness for both inducing and maintaining sleep over 14 consecutive nights[1] has long been an exclusive benefit of Dalmane. No other available sleep agent, including chloral hydrate or glutethimide,[1] has been able to demonstrate such effectiveness in the sleep research laboratory.

The safety record of Dalmane (flurazepam HCl) is well established

Dalmane is relatively safe and well tolerated. Prolonged administration is not generally necessary, but if used repeatedly, periodic blood counts and liver and kidney function tests should be performed.

Dalmane®
(flurazepam HCl)

relatively safe...
no need
to increase
dosage

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28 consecutive nights:
3 in sleep laboratory, 7 at home,
4 in sleep laboratory, 10 at home,
4 in sleep laboratory[3]

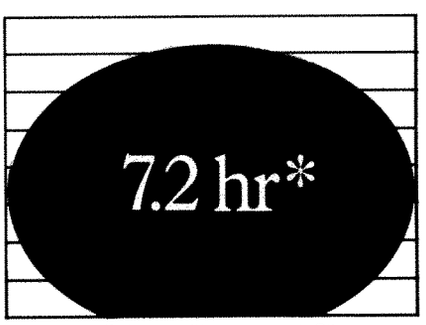
Please see following page for a summary of product information.

1,5,7,14,28 nights... as needed, the effectiveness of Dalmane® one *h.s.* (flurazepam HCl) C-IV meets the challenge of insomnia

One 30-mg capsule *h.s.*—usual adult dosage (15 mg may suffice in some patients).
One 15-mg capsule *h.s.*—initial dosage for elderly or debilitated patients.

- **Patients fall asleep rapidly, sleep longer on a single *h.s.* dose**
- **Nighttime awakenings and time spent awake are reduced**
- **Effectiveness maintained without dosage increase from night to night**
- **Patients generally awaken refreshed; morning "hangover" is infrequent**

TOTAL SLEEP TIME (OVERALL MEAN)[3]
(5 patients with insomnia)



Dalmane (flurazepam HCl)
nights 1-3, 12-14, 26-28

*Significantly increased over baseline placebo ($p < .05$).

REFERENCES:

1. Kales A, *et al*: *Arch Gen Psychiatry* *23*:226-232, Sep 1970
2. Kales A, *et al*: *Clin Pharmacol Ther* *18*:356-363, Sep 1975
3. Dement WC, *et al*: Long-term effectiveness of flurazepam 30 mg *h.s.* on chronic insomniacs. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, Jun 30-Jul 4, 1975

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

***Usage in Pregnancy:* Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane (flurazepam HCl), a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

1. PSYCHIATRIC EDUCATION: PROLOGUE TO THE 1980's, Library Bound, 544 pages, $15.00.

This is a substantive, highly readable, report of the Conference, written by Anne H. Rosenfeld with an Editorial Board comprising: Ewald W. Busse, M.D., Chairperson of the Conference, Walter E. Barton, M.D., Bernard Holland, M.D., Donna Norris, M.D., Melvin Sabshin, M.D., Jeanne Spurlock, M.D., and Robert L. Robinson, M.A. (Staff Consultant).

The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

2. THE WORKING PAPERS OF THE 1975 CONFERENCE ON EDUCATION OF PSYCHIATRISTS Paper bound, 432 pages, $12.50.

Sixty two leading psychiatric educators served on seven preparatory commissions, one for each of seven topic areas, and prepared advance working papers as a basis for Conference deliberations. The topics and Commission Chairpersons were: Social Issues and Needs (James N. Sussex, M.D.); Goals and Objectives (Morton F. Reiser, M.D.); The Resident (David R. Hawkins, M.D.); Content, Methodology, Faculty and Environment (Milton Greenblatt, M.D.); Research (Daniel X. Freedman, M.D.); Economics (Herzl Spiro, M.D.); Evaluation and Quality Control (Eric Pfeiffer, M.D.). The Chairpersons, together with the Conference Chairperson, served as the Editorial Board for this volume.

Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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in clinically significant depression...

penetrating the symptom barrier to productive psychotherapy

symptoms often interfere with therapy

In clinically significant depression, the patient often becomes barricaded behind his own symptoms during psychotherapy. Because these symptoms can be so sharply defined and somatic in expression, he may reject the fact that they are psychic in origin. Sleep disturbance, gastrointestinal complaints, anorexia, chronic fatigue, and other psychosomatic symptoms may so preoccupy—or overwhelm—the depressed patient that truly productive therapeutic sessions are difficult to achieve. Instead of being able to concentrate on the core emotional problems of the depression itself, he may for example go on at length about how broken his sleep is or keep reiterating (while the therapeutic hour slips by) how physically miserable he feels.

ELAVIL®
(AMITRIPTYLINE HCl, MSD)
to help penetrate the symptom barrier in depression requiring medication

ELAVIL, a highly effective tricyclic antidepressant, will often alleviate these symptoms. With the help of ELAVIL, sleep disturbance—so common in clinically depressed patients and often the symptom that distresses them most—as



well as other "barrier symptoms" may be lessened to the point where they no longer come between you and the patient.

psychotherapy takes direction

As these preoccupying complaints are lifted, psychotherapy sessions can become more productive. Patients are enabled to respond more fruitfully during therapeutic hours and to resume or pursue more efficiently the necessary activities in their personal lives.

meeting therapeutic goals

And as the antidepressant activity of ELAVIL (Amitriptyline HCl, MSD) takes hold, it helps relieve the depressive condition itself. Sometimes only a minimum of actual psychotherapy is required. The patient may experience a more rapid recovery—while you conserve valuable time.

Prescribe ELAVIL then, to help lighten the patient's burden in depression—and yours in its management.

ELAVIL should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks; or in children under 12. Patients with cardiovascular disorders should be watched closely. Safe use during pregnancy and lactation has not been established. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Hospitalize as soon as possible any patient suspected of having taken an overdose.

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, 100 mg, and 150 mg
INJECTION: 10 mg per ml

ELAVIL®
(AMITRIPTYLINE HCl | MSD)

an antidepressant with an anxiety-reducing sedative component to its action



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For a brief summary of prescribing information, please see following page.

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addendum

To further simplify once-daily therapy for many depressed patients

Tablets

ELAVIL®

(AMITRIPTYLINE HCl | MSD)

ONE TABLET, ONCE DAILY

—an appropriate way to prescribe for many depressed adult outpatients. Because of its simplicity, this regimen helps improve patient compliance. Of course, ELAVIL may also be prescribed in divided daily doses.

ONCE-DAILY DOSAGE SCHEDULE FOR ADULT OUTPATIENTS:

A single 75-mg tablet
Usual starting dosage



A single 100-mg tablet
Maximum starting dosage

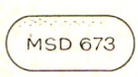


A single 50-mg tablet
Minimum starting dosage



Dosage may be increased by 25 or 50 mg as necessary until a total of 150 mg per day is reached.

A single 150-mg tablet
Maximum daily dosage



ELAVIL MAY ALSO BE PRESCRIBED IN DIVIDED DAILY DOSES

The 25-mg tablet



This strength may prove useful when therapy is initiated with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime dose.

The 10-mg tablet



This strength may prove useful for patients who require lower doses, e.g., adolescent and elderly patients. For these patients who can not tolerate higher doses, 10 mg three times a day with 20 mg at bedtime may be satisfactory.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.
When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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ELAVIL®
(AMITRIPTYLINE HCl, MSD)

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MERCK SHARP & DOHME



In the psychoneurotic patient, the pattern of anxiety – psychophysiological complaints and secondary depressive symptoms – often responds to Valium®(diazepam)

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Before prescribing, please consult complete product information, a summary of which appears on the following page.

In the psychoneurotic patient

Valium® (diazepam) IV

2-mg, 5-mg, 10-mg scored tablets

can relieve anxiety and its secondary depressive symptoms

Initial calming in hours

Your anxious psychoneurotic patient will find the prompt antianxiety action of Valium (diazepam) highly reassuring. It's immediate, tangible proof that his medication is working.

Significant improvement in days

As Valium reduces overwhelming symptoms of anxiety and psychic tension, your patient begins to feel better often within a few days.

Patient response you know, want and trust

You've come to expect a certain kind of response with Valium—and that's the kind you usually get. A pronounced antianxiety response. A response that starts to benefit the anxious patient almost immediately.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

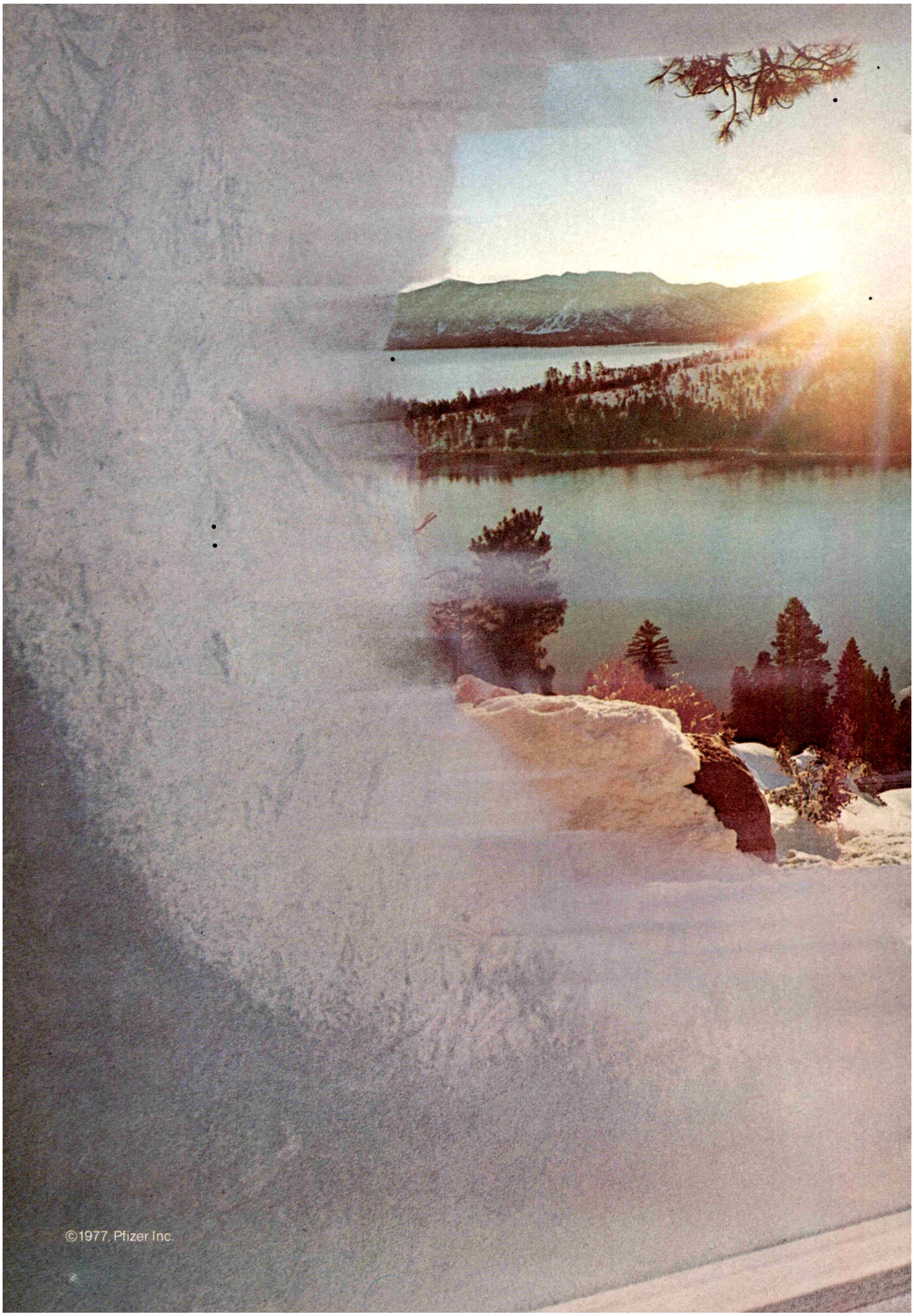
Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110


INTRODUCING
NEW
SINEQUAN® 75 mg
(DOXEPIN HCl) CAPSULE


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CLEARING OF DEPRESSION
ANTIDEPRESSANT EFFECTIVENESS...
with convenient once-a-day h.s. dosage*
SINEQUAN®
(DOXEPIN HCl)
NEW 75-mg CAPSULE
Also available:
100-mg, 50-mg, 25-mg, 10-mg CAPSULES and
ORAL CONCENTRATE 10 mg/ml, in 120-ml (4-oz) bottles
*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.
See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

IN THE TREATMENT OF CLINICAL DEPRESSION—

NEW SINEQUAN® 75 mg (DOXEPIN HCl) CAPSULE

EXTENDS THE RANGE OF DOSAGE STRENGTHS

PROVIDING ANTIDEPRESSANT EFFECTIVENESS

IN ADDITION TO OTHER, OFTEN BENEFICIAL, PROPERTIES...

CONVENIENT ONCE-A-DAY *h.s.* DOSAGE

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY GOOD TOLERATION

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have occasionally been reported. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

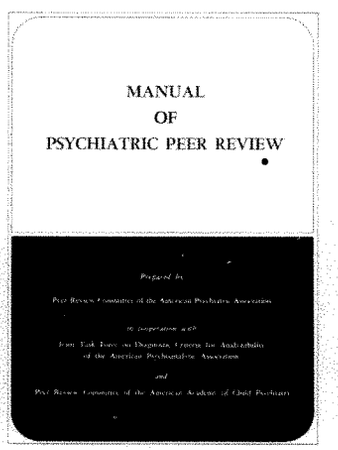
BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate
Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.
Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.
Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.
Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.
Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.
MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.
Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug. Patients should also be cautioned that their response to alcohol may be potentiated.
Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.
Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.
Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.
Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.
Central Nervous System Effects. Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.
Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.
Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.
Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.
Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)
Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.
Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.
Dosage and Administration. For most patients with illness of mild to moderate severity, a starting dose of 25 mg t.i.d. is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.
In more severely ill patients an initial dose of 50 mg t.i.d. may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.
In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.
As an alternative regimen, the total daily dosage, up to 150 mg, may be given on a once-a-day schedule without loss of effectiveness. This dose may be given at bedtime.
Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.
Overdosage.
A. Signs and Symptoms
1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.
Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.
B. Management and Treatment
1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.
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ADMISSION RECORD

Name	First	Middle	Sex	Age
R.*	Henry		Male	33

Marital status

S. M. W. D.

Occupation	Dates of previous In-Patient Admissio
Milkman	

Chart No. _____

Hospital _____

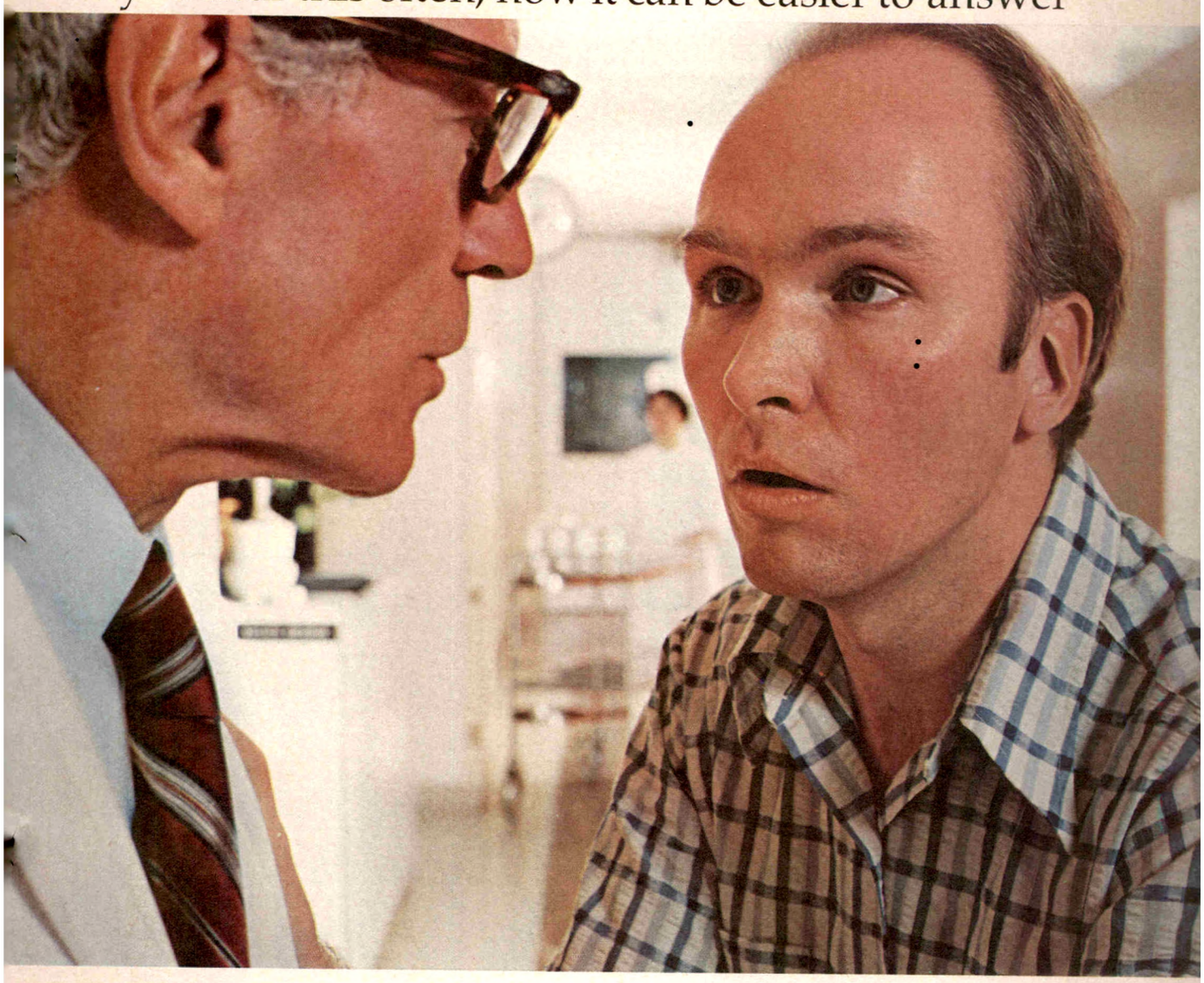
CASE SUMMARY SHEET

History:	First hospitalization four years ago following divorce. Dx: Acute schizophrenia. Treated with neuroleptics and ECT. On convalescent status, receiving medication through OPD for 18 months. Living with mother, working for past six months
Onset of current illness:	Became depressed when milk route was changed, but did not complain to employer. Developed sleeping problems, had delusions that customers had it "in for him" because of previous mental illness. Hallucinated that ex-wife living out-of-state visited him in his room complaining of money problems. Patient admitted to hospital asking for wife to leave him alone
Diagnosis:	Chronic schizophrenia, acute exacerbation
Treatment:	LOXITANE® C Loxapine Hydrochloride, 75 mg stat, then 50 mg b.i.d. increased over one week to 50 mg q.i.d.
Therapeutic course:	First week, less excited and hyperactive, fewer hallucinations, sleeping better. Second week, questioning reality of delusions and hallucinations, developed stiffness, rigid gait, and masked facies which responded to reduced dosage – 50 mg capsules t.i.d. – and appropriate antiparkinson medication
Follow-up:	4-1/2 weeks: No extrapyramidal symptoms noted. Taking part in OT and PT, relating better but affect still blunted. Planning with social worker for future weekend passes; mother cooperative. Antiparkinson medication discontinued. LOXITANE 50 mg b.i.d.

*Not an actual patient. The collection of clinical data upon which this simulation is built illustrates the action of LOXITANE® Loxapine Succinate as reported in clinical studies.

I Go Home?"

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For the symptoms that brought him there

See LOXITANE prescribing information on following page for indications, warnings and precautions and for more detailed information concerning side effects.

Loxitane®
LOXAPINE Lederle SUCCINATE

For the symptoms that brought him there

Recommended Daily Dosage

Initial Dosage	MILD 10 mg b.i.d.	MODERATE 10 mg t.i.d. or q.i.d.	SEVERE 25 mg b.i.d.
First 7 to 10 Days	Increase dosage until psychotic symptoms are controlled. Dosage should not exceed 250 mg/day. Usual dosage during titration: 50 to 150 mg/day		
Maintenance Dosage	Adjust to lowest effective level. Usual maintenance dosage: 60 to 100 mg/day Many patients are controlled with dosages as low as 20 to 60 mg/day		



Brief Summary

LOXITANE® Loxapine Succinate Capsules
OR
LOXITANE® C Loxapine Hydrochloride Oral Concentrate

Description: A dibenzoxazepine compound, representing a new subclass of tricyclic antipsychotic agent, chemically distinct from the thioxanthenes, butyrophenones, and phenothiazines.

Indications: For the manifestations of schizophrenia. Loxapine has not been evaluated for the management of behavioral complications in mental retardation, and therefore cannot be recommended.

Contraindications: In comatose or severe, drug-induced depressed states (alcohol, barbiturates, narcotics, etc.); and in individuals with known hypersensitivity to the drug.

Warnings: In Pregnancy: Safe use during pregnancy or lactation has not been established; in pregnancy, nursing mothers, or women of child-bearing potential, weigh potential benefits against possible hazards to mother and child. No embryotoxicity or teratology was observed in studies in rats, rabbits or dogs, although with the exception of one rabbit study, the highest dosage was only two times the maximum recommended human dose and in some studies they were below this dose. Perinatal studies have shown renal papillary abnormalities in offspring of rats treated from mid-pregnancy with doses of 0.6 and 1.8 mg./kg., doses which approximate the usual human dose but which are considerably below the maximum recommended human dose. **In Children:** Studies have not been performed in children; therefore this drug is not for use below the age of 16.

May impair mental and/or physical abilities, especially during the first few days of therapy; warn ambulatory patients about activities requiring alertness (*e.g.*, operating vehicles or machinery), and about concomitant use of alcohol and other CNS depressants.

Precautions: Use with extreme caution in patients with history of convulsive disorders; seizures have been reported in epileptics receiving this drug at antipsychotic dose levels, and may occur even with maintenance of routine anticonvulsant drug therapy. Has an antiemetic effect in animals; this effect in man may mask signs of overdosage of toxic drugs and obscure conditions such as intestinal obstruction and brain tumor. Use with caution in patients with cardiovascular disease. Increased pulse rates in the majority of patients receiving antipsychotic doses and transient hypotension have been reported. In hypotension requiring vasopressor therapy, preferred drugs may be norepinephrine or angiotensin. Usual doses of epinephrine may be ineffective because of inhibition of its vasopressor effect by loxapine. Possibility of ocular toxicity from loxapine cannot be excluded at this time, therefore observe carefully for pigmentary retinopathy and lenticular pigmentation (observed in some patients receiving certain other antipsychotic drugs for prolonged periods). Because of possible anticholinergic action, use with caution in patients with glaucoma or a tendency to urinary retention, particularly with concomitant administration of anticholinergic-type antiparkinson medication.

Adverse Reactions: CNS: Adverse effects other than extrapyramidal, infrequent. Drowsiness, usually mild, may occur at beginning of therapy or when dosage is increased, usually subsiding with continued therapy. Incidence of sedation is less than that of certain aliphatic phenothiazines and slightly more than piperazine phenothiazines. Dizziness, faintness, staggering gait, muscle twitching, weakness, and confusional states have been reported. Extrapyramidal reactions during use of this drug have been reported frequently, often during the first few days of treatment. In most patients, these involve Parkinson-like symptoms such as tremor, rigidity, excessive salivation, masked facies, akathisia, usually not severe and controlled by dosage reduction or use of antiparkinson drugs in usual dosage. *Less frequent, but more severe:* Dystonias, including spasms of muscles of neck and face, tongue protrusion, oculogyric movement; dyskinesia in the form of choreo-athetoid movements. These sometimes require reduction or temporary withdrawal of drug dosage in addition to appropriate counteractive drugs. *Persistent Tardive Dyskinesia:* May appear in some patients on long-term therapy, or after therapy has been discontinued; the risk greater in the elderly, especially females, on high dosage. These symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of tongue, face, mouth or jaw (*e.g.*, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements, sometimes accompanied by involuntary movements of extremities). No known effective treatment; discontinue all antipsychotic agents if these symptoms appear. The necessity to reinstitute treatment or increase dosage, or switch to a different antipsychotic agent, may mask syndrome. If medication is stopped when fine vermicular movements of the tongue occur, the syndrome may not develop. *Cardiovascular:* Tachycardia, hypotension, hypertension, lightheadedness, syncope. A few cases of EKG changes similar to those seen with phenothiazines have been reported, not known to be related to loxapine use. *Skin:* Dermatitis, edema (puffiness of face), pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of uncertain etiology seen in a few patients during hot summer months. *Anticholinergic:* Dry mouth, nasal congestion, constipation, blurred vision—more likely to occur with concomitant use of antiparkinson agents. *Other:* Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of uncertain etiology.

Dosage and Administration: Administered orally, usually in 2 to 4 divided doses a day. Adjust dosage to patient's need relative to severity of symptoms and history of response to antipsychotic drugs. Recommended initial dosage is 10 mg. b.i.d.; in severely disturbed patients up to 50 mg. daily. Increase dosage during first 7 to 10 days until psychotic symptoms are controlled. Usual therapeutic and maintenance range is 60 mg.-100 mg. daily. More than 250 mg. daily is not recommended. Maintenance dosage should be at lowest level to control symptoms; many patients have been maintained on 20 mg. to 60 mg. daily.

LOXITANE C Oral Concentrate should be mixed with orange and grapefruit juice shortly before administration. Use only enclosed calibrated dropper.

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In the hospitalized patient..
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ANTIPSYCHOTIC EFFICACY... INFREQUENT EXTRAPYRAMIDAL SYMPTOMS



Antipsychotic medication should not interfere with the patient's ability to participate in your total therapeutic program. That is why Mellaril (thioridazine) is an excellent choice. It is highly effective, and although extrapyramidal symptoms are characteristic of this class of drug, with Mellaril (thioridazine) extrapyramidal stimulation—notably pseudoparkinsonism—is infrequent. Adding an antiparkinsonian agent—which can cause its own side effects—can usually be avoided.

Mellaril (thioridazine) is not habituating and usually does not cause euphoria or undue sedation. (But, warn patients about undertaking activities requiring complete mental alertness.) And Mellaril (thioridazine) is contraindicated in patients with severe hypotensive or hypertensive heart disease.

MELLARIL® (THIORIDAZINE)

TABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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THE AMERICAN JOURNAL OF PSYCHIATRY

Psychotherapies: An Overview

BY TOKSOZ B. KARASU, M.D.

The author schematically describes the three predominating themes in the development of the psychotherapies: dynamic, behavioral, and experiential. These themes represent different conceptualizations of the fundamental nature of man and his ills, therapeutic processes or change agents, the basic nature of the physician-patient relationship, and the primary techniques and methods used. Although there appears to be some unifying thread connecting the four different areas within each theme, a comparable bond does not exist across the three themes. The author questions the potential unification of the psychotherapies and poses specific dimensions for further exploration.

THE EMERGENCE of numerous competing systems of psychotherapy during the last few decades has been cited as one of the principal problem areas in the field (1). In 1975 Parloff polled more than 140 presumable forms of currently practiced psychotherapy (2). Although London described this confusing proliferation as a reflection of changing times in answer to pervasive repression, anxiety, and/or boredom of man (3), the specific ways in which each new modality may or may not differ from its predecessors are far from clear. For the most part, dogmatic partisan claims of the superiority of each successive system continue to persist in spite of increasing evidence that such separatist claims may be largely unfounded (4, 5).

In attempts to comprehend the therapeutic influence in the total range of psychological treatments that have made their appearance at various points in the course of time, many authors have focused on the nonspecific or common elements that all psychotherapies are presumed to share (4, 6–19). The following features have been repeatedly cited as basic to all psychotherapies: an emotionally charged, confiding relationship; a therapeutic rationale (myth) that is accepted by patient and therapist; the provision of new information, which may be transmitted by precept, example, and/or self-discovery; the strengthening of the patient's expectation of help; the provision of success experiences; and the facilitation of the arousal of one's emotions (9).

Alternatively, Wittkower and Warnes pointed out that "while such universal features undoubtedly exist, it seems absurd to minimize differences in psychotherapy" (19). The originators and strong proponents of individual systems of psychotherapy who felt the need to differentiate and/or disassociate themselves from their predecessors and peers to justify their efforts might share this view (20–23). In addition, comparative conceptual studies of various forms of psychotherapy typically cite striking contrasts among various therapies (24–33). More recently, experimental studies of different schools have lent some scientific support to the separatist stance. Exemplary of such findings are the systematic studies of analytically oriented psychotherapy versus behavior therapy supporting the view that these are, in total, highly contrasting styles of treatment (34, 35). Moreover, the treatment procedures created, developed, and chosen in one society or within the context of a particular belief system may not be transposable to another. This is especially evident in attempts at cross-cultural psychotherapy (19, 36).

The current state of the art attests to the lack of clarity and lack of resolution of the specificity versus common elements controversy in explaining what is the quintessence of the therapeutic cure. This conflicting state of affairs is further compounded by those comparative studies of various psychotherapies which suggest that one's espoused theoretical orientation regarding the nature of the healing process may not always be synchronous with one's actual practices (37–39). For example, a comparison study of Freudian,

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TABLE 1
Examples of Three Therapeutic Themes in the Psychotherapies and Their Variations*

Dynamic		Behavioral		Experiential	
Theme or Variation	Representative	Theme or Variation	Representative	Theme or Variation	Representative
Classical psychoanalysis	Freud	Reciprocal inhibition therapy	Wolpe	Existential analysis	Binswanger
Analytical psychology	Jung	Implosive therapy	Stampfl	Daseinanalysis	Boss
Will therapy	Rank	Conditioned reflex therapy	Salter	Logotherapy	Frankl
Active analytical therapy	Stekel	Learning theory therapy	Dollard	Client-centered therapy	Rogers
Individual psychology	Adler	Social learning psychotherapy	Rotter	Gestalt therapy	Perls
Interpersonal psychiatry	Sullivan	Modeling therapy	Bandura	Psycho-imagination therapy	Shorr
Intensive psychotherapy	Fromm-Reichmann	Directive psychotherapy	Thorne	Experiential therapy	Whitaker
Character analysis	Horney	Direct decision therapy	Greenwald	Experiential therapy	Gendlin
Cultural school	Fromm	Confrontation problem solving	Garner	Primal scream therapy	Janov
Ego analysis	Klein	Assertion-structured therapy	Phillips	Bioenergetic analysis	Lowen
Chicago school	Alexander, French	Personal construct therapy	Kelly	Structural analysis	Rolf
Sector therapy	Deutsch	Rational therapy	Ellis	Autogenic training	Luthe
Objective psychotherapy	Karpman	Integrity therapy	Drakeford	Transcendental Meditation	
Short-term psychotherapy	Sifneos, Malan, Bellak	Reality therapy	Glasser	Nirvana therapy	
Direct analysis	Rosen	Philosophical psychotherapy	Sahakian	Zen psychotherapy	Watts
Psychobiological therapy	Meyer	Biofeedback training	Green	Psychedelic therapy	Osmond
Biodynamic therapy	Masserman				
Adaptational psychodynamics	Rado				
Hypnoanalysis	Wolberg				
Character analysis, vegetotherapy	Reich				

*Although some of the more recent psychotherapies may combine individual and group techniques, this organization of psychotherapeutic systems focuses on modalities that are essentially dyadic in nature. It therefore does not include family, group, or milieu therapies per se, nor such adjunct therapies as art, music, and dance.

Kleinian, Jungian, and Gestaltist therapists found that descriptive ratings of the different approaches in action did not differentiate the respective schools of thought as well as the investigators (and, no doubt, the proponents themselves) would have expected (39).

As treatments useful in one area of human disturbance are found to be less valuable in another (5, 40), efforts are made to arrive at better criteria for selecting patients for particular forms of therapy and for modifying existing forms. Here the stated task for the future is to achieve greater specificity concerning the effects of particular kinds of interventions (13). Thus the tantalizing need for increased examination and clarification of the therapeutic process continues.

Doubtless there are many ways to slice the therapeutic pie. In early attempts to schematize the many psychotherapeutic methods and techniques throughout history, Menninger (41) and Bromberg (42) ultimately subsumed the various forms under two dichotomous heads: those which they thought used a principle of suppression in their treatment approach versus those which represented the use of a principle of expression.

Beginning where Menninger and Bromberg left off, Harper's descriptive overview of 36 established Freudian and post-Freudian psychotherapeutic schools, or systems of psychotherapy, attempted to divide the various approaches into two overriding categories: those which reflected emotionally oriented or affective forms of treatment versus those which Harper considered essentially intellectually oriented or cognitive (28). On a more philosophical plane, Rychlak addressed the possible implications of ideology for methodology and assessed psychotherapies on the basis of whether they represented therapeutic models that were essentially Lockean (mechanistic) or Kantian (humanistic) (43). Rychlak also conceptualized psychotherapies according to a comparison of their respective motives: those with a scholarly motive, which are primarily concerned with unraveling the depths of

TABLE 2
Summary of Thematic Dimensions of Three Kinds of Psychotherapy

Theme	Dynamic	Behavioral	Experiential
Prime concern	Sexual repression	Anxiety	Alienation
Concept of pathology	Instinctual conflicts: early libidinal drives and wishes that remain out of awareness, i.e., unconscious	Learned habits: excess or deficit behaviors that have been environmentally reinforced	Existential despair: human loss of possibilities, fragmentation of self, lack of congruence with one's experiences
Concept of health	Resolution of underlying conflicts: victory of ego over id, i.e., ego strength	Symptom removal: absence of specific symptom and/or reduction of anxiety	Actualization of potential: self-growth, authenticity, and spontaneity
Mode of change	Depth insight: understanding of the early past, i.e., intellectual-emotional knowledge	Direct learning: behaving in the current present, i.e., action or performance	Immediate experiencing: sensing or feeling in the immediate moment, i.e., spontaneous expression of experience
Time approach and focus	Historical: subjective past	Nonhistorical: objective present	Ahistorical: phenomenological moment
Type of treatment	Long-term and intense	Short-term and not intense	Short-term and intense
Therapist's task	To comprehend unconscious mental content and its historical and hidden meanings	To program, reward, inhibit, or shape specific behavioral responses to anxiety-producing stimuli	To interact in a mutually accepting atmosphere for arousal of self-expression (from somatic to spiritual)
Primary tools techniques	Interpretation: free association, analysis of transference, resistance, slips, and dreams	Conditioning: systematic desensitization, positive and negative reinforcements, shaping	Encounter: shared dialogue, experiments or games, dramatization or playing out of feelings
Treatment model	Medical: doctor-patient or parent-infant (authoritarian), i.e., therapeutic alliance	Educational: teacher-student or parent-child (authoritarian), i.e., learning alliance	Existential: human peer-human peer or adult-adult (egalitarian), i.e., human alliance
Nature of relationship to cure	Transferential and primary for cure: unreal relationship	Real but secondary for cure: no relationship	Real and primary for cure: real relationship
Therapist's role and stance	Interpreter-reflector: indirect, dispassionate, or frustrating	Shaper-adviser: direct, problem-solving, or practical	Interactor-acceptor: mutually permissive or gratifying

man's nature; those with an ethical motive, which concern themselves primarily with man's self and his values; and those with a curative motive, which directly aim at scientifically derived cure (44). Offenkrantz and Tobin's configuration is more implicit. They suggested that all learning (including psychotherapeutic learning) occurs in three modes: by identification, by conditioning, and by insight (45). Thus one might assume to unite the various psychotherapies on the basis of their primary modes of therapeutic learning or the major ways in which they presume to effect change in or cure of the patient.

With the preceding topographies in mind, I shall organize the morass of systems practiced today according to a troika of basic themes around which each school may be said to broadly, but distinctively, pivot. These, representing a composite of dimensions, are referred to as dynamic, behavioral, and experiential (see table 1). Although these rubrics per se are not completely new (2, 46), to my knowledge no study has attempted to portray schematically their respective features in relation to the increasing array of modalities being practiced today. A number of contrasting dimensions constitute or reside behind these three themes (see table 2). Each theme represents something of a unity, that is, one's conceptual framework or belief system regarding the nature of man and his ills will have a bearing on one's concept of therapeutic modes or curative processes and the nature of the therapeutic relationship between patient and therapist and, ultimately, on one's methods or techniques of treatment.

THE DYNAMIC THEME

The Nature of Man and His Ills

The dynamic point of view, as originally conceived in the context of physics, subscribes to the idea that all mental phenomena are the result of an interaction of forces (47). It pertains to an appreciation of the complexity of man as reactor to or victim of turbulent intrapsychic forces from which he continually struggles to be set free. Freud described the major force of this struggle in the origin of the neuroses of man according

to the theory of instinctual conflict: "From the very first we have said that human beings fall ill of a conflict between the claims of instinctual life and the resistance which arises within them against it" (48, p. 57). More specifically, Freud considered the etiology of the neuroses of man to be decidedly sexual in nature: "No neurosis is possible with a normal *vita sexualis*" (49, p. 276). In the orthodox dynamic tradition man is portrayed as fraught with inner and unknown urgings and contradictions, subject to and resisting against a reservoir of impulses largely inaccessible to his conscious self.

The psychoanalytic perspective typically incorporates five fundamental principles, i.e., dynamic, economic, structural, developmental, and adaptive (50). Taken together, these principles offer an analytic legacy that includes the following allegiances: 1) a primary concern with the vicissitudes of man's instinctual impulses, their expression and transformation, and, more crucially, their repression, by which is meant the pervasive avoidance of painful feelings or experiences by keeping unpleasant thoughts, wishes, and affect from awareness; 2) the belief that such repression is of an essentially sexual nature and that the roots of disturbance reside in faulty libidinal or psychosexual development; 3) the idea that faulty psychosexual development has its origins in early past and childhood conflicts or traumata, especially those concerning a parental oedipal configuration as manifested in the classic desire for one's opposite-sexed parent; 4) the belief in the resilience, persistance, and inaccessibility of oedipal yearnings (i.e., these underlying conflicts remain alive and ever active but out of awareness or unconscious; man's consciousness describes the exceptional rather than the standard state of affairs); 5) the idea that we are dealing essentially with the psychic struggle and torments of biological man's innate impulses or instincts (id), their derivatives, and the primarily defensive mediation with external reality (ego) in light of one's moral precepts or standards (superego); and, finally, 6) an adherence to a concept of psychic determinism or causality according to which mental phenomena as well as behaviors are decidedly not chance occurrences but meaningfully related to events that preceded them and, unless made conscious, unwittingly subject to repetition.

Therapeutic Change or Curative Processes

In accordance with these tenets, for the dynamic therapist the ultimate task in its most parsimonious and famous form is to make conscious the unconscious. This means that it is the ongoing therapeutic charge of the therapist who subscribes to the dynamic view to facilitate the emergence and comprehension of unconscious and largely libidinal content. That is, the dynamic therapist seeks to undo the essentially sexual repression of the patient and to overcome the patient's natural resistances to this endeavor. The dynamic therapist attempts to accomplish this by means of a slow and scrupulous unraveling of the largely historical meanings of mental events and the devious ways in which they may serve to ward off the underlying conflicts through defensive camouflage. Understandably, the dynamic goal is thereby a long-range one, perhaps even interminable. At best this concept of cure means opting for total personality reorganization in the final resolution of neurotic conflicts. The most crucial manifestation of this is the resolution of the oedipal conflict, which is traditionally regarded as requisite for a healthy personality. This ultimate integration of personality would translate itself into final mastery of ego over id impulses or, as classically stated, where id was, there ego shall be.

Greenson defined the therapeutic process as "an interrelated series of psychic events within the patient, a continuity of psychic forces which have a remedial aim or effect" (51, p.7). The dynamic psychotherapeutic systems have consecutively considered as their hallmarks of change the processes of catharsis (following abreaction) and insight. Harper broadly defined insight as "the process by which the meaning, significance, pattern, or use of an experience becomes clear—or the understanding which results from this process" (28, p.163). He defined catharsis as "the release of tension and *anxiety* by recounting and/or *acting out* past experiences" (28, p.158; author's italics).

Although both processes have been considered in the psychodynamic tradition, it may be noted that Freud never used the term "insight" per se. The therapeutic process transferred its emphasis from the primary importance of abreaction (catharsis) to the removal of amnesia and the recovery of repressed memories. Following this shift, Greenson pointed out, "catharsis [is] no longer the ultimate aim of therapy" (51, p.13). In its stead, the process of insight has been extensively singled out not only to refer to a phenomenon specially applicable to the psychodynamic therapies in contrast to other therapies (51–53) but also as the patient's ultimate aspiration.

According to Hutchinson, there are four successive stages in attaining therapeutic insight: 1) a stage of preparation, which is characterized by frustration, anxiety, a feeling of ineptness, and despair and may be followed by much trial-and-error activity relevant to the solution of a certain problem and the falling into habitual patterns or ways of thinking, foreseeing no apparent solution to the problem; 2) a stage of incubation or renunciation in which one desires to hide or escape from the problem and one is resistant or unmotivated in therapeutic or insightful efforts; 3) a stage of inspiration or illumination in which the whole problem becomes illuminated and a solution or solutions suggest themselves (often there is a flood of vivid ideas and a sense of finality accompanied by a conviction in the truth of the insight); and 4) a stage of elaboration and evaluation in which the validity of the insight is checked against external reality (54).

Although the third stage tends to be most frequently identified with the idea of insight (suggesting an essentially eureka phenomenon), Ludwig pointed out that

> during the typical course of psychotherapy, it is much more common for the patient to experience insight in a . . . drawn out and emotionally attenuated from. The sudden tidal wave of illumination or enlightenment is rare compared to the numerous small ripples of insights which are experienced and intellectually assimilated over a long period of time. Moreover, the therapeutic insights tend to be circumscribed and specific to certain problem areas than the profound and general eureka experiences, such as those described to occur during religious conversion or revelation in which the "whole truth" suddenly is revealed. (55, p. 315)

In terms of the therapeutic value of the process of insight, Ludwig noted that "there is no necessary relationship between the truth or falseness of insight and therapeutic results" (55, p. 313). In addition, since intellectual insight alone is felt to be of minimal value, attempts have been made to distinguish between intellectual and emotional insight. However, it is difficult to validate such a distinction (56).

In brief, Ludwig hypothesized that insight is therapeutic when it meets all of the following specifications: 1) consistency, that is, the deductions based on the original insight are stable and logically sound regardless of the truth or falsity of the particular content of the insight; 2) continuity, that is, insights must take place within some existing theoretical framework or stream of tradition in which the insight can be tested; 3) personal consequences, that is, the insight must be judged by the fruit it bears in terms of the ultimate use to which the insight is put; and 4) social consequences, that is, the acquisition of insight should allow the person to interact with others in a more honest and meaningful manner (55).

Although analysts have confidence in the role of insight as a therapeutic agent, doubt has been cast on the "insight leads to change" dictum. Schonbar, for example, has observed that "not all change is attributable to insight" and "not all insight leads to change" (57). However, the fact that insight, even as an ultimate change agent, does not occur in isolation is more important for consideration of the analytic therapies. Intricately embedded in the psychodynamic curative process is the critical role of the therapeutic relationship, expressly manifested in the phenomenon of the transference relationship.

The Nature of the Therapeutic Relationship

In 1970 Strupp noted, "There can be no doubt that the patient's relationship to the therapist . . . embodies one of the most powerful forces in the therapeutic enterprise. . . . Psychotherapeutic changes always occur in the context of an interpersonal relationship, and are to some extent inextricable from it" (13, pp. 396, 400). The specific therapeutic agent, according to Rosen, seems to be the "complex, emotionally-charged parent-child kind of relationship between the psychiatrist and the individual whom he is treating" (58, p.126). In terms of the special nature of that therapeutic relationship, it should be pointed out that deliberate systematic attention to the vicissitudes of the special relationship between therapist and patient is crucial to the conduct of the psychoanalytic psychotherapies. It constitutes both the subject and the object of analysis.

Historically, two roles or stances for the therapist have been described in portraying the psychodynamic psychotherapies: the primary stance with regard to the making of the transference relationship and, more recently, the secondary stance with regard to the making of a working or therapeutic alliance. Despite increasing acceptance of the latter into the therapeutic situation, these represent dual postures, which Greenson explicitly depicted as antithetical to each other, both in their essential purposes and in the actual requirements they make of the therapist (51).

The primary stance reflects Freud's original recommendations 1) that the analyst be like a mirror to the patient, reflecting only what is reflected to him/her by the patient and not bringing his/her own feelings (attitudes, values, personal life) into play (59) and 2) that the analyst follow a posture of privation or rule of abstinence, that is, technical motives must unite with ethical ones in preventing the therapist from offering the patient the "love" that the patient will necessarily come to crave (59, pp. 157–171).

These dictums have been taken to mean that two basic requirements are traditionally made of the analyst if he/she is to best accomplish the therapeutic task: 1) he/she must continue to judiciously frustrate and avoid gratifying the wishes of the patient; and 2) he/she must remain relatively removed and anonymous, a deliberately dispassionate observer and reflector of the patient's feelings. The therapeutic relationship is asymmetrical. Henry and associates described the relationship as follows:

> Only the patient is supposed to reveal the intimate details of his life. The psychotherapist is not only free to determine what he will reveal and conceal about himself, but also to choose how to react to what the patient is saying, if indeed he decides to respond at all. The relationship is also asymmetrical in that only the therapist is supposed to interpret and impute meaning to what the patient is saying and only the therapist can evaluate the degree to which therapeutic objectives are being achieved in the relationship. (60, p. 218)

Conversely, within the same framework, the more recent concept of a working or therapeutic alliance reflects an alternatively nonregressive, rational relationship between patient and therapist. Although still in the service of analyzing transference and resistances, according to Chessick it means "that the therapist aims at forming a real and mature alliance with the conscious adult ego of the patient and encourages him to be a scientific partner in the exploration of his difficulties" (53). The real object need of the patient, deliberately frustrated by the transference relationship, is satisfied by the therapeutic alliance.

Techniques and Methods

Bellis' statement that "no technique is therapeutic in itself. . . . Every clinician must understand and calibrate his own instruments" (61) bears repeating in a discussion of the techniques of dynamic psychotherapy.

The major instruments of the prototypic dynamic approach are primarily verbal in nature. They rest in part on the proverbial talking cure and may be regarded as free association on the part of the patient and analysis of transference reactions and resistances on the part of the therapist. Analysis, the task of the therapist, is facilitated by four specific procedures: confrontation, clarification, interpretation, and working-through. It is the third of these, interpretation, that, according to Greenson, is regarded as the "ultimate and decisive instrument" (51).

With regard to the input of the patient, the technique of free association early reflected the major vehicle for the communication of uncensored content from patient to therapist. It has constituted the primary procedure for eliciting the raw material on which the analysis ultimately rests. This includes the eliciting of dreams, which Freud regarded as "the royal road to a knowledge of the unconscious activities of the mind" (62, p. 608). Methodologically, the attempt to solicit free associations and dreams from the patient accounts for the most notorious material ingredient of the analytic method in classical analysis—the couch. Having the patient in a supine position out of direct view of the therapist and without extrinsic environmental intrusions is meant to create conditions of relative sensory deprivation that in turn serve to maximize the evocation of repressed memories.

With regard to the therapist's task and response to the verbal material elicited from the patient, the crux of the psychoanalytic method remains the analysis of transference, which comprises the major instrument of analysis as well as its major obstacle. The deliberate elucidation of transference reactions results in inevitable resistances to this endeavor, which must also be overcome as part of the treatment. Methodologically, the reflective, ambiguous stance of the therapist in concert with the high frequency and regularity of contacts between patient and therapist are meant to encourage the regressive transference process and the intensity of feelings in the treatment situation.

Despite the endowment of the value of interpretation, the therapeutic path must be carefully paved in order for it to have its most beneficial effects. In this regard, the role of the techniques of confrontation, clarification, and working-through have been noted. Confrontation refers to having the patient discern or face the particular mental event to be investigated: clarification refers to placing the same event in sharp focus, separating important aspects from insignificant ones (both of these processes prepare for the actual interpretation); interpretation then goes beyond the manifest material by assigning an underlying meaning or cause to the event or phenomenon in question; finally, working-through refers to the repetitive, progressive, and elaborate explorations of the interpretations and resistances to them until the presented material has become fully integrated into the patient's understanding. This is perhaps the most time-consuming aspect of dynamic psychotherapy. Although the major thrust of treatment occurs within the therapist's office, working-through necessarily includes the tacit work done by the patient outside of the therapeutic hour.

In terms of comparisons with other forms of treatment and their reputed curing powers, certain techniques or procedures are considered to be expressly anti-analytic, i.e., to block or lessen one's understanding or insight rather than facilitating it. Foremost in this regard are (perhaps ironically) abreaction, which may still be used but is not felt to directly bring insight; direct suggestion or advice, which is only useful to the extent that it is openly acknowledged and analyzed within the therapy setting; manipulation, allowable only to the extent that it can be brought into the analytic arena and does not occur without the ultimate knowledge of the patient; and the deliberate or conscious assumption of roles or attitudes that create an unanalyzable situation by their very nature.

Variations on the Dynamic Theme

The prototypic embodiment of the psychodynamic theme is, of course, classical psychoanalysis. The variations on the dynamic theme reflect overt and covert modifications of theoretical conceptualizations as well as methodological and technical applications in practice. These include attempts to partially or completely transcend the biological focus of Freud with more interpersonal, social, ethical, and cultural considerations (e.g., Adler, Horney, Sullivan, Fromm, Fromm-Reichmann, Meyer, and Masserman); to extend or enhance the ego with earlier or more adaptive endowments (e.g., Federn and Klein); to enlarge man's temporality with a time focus on his primordial past (e.g., Jung), his present, and/or his future (e.g., Adler, Stekel, Rank, and Rado); to expand treatment procedures by altering the range and goals of treatment (e.g., Rank, Alexander, Deutsch, and Karpman); to develop guidelines for short-term psychotherapy with anxiety-provoking techniques (e.g., Sifneos) and even brief treatment of serious illness within the context of a single interview (e.g., Malan) (63–67); to revise the role of the therapist's personality and relationship to the patient by making the therapist a more direct, flexible, and/or active participant (e.g., Adler, Sullivan, Rank, Alexander, Stekel, Ferenczi, and Rosen); and, at perhaps the opposing end of the analytic spectrum, to restore the psychophysical balance of man by focusing equally on the physical half of the psychophysical split (e.g., Rado and Masserman) and/or substituting an approach to therapeutic cure from the somatic side by trading the traditional change mode of insight for a reversal back to the earlier ca-

tharsis by means of the bodily release of conflictual tensions (e.g., Reich).

THE BEHAVIORAL THEME

The Nature of Man and His Ills

The behavioral theme presumes that all behavior, both normal and abnormal, is a product of what man has learned or not learned. Neuroses or neurotic symptoms are construed as simple learned habits, involuntarily acquired, repeated, reinforced responses to specific stimuli in the environment. Indeed, Eysenck said, "there is no neurosis underlying the symptom, but merely the symptom itself" (68). Conversely, since external behaviors constitute the essence of the therapeutic problem, the therapeutic cure is simple: "Get rid of the symptom and you have eliminated neurosis" (68).

Behavioral psychotherapy is an outgrowth of animal laboratory experiments on classically conditioned responses in which animals were observed to have habits that were like human phobias. Traditionally, behavioral psychotherapy also presumes that human neuroses have the same basic vicissitudes as those of the animal, in which anxiety (equated with fear) is regarded as its central manifestation (69).

Thus behavioral man is reducible to stimulus-response connections that can be isolated and altered in a piecemeal manner by reinserting new associations. Viewed in this way, behavioral man is infinitely manipulable and therefore controllable by external events in the environment.

The prototype of orthodox behavior therapy is exemplified in Wolpe's reciprocal inhibition therapy, which is based on a classical conditioning paradigm.[1] Wolpe's main thesis is that neurotic symptoms are all essentially phobias based on the adverse learning of unrealistic fears. In behavioral theory this means that anxiety has been conditioned during highly disturbing or traumatic experiences. Wolpe would regard a complex symptom like neurotic passivity as a learned fear of rejection or disapproval reflecting one form of a phobic or anxious reaction to others.

Therapeutic or Change Processes

For the behaviorist all problems are construed as pedagogical in nature and therefore alterable only through direct teaching and learning of new behavioral associations, i.e., stimulus-response connections. The patient must be taught new alternatives that must be repeated and practiced within as well as outside of the therapy situation. These alternative modes of functioning do not occur simply as a concomitant of cognitive or emotional understanding of one's problems; the patient must rehearse the new alternatives directly. Thus, in direct contrast to the psychodynamic schools, the behavioral approaches, according to Cautela, tend to sustain the view that insight is not only unnecessary but usually hinders the treatment for deviant behavior (70). Wolpe's principle of reciprocal inhibition implies the rejection of catharsis as well. Wolpe sees abreaction (the symbolic re-evocation of a fearful past experience) as a special case in point, asserting that no permanent effects are achieved if unrelieved terror is the only emotional factor involved and is not counterposed by relaxation responses.

One implication of this view of the mode of therapeutic change is that change can presumably occur within a short period of time. In contrast to the dynamic theme, Eysenck stated that "all treatment of neurotic disorders is concerned with habits existing in the present; their historical development is largely irrelevant" (68). Moreover, Cautela stated, "In fact, it is possible to have a situation in which symptoms have been removed with no knowledge at all of the etiology" (70).

Although all behaviorists may be viewed as seeking change through direct conditioning, shaping, or training, Wolpe, in accordance with his classical conditioning paradigm, sees all therapeutic learning or change (not just behavior therapy) as occurring within the reciprocal inhibition framework per se. All therapies necessarily incorporate the substitution of relaxation for anxiety in the reduction or elimination of symptoms. However, more critically, the difference between behavior therapy and other therapeutic situations is that in the latter, counterconditioning of relaxation over anxiety occurs fortuitously or unsystematically, whereas in behavior therapy this process is overt, systematic, and under the direct control of the therapist.

The Nature of the Therapeutic Relationship

The nature of the therapeutic relationship between therapist and patient in the behavioral therapies is, according to Hollander, an essentially "educative, teacher-pupil relationship" (71). In contrast to the psychodynamic transferential relationship but comparable to the working or therapeutic alliance in certain respects, the behavioral relationship may be portrayed as a deliberately structured learning alliance, in which, at its best, attention is drawn to the more current and presumably constructive aspects of the patient's personality in collaborating on the course of therapy.

Krasner depicted the behavior therapist as a learning technician or "social reinforcement machine" (72). Although this rubric may apply to all therapies to greater or lesser degrees, usually the behavioral therapist openly regards himself/herself as an instrument of direct behavioral influence or control, one who directly and systematically manipulates, shapes, and/or inserts his/her own values in the therapeutic encounter. In a comparable context, the thera-

[1]The terms "classical" versus "operant" conditioning procedures refer essentially to the respective sequence in the application of the stimulus. In classical conditioning the stimulus precedes and in operant conditioning the stimulus follows the behavioral response to be changed.

pist shapes his/her own behavior so as to be a social reinforcer for the patient. If the therapy does not proceed smoothly or effectively, the behavior therapist revises the behavioral plan or schedule to better fit the patient to treatment (71).

Behavior therapy deliberately does not dwell on the therapist-patient relationship; at most, it does so secondarily, that is, according to APA's Task Force on Behavior Therapy, "only to the extent that this is seen to be important in securing the patient's cooperation with the therapist's treatment plan" (73, p. 27).

The behavior therapist's use of warmth, acceptance, and any other relationship skills is common but relegated to the realm of secondary "relationship skills" that are not crucial therapeutic requirements for desired change to occur in the patient (71).

Techniques and Methods

In Ehrenwald's words, the behavioral schools of psychotherapy actively relinquish "the methods of the couch" and replace them with "the methods of the classroom and the pulpit" (7). The behavior therapist has at his/her disposal a large variety of conditioning, training, and other directive techniques. This repertoire may include any or all of the following: the more classical conditioning techniques of systematic desensitization combined with deep muscle relaxation, implosion, or assertiveness training; the operant techniques of positive or negative reinforcement; aversiveness training; shaping or modeling; and/or the more flexible directive techniques pertaining to the direct transmission of advice, guidance, persuasion, and exhortation. The latter methods more typically reflect the means by which behavior modification has been extended recently to the teaching or conditioning of cognitive behaviors or attitudes underlying specific behaviors, methods of philosophical indoctrination, or cognitive programming.

More generally, the behaviorist initially sets out to identify the patient's specific target behaviors or responses that need to be modified. These, in conjunction with the stimuli or environmental situations that give rise to the specific behaviors, constitute a behavioral formulation that may be regarded as the behaviorist's counterpart of psychodynamic formulation. The behavioral formulation is used for the purpose of setting specific treatment goals, which are usually made explicit to the patient at the outset. The initial interview typically aims to specify what situations or factors contribute to the maintenance of the particular responses in question and on what occasions those responses are most likely elicited. On this basis the behavioral conditioning program can then begin.

Wolpe's classical reciprocal inhibition therapy, which has as its direct aim the reduction or inhibition of anxiety responses through the substitution of relaxation responses for anxiety, typically uses two basic techniques for the purpose of juxtaposing relaxation with anxiety. The first technique is progressive deep muscle relaxation training and the second is systematic desensitization of anxiety through imagination. The patient is instructed to relax and then tense up for intervals of 10–15 seconds each; the patient repeats this maneuver using various different muscle groups or parts of the body followed by breathing exercises. Then, after discussing with the patient which real-life situations arouse the phobic symptoms, the therapist incorporates each scene into an anxiety list or anxiety hierarchy; this list constitutes the basic therapeutic tool. (Each scene or situation is arranged hierarchically on the basis of level of anxiety generated in the patient, with a ranking system of 1–10.) Starting with the weakest elicitor of anxiety in the hierarchy, the therapist asks the patient to imagine the anxious circumstance for a few minutes and then instructs the patient to concentrate on relaxing; this process is repeated until the patient can imagine the scene without feeling any anxiety. The therapist ascends the anxiety list item by item in the same manner. When this process is finished, the real-life situation that has created the phobia has lost its capacity to elicit anxiety.

A more recent variation of this approach is, ironically, a direct reversal of this procedure. The therapist starts not with the bottom but with the top of the anxiety continuum; the patient is flooded with the strongest anxiety-eliciting stimulus situation of his/her imagination and keeps this in mind until the anxiety dissipates. Then, with successive repetitions of the same scene, the patient's anxiety progressively lessens until he/she is immune to anxiety in that situation. This basic technique is referred to as implosion (Stampfl's implosive therapy).

A cognitive variation of this approach is the technique of thought-stopping. In this variation the patient puts into words the anxiety-producing situations instead of merely imagining them. As the patient speaks about himself/herself in these situations the therapist suddenly interrupts the train of anxious verbalizations by shouting "Stop!" This procedure is repeated on successive occasions until the patient validates the fact that this overt suppression has indeed served to reduce the frequency of the anxiety-loaded thoughts. Ellis' rational therapy represents an elaboration of this cognitive approach on a larger and more varied scale.

The behavioral counterpart of the psychodynamic procedure of working-through is behavioral rehearsal within the confines of therapy as well as assignments to be worked on outside of therapy; these are important parts of the total behavioral treatment. For example, the patient might be directly trained in certain social skills that may first be role-played or rehearsed within the course of therapy as well as explicitly instructed, tested out in outside real-life situations, and reviewed in subsequent sessions.

A special instance of this method is assertiveness training, a technique of instruction and practice of interpersonal behaviors involving the relatively direct expression of one's positive and negative responses to others. Wolpe claimed that assertive responses consti-

tute a major class of behaviors that could be used as an alternative to relaxation responses in the function of reciprocally inhibiting anxiety. Assertive training by means of behavior rehearsal, whether or not it is used as a technique for expressly countering anxiety, has been incorporated into a variety of schools that use the methods of the behavioral laboratory.

Variations on the Behavioral Theme

Three broad types of behavior therapies or behavior modification are considered under the umbrella of the behavioral theme (74): one, based on the early classical Pavlovian paradigm, primarily uses systematic desensitization or extinction of anxiety techniques (e.g., Wolpe's reciprocal inhibition therapy); a second type, based on an operant Skinnerian paradigm, uses direct reinforcement by means of reward/punishment procedures (e.g., Allyon and Azrin's token economy); and a third type, based on a human social learning paradigm, is contingent on direct modeling or shaping procedures (e.g., Bandura's modeling therapy). The latter type of therapy extends to a variety of new systems of directive psychotherapy that expressly aim at attitudinal or philosophical restructuring, using methods of the behaviorist's laboratory. Such so-called integrity therapies, although they share the fundamental learning or problem-solving stance, are usually more actively advisory and/or exhortative in their therapeutic techniques (e.g., Ellis' rational therapy, Glasser's reality therapy, and Sahakian's philosophic psychotherapy).

Another way of viewing the scope of these behavioral variations is through the evolution of their targets of change from external to internal alterations in man's learnings. The earlier behavior therapeutic systems addressed overt behaviors and fears (e.g., Wolpe); the more recent systems are directed to more covert values and beliefs (e.g., Ellis). The most recent approaches venture into the reaches of the most inaccessible and involuntary mental and physiological states and responses, such as heart rate, blood pressure, and brain waves (e.g., biofeedback).

THE EXPERIENTIAL THEME

The Nature of Man and His Ills

In terms of therapeutic ideology, a major source of divergence and disagreement for the founders of schools of psychotherapy since Freud has been their inability to reconcile what they regard as a largely deterministic or defeatist image of man. This includes the renunciation of a dynamic conceptualization of man as a predominantly passive or instinctually regressive recipient of his conflictual drives, subservient to the less conscious aspects of himself. The experientialists criticize classical psychotherapy's overcommitment to the canons of science and its concurrent underplaying of man's ethical dimension, that is, his will, choices, and moral relation to others. Through psychotherapeutic practices and overemphasis on technique, man becomes impersonalized, compartmentalized, calculated, managed, and/or analyzed and thereby diminished instead of truly experienced by others or himself (20, 26, 53, 75).

Haigh, an experientialist, has criticized the behavioral conceptualization as follows:

> The therapeutic process [is] essentially concerned with the experiential anguish of isolation and alienation. These experiences of isolation, encapsulation, alienation derive from social programming. . . . The central problem cannot be understood exclusively at the level of overt behavior, [but] must be understood as involving incongruence between overt behavior and inner experience. Reinforcement learning theory is inadequate for translating this central problem because it doesn't include concepts representing human experience. The behavioral therapy techniques associated with reinforcement learning theory are potentially harmful because they involve the very same programming approach which induced these neurotic problems in the first place (76).

The experiential conceptualization represents an increasingly emerging exaltation of man in order to counter alienation, that is, the fostering of the fullest exploration of the unique and universal nature of man's self. It is expressly devoted to the self-transcendent quality of human experience. In Frankl's words, "Man is basically striving to find and fulfill meaning and purpose in life" (77, p. 252).

This reaching out involves a transpersonal as well as intrapersonal dimension. According to Arendsen-Hein, the intrapersonal refers to an "ego-centered level . . . where the main concern is the discovery of one's individuality, of one's emotional states and their representation in the physical body"; the transpersonal, on the other hand, is "spiritually oriented towards ultimate reality, . . . unity on the human, universal, or cosmic plane . . . in which [the person] experiences a transcendence of his ego boundaries into a universal consciousness" (78).

The experientialist tends to view man as an inherently active, striving, self-affirming, and self-potentiating entity with almost limitless capacity for positive growth, and the experiential therapies typically opt for growth and not mere healing of illness. Their therapeutic goal is attaining maximal awareness or a higher state of consciousness, in which, according to May and associates, "to be aware of one's world means at the same time to be designing it" (20, p. 60). Experientialists therefore direct themselves to such expansive dimensions as self-determination, creativity, and authenticity and make use of a potpourri of methodologies that aspire to an ultimate integration of the mind, body, and more recently, soul of fragmented man.

The experiential stance historically reflects the incorporation of the basic philosophy of the European existentialist, with his/her concern for the essential issues of man's being or becoming; the methodology of the phenomenologist, who attempts to address data as

given in order to tap their meaning and to examine patients on their own terms without recourse to preconceived theoretical formulations of a causal or diagnostic nature; and, now more than ever, the religious teachings and techniques of the Eastern mystic, who presumes to bridge the mind-body split in seeking man's spiritual center. All, according to May and associates, focus on "man's most immediate experience . . . [i.e.,] that to fully know *what* we are doing, to feel it, to experience it all through our being, is much more important than to know *why*. For they hold, if we fully know the *what*, the *why* will come along by itself" (20, p. 83).

Pathology is regarded as the reduced expression of one's potential, the result of blocking and the loss of congruence with or repressing of one's internal self-experience. Both the psychodynamic and the experiential (essentially existential) themes depict the neurotic personality as suffering from repression and fragmentation. The dynamic view postulates repression of instinctual drives, especially sexual ones, while the experiential view construes repression as an ontological phenomenon, "the loss of a sense of being, together with the truncation of awareness and the locking up of potentialities which are the manifestation of this being" (20, p. 86).

Neurosis is a fundamental universal despair resulting from the individual's estrangement from himself and his society (or world). Comparably, anxiety, in marked contrast with the behavioral equation of anxiety with specific circumscribed fears, refers to "the anxiety of man facing the limits of his existence with the fullest implications . . . death, nothingness" (20, p. 118). Such anxiety manifests itself at every moment as man stands against the reaches of his own possibilities. Moreover, the origin of guilt accrues from the forfeiting of one's potential, for which the person alone is responsible.

In Maslow's words, pathology is "human diminution" (instead of neurosis), "the loss or not-yet-actualization of human capacities and possibilities" (79, p. 124). Thus health and illness, including all of the standard psychiatric categories, reside on a continuum—the differential between what one construes himself to be versus what one can become. The overall ideal or ultimate state of health refers to man as maximally conscious and real at every moment, to wit, according to Denes-Radomisli, man as "vital, immediate, spontaneous, authentic, and active" (80, p. 104).

Therapeutic or Change Processes

The experiential schools of psychotherapy trade intellectual cognition and insight for emotion and experience, forsaking the there-and-then of the distant past for the here-and-now of the immediate present.

Experiencing is a process of feeling rather than knowing or verbalizing; occurs in the immediate present; is private and unobservable but can be directly referred to by an individual as a felt datum in his own phenomenal field; acts as a guide to conceptualization; is implicitly meaningful, although it may not become explicitly so until later; and is a preconceptual organismic process. The many implicit meanings of a moment's experiencing are regarded not as already conceptual and then repressed; rather, they are considered in the awareness but as yet undifferentiated. In total, according to Gendlin, "therapeutic change occurs as a result of a process [of experiencing] in which implicit meanings are in awareness, and are intensely felt, directly referred to, and changed, without ever being put into words" (81, p. 239).

Therapeutic change through experiencing usually occurs by means of a real or congruent interpersonal relationship between the patient and the therapist. In the latter regard, May and associates said,

> Beyond all considerations of unconscious determinism—which are true in their partial context—the only thing that will grasp the patient, and in the long run make it possible [for him/her] to change, is to experience fully and deeply that [he/she] is doing precisely this to a real person . . . in this real moment. (20, p. 83)

One variation of this thesis, especially applicable to Roger's client-centered therapy, reflects the underlying positive belief that every organism has an inborn tendency to develop its optimal capacities as long as it is placed in an optimal environment. Thus, according to Hoehn-Saric, the patient is offered "an optimistic self-image; he understands that he is basically good and full of potentials. . . . Therefore, the therapist does not need to challenge or shape the patient, he has only to provide the warm and understanding milieu which will enable the patient to unfold his latent potentials" (82, p. 261).

Unlike transference, which is dependent on the revival of a former interpersonal relationship, experiential encounter works "through the very fact of its novelty." Through encounter the therapist serves as a catalyst in whose presence the patient comes to realize his own latent and best abilities for shaping his own self (20).

The Nature of the Therapeutic Relationship

Although methods may vary, the real here-and-now therapeutic dialogue or mutual encounter between therapist and patient is the sine qua non of many of the experiential schools. It is, according to Ford and Urban, "an emotionally-arousing human relationship in which each person tries to communicate honestly with the other both verbally and nonverbally" (26, p. 470).

Rogers described the flavor of the therapeutic encounter as follows:

> I let myself go into the immediacy of the relationship where it is my total organism which takes over and is sensitive to the relationship, not simply my consciousness. I am not consciously responding in a planful or analytic way, but simply in an unreflective way to the other individual, my reaction being based (but not consciously) on

> my total organismic sensitivity to this other person. I live the relationship on this basis. (83, pp. 267–268)

These approaches to psychotherapy ideologically aspire to an egalitarian treatment model. The human alliance is not of physician to patient or teacher to student but of human being to human being. May and associates presented the following rationale: "The therapist is assumedly an expert; but, if he is not first of all a human being, his expertness will be irrelevant and quite possibly harmful" (20, p. 82). Rogers stated that if the patient is viewed as an object the patient will tend to become an object (83). Therefore, the therapist says, "I enter the relationship not as a scientist, not as a physician who can accurately diagnose and cure, but as a person, entering into a personal relationship" (83, p. 267). Naturally, what one construes to fall within the domain of personal or real in a therapeutic relationship is open to interpretation.

In sum, although Seguin pointed out that the quality of the healer-patient relationship (and the form of therapeutic love or eros that is transmitted) is "different from the one between father and son, teacher and pupil, friends, and, of course, lovers" (84), the experiential theme rejects the paradigm of the parent-child relationship of the dynamic theme and the teacher-student paradigm of the behavioral theme and opts for something closer to the latter conceptualization (i.e., friends and lovers) in its paradigm of the therapeutic relationship.

Techniques and Methods

There is an assortment of schools of psychotherapy within the experiential theme that recoils at the idea of therapeutic technology. These schools, which are predominantly existential, renounce technique as part of their philosophy of understanding human existence. They feel that the chief block in the understanding of man in Western cultures has been an overemphasis on technique and a concomitant tendency to believe that understanding is a function of or related to technique. Rather, according to May and associates, they feel that "what distinguishes [forms of] existential therapy is not what the therapist would specifically do, . . . but rather the *context* of his therapy" (20, p. 77). That is, according to Chessick, it is "not so much what the therapist says [or does] as what he *is*" (53, p. 243). Indeed, in this regard the existential schools of psychotherapy have been criticized for their vagueness regarding technical matters in the conduct of psychotherapy. Ford and Urban's analysis of their approach concluded that "they have developed a new way of thinking about patients, but it does not lead them to *do* anything different in treatment" (26, p. 469).

Less harshly, the experiential schools aspire to flexibility or innovation in their actual methods as long as these methods are useful in the therapist's attempt to experience and share as far as possible the being of the patient. Here the aim or rationale of all techniques would be to enter the phenomenological world of the patient. In direct contrast to the view of the dynamic therapist, the experiential therapist does not concern himself/herself with the patient's past, the matter of diagnosis, the aspiration of insight, the issue of interpretation, or the subtle vicissitudes of transference and countertransference. Unlike the behavioral therapist, the experiential therapist expressly does not set goals for the patient and does not direct, confront, or otherwise impose his/her personality on the patient with directives in the form of behavioral instructions or problem-solving preferences. Techniques that involve placing the therapist's judgments or values above those of the patient are considered anathema to the requirements of unconditional acceptance of the patient and placing the locus of control within the patient. It may also be noted here that Rogerian methods as well as others within the more classically existential framework retain a methodological framework of essentially verbal interchange between therapist and patient.

Although they share the same basic faith in the therapeutic encounter and the emphasis on feelings, other schools under the experiential umbrella are often antiverbal in approach. Such schools (e.g., Gestalt therapy), view overintellectualization as part of the patient's problem, i.e., a manifestation of defense against experiencing or feeling, and discourage it as part of the therapeutic endeavor. These therapies attempt to accentuate activity over reflection, emphasize doing rather than saying, or, at the minimum, aim to combine action with introspection. The goal of experiencing oneself includes developing the patient's awareness of bodily sensations, postures, tensions, and movements, with an emphasis on somatic processes. Awareness of oneself as manifested in one's body can be a highly mobilizing influence. The main thrust of therapy is therefore to actively arouse, agitate, or excite the patient's experience of himself/herself, not simply let it happen.

Among the techniques for expressing one's self-experience in such schools is the combination of direct confrontation with dramatization, i.e., role-playing and the living out of fantasy in the therapeutic situation. This means that under the direction (and often the creation) of the therapist the patient is encouraged to play out parts of himself/herself, including physical parts, by inventing dialogues between them. Performing fantasies and dreams is typical and considered preferable to their mere verbal expression, interpretation, and cognitive comprehension. In variations of the somatic stance, body and sensory awareness may be fostered through methods of direct release of physical tension and even manipulations of the body to expel and/or intensify feeling.

In yet other attempts to unify mind, body, and more especially, spirit, the immediate experience of oneself by focusing on one's spiritual dimension is sought. This is most often accomplished through the primary technique of meditation. The ultimate state of profound rest serves to transcend the world of the indi-

vidual ego in that it is a higher reality or state of consciousness that the individual ego subserves. Major methods of will training and attention focused on a special word-sound or mantra, for example, serve to create an egoless or nonegocentered transcendent state.

Variations of the Experiential Theme

The therapeutic systems that have evolved under the experiential theme represent various approaches, each propelled by the immediate moment and geared toward the ultimate unity of man. These include the following: 1) a philosophic type, which reflects existential tenets as a basis for the conduct of psychotherapy and pivots on the here-and-now mutual dialogue or encounter while retaining essentially verbal techniques (e.g., Roger's client-centered therapy and Frankl's logotherapy); 2) a somatic type, which reflects a subscription of nonverbal methods and aspiration to an integration of self by means of focusing attention on subjective body stimuli and sensory responses (e.g., Perls's Gestalt therapy) and/or physical-motor modes of intense abreaction and emotional flooding in which the emphasis is on the bodily arousal and release of feeling (e.g., Lowen's bioenergetic analyses and Janov's primal scream therapy); and, finally, 3) a spiritual type, which emphasizes the final affirmation of self as a transcendental or transpersonal experience, extending man's experience of himself to higher cosmic levels of consciousness that ultimately aim to unify him with the universe. This is primarily accomplished by means of the renunciation of the individual ego in the establishment of an egoless state by meditation (i.e., relaxation plus focused attention), in which one reaches a state of profound rest (e.g., Transcendental Meditation), a spiritual synthesis that may be amplified by various techniques of self-discipline and will-training and practice of disidentification (e.g., Assagioli's psychosynthesis).

CONCLUSIONS

Individual names given to different schools of psychotherapy often attempt to capture, if not exalt, their uniqueness; at the same time, the very names may serve to mask their derivations and the similarities they share with other systems. The schema presented here subsumes a large array of therapeutic schools that have proliferated under three broad themes since Freud. Each major theme as depicted here in its prototypic or pure form has a unity of its own; each dimension is broadly congruent with the others in its domain and is overtly antithetical to those dimensions described in the other two themes. However, in reality these are rarely categorical distinctions. Therapeutic boundaries in actual practice can and do overlap.

The systematic approach presented in this paper allows us to consider the dimensions of greatest departure as well as convergence in the ever-expanding field of the psychotherapies. In so doing, it is hoped that psychotherapeutic schools and their therapeutic processes will be more closely and critically explored.

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Psychotherapy of Severe Depression

BY SILVANO ARIETI, M.D.

The author discusses his clinical experiences in treating severely depressed patients with psychotherapy. The psychotically depressed person cannot use normal mechanisms to recuperate from sadness because he is prevented from doing so by a preexisting life ideology that may include living for a dominant other or a dominant goal and that restricts his vision of alternative approaches to living. The therapist treating the severely depressed must initially assume a strong role; later, he helps the patient recognize depressive cognitive patterns and guides and motivates him to try other ways of living.

PSYCHOTHERAPY of affective psychoses is still in the pioneer stage. Although depression has been treated with psychotherapy for a long time, generally the cases treated had not reached the psychotic level. It is true that, unlike schizophrenia, psychotic depression was treated psychoanalytically relatively early in the history of psychoanalysis (1, 2). However, these cases have been very few and as a rule the patients were treated in the intervals between the attacks of depression.

According to my clinical experiences, the intensity of the depression should not deter the therapist from making psychotherapeutic attempts (3). Psychotherapy becomes a necessity when drug therapy and shock therapy have been ineffective, when the syndrome recurs in spite of these treatments, or when the patient refuses to try physical therapies again. Contrary to common belief, most cases of psychotic depression do respond to psychotherapy if a proper therapeutic procedure is adopted.

The procedure I am going to describe was applied to 12 cases of extremely severe depression treated on an ambulatory basis. A much more extensive report of these cases will appear elsewhere (4). The patients were 9 women and 3 men; 10 of them had suicidal ideation. The patients were treated for at least 18 months with a minimum of 2 sessions a week. After at least 3 years from termination of treatment I can now report that full recovery with no relapses was obtained in 7 patients, marked improvement in 4, and failure in 1.

It may be useful before proceeding to compare the psychotherapist's tasks in treating a case of psychotic depression and a case of schizophrenia. Both the schizophrenic and the very depressed patient lament what they have come to know and feel about life. In both conditions there is a close interweaving of a drama of the present with a drama of the past. In both conditions the therapist must understand each of the two dramas. The schizophrenic rejects and symbolically destroys the world, which he perceives as hostile, and attempts through projection and special cognition to rebuild his own private universe. Thus his failure and his concern tend to become of cosmic magnitude. The person who is depressed to a psychotic degree does not reject the world, and acts to a certain extent like a mother who does not reject a bad child. The psychotically depressed patient does not even reject his suffering, but accepts it, all of it, and the suffering expands more and more, into a psyche that seems endless in its capacity to experience sorrow.

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PSYCHODYNAMIC VIEW OF PSYCHOTIC DEPRESSION

Normal Sadness

The patient cannot put into operation the mechanisms adopted by a normal person who recuperates from a feeling of sadness, grief, or bereavement (5). For example, a normal individual hears the news of the unexpected death of a person he loved. After he has understood and almost instantaneously evaluated what that death means to him, he experiences shock, then sadness. For a few days all thoughts connected with the deceased person will bring about a painful, almost unbearable feeling. Any group of thoughts even remotely connected with the dead person will elicit sadness. The individual cannot adjust to the idea that the loved person does not live any more. And, since that person was so important to him, many of his thoughts or actions will be directly or indirectly connected with the dead person and will therefore elicit sad reactions.

Nevertheless, after a certain period of time, that individual adjusts to the idea that the person is dead. By being unpleasant, sadness seems to have a function—its own elimination. It will be removed only if the individual is forced to reorganize his thinking and to search for new ideas so that he can rearrange his life. He must rearrange especially those ideas that are connected with the departed, so that the departed will no longer be considered indispensable—indispensable,

that is, to the equilibrium of the psychological structure of the survivor. Like pain, sadness stimulates a change in order to be removed, but it is a psychological change, an ideational change, a rearrangement of thoughts and clusters of thoughts. Eventually actions will also be altered as a consequence of this cognitive rearrangement. Bowlby (6–8) has described the active search in children deprived of their mother, and Parkes (9) has reported the various ways in which widows search to repair their losses. In other words, in normal sadness, after a period of slowness or inactivity that prevents quick responses there is a reorganization of functions, first cognitive and then motor, which aims at bringing about a return to a state of normality.

The person who is psychotically depressed cannot use these recuperative mechanisms. He finds himself in a psychological trap from which he cannot escape. The overpowering feeling of depression keeps in a state of unconsciousness or dim consciousness cognitive structures and patterns of life that started at an early age. In other words, depression has, among other functions, that of repressing cognitive structures.

Ideology of Psychotically Depressed Patients

My studies of patients treated with psychotherapy indicate that there is always a preceding ideology, a way of seeing oneself in life in general, in relation with another particular person, or in relation to a goal to be achieved that prepares the ground for the depression. Although depressive affect prompts the person to search for and find pessimistic ideas that justify the affect, the opposite initially occurs. The patient's ideology preexisted and was kept alive for a long time in conscious or unconscious forms. Although the patient might have been aware of the importance of these systems of cognitive constructs, he was not conscious of their origin, of how much they involved, or of all the ramifications, assumptions, presuppositions, and feelings connected with them. These cognitive domains in most cases originated in childhood and continued to accrue throughout life.

In many cases these cognitive structures and patterns made the patient live not for himself but for another person, whom I have called the dominant other. The dominant other is represented most often by the spouse, less frequently by the mother, a lover, an adult child, a sister, the father. The dominant other may be represented through anthropomorphization by the firm where the patient works, a social institution, or class of people. Some other patients live for an inaccessible aim, which I have called the dominant goal.

When the patient realizes the failure of his pattern of living, he also recognizes that he is unable to change it. Thus he finds himself in a state of helplessness and hopelessness. A frequent example is that of a woman who used to consider her husband a dispenser of love and affection, a protector, a friend, a partner in every respect. She then starts to see him as an authoritarian person who imposed his rule, at times in a subtle, hardly visible way, at other times in a manifest manner. The patient's own life, at first believed devoted to love and affection, is seen now as not genuine. The patient has been excessively compliant, submissive, and accommodating. By doing always what the husband wanted and denying her wishes, she has not been true to herself; as a matter of fact, she feels she has betrayed herself. The patient may exaggerate and see the authoritarian husband as a real tyrant, somebody who deserves not love, but hate, somebody who enslaved her and changed her real nature. In most cases she does not realize that she also played a role in establishing this type of interpersonal relation. By following a pattern of submissiveness that started early in life she developed ingratiating attitudes and was willing to accept fully a patriarchal type of society and family and to keep the dominant other, in the belief that she was perfectly contented or even happy in her way of living.

The emerging new ideas about the husband, however, cannot be accepted. A woman who had not followed the pattern of submission and dedication at any cost would now try new ways for what remains of her life (e.g., separation, divorce, new affections), but the patient cannot do this. She cannot conclude that she has wasted so much of her life. She still needs the same dominant other to praise her and to approve of her to make her feel worthwhile. How could he continue in that capacity if she expresses hate, rebellion, or, in some cases, even self-assertion? The patient has reached a critical point at which a realignment of psychodynamic forces and a new pattern of interpersonal relationships are due, but she is not able to muster them. This is her predicament. She is helpless. She either cannot visualize alternative cognitive structures that lead to recuperative steps or, if she is able to visualize them, they appear unsurmountable. At other times these alternatives do not seem unrealizable, but worthless, since she has learned to invest all her interest and desires only in the relationship that failed.

Depression following realization of failure to reach the dominant goal is more common among men. The patient realizes that he is not going to be a great doctor, lawyer, politician, writer, actor, industrialist, lover, or musician, but he cannot change the direction of his goal. If the goal is not achieved, life is worthless. If he wanted to be a great conductor, a Toscanini, now he has to face the fact that he is not a Toscanini, that he is himself, John Doe. But he has no respect for John Doe, as he should. He believes John Doe is nothing. There is some tangential and partial justification in the patient's assessing himself in this negative way because he spent so much of his thoughts in being Toscanini, and his psychological life without this overpowering fantasy seems empty. The limitations of the patient, determined by his rigidity and adherence to an inflexible pattern of life, do not provide him with alternatives. As in the patient who experienced failure in the relation with the dominant other, the patient may visualize alternatives but they seem either unsurmountable or worthless.

If space permitted, I would also touch on the inability to find alternative cognitive structures in patients who become depressed after loss of spouses, parents, positions, hopes, etc. The post-partum depression occurring in the woman who cannot accept the new pattern of life required by motherhood is another very complex situation (10).

In these cases sadness cannot be removed and is transformed into deep depression. When unpleasant ideas occur they are submerged by the wave of depression. The cognitive basis of the condition is repressed, but the painful feeling is very intensely experienced at the level of consciousness. This last remark may seem at variance with what we observe in many patients who express depressive ideas about the way they view themselves, the world, and the future. Beck (11–13) has carefully described the cognitive elements that keep the patient in a state of depression. In my experience with cases of severe depression, these depressive thoughts are used by the patient to justify his preexisting depression. They replace much more basic and more intense depressive trends, which have to be studied longitudinally, since they go back to the early life of the individual (5).

THERAPEUTIC PROCEDURE

Initial Tasks of the Therapist

The first task of the therapist is not that of interpreting to the patient the inappropriateness of his patterns of living or the maladaptive quality of his endless sorrow but that of entering into his life with a strong and significant impact.

The therapist assumes an active role. He is a firm person who makes clear and sure statements. He is compassionate, but not in a way that can be interpreted as acknowledgment of helplessness. He tells the patient that he knows how deep his anguish can be, but that he also knows that depression does not come from nothing. There is always a reason, which the patient, alone, cannot find. In other words, the patient is invited to lean on the therapist.

When the therapist succeeds in establishing rapport and proves his genuine desire to help, to reach, to nourish, to offer more clarity about certain issues and hope about others, he will often be accepted by the patient, but only as a dominant third, i.e., a third person in addition to the patient and the dominant other. Immediate relief may be obtained because the patient sees in the therapist a new and reliable love object. Although the establishment of this type of relatedness may be helpful to subsequent therapy, it does not constitute a real cure; as a matter of fact, it may be followed by another attack of depression if the patient realizes the quality and limitation of this relation. The therapeutic approach must proceed toward a more advanced stage, in which the therapist is no longer a dominant third, but a significant third—a third person who, with his firm, sincere, and unambiguous type of personality, wants to help the patient without making threatening demands or requesting a continuation of the patient's pathogenetic pattern of living. Whereas the dominant other is rigid and static, the significant third will change rapidly and will eventually appear as a person who shares life experiences without aiming at domination.

The point must be stressed, however, that in the initial stage of the psychotherapy of severe depression, like in the psychotherapy of schizophrenia, the therapy must somewhat conform to the patient's pathology and, often, in less pronounced forms, even repeat it. Otherwise treatment is immediately experienced by the patient as dangerous and is rejected before a meaningful therapeutic relatedness has been established.

Once the therapist has been able to gather enough information, the relation with the dominant other must be interpreted. The patient must come to the conscious realization that he did not know how to live for himself. He never listened to himself; in situations of great affective significance he was never able to assert himself. He cared only about obtaining the approval, affection, love, admiration, or care of the dominant other. As Bemporad (14) described, the patient was incapable of autonomous gratification; he had to obtain it from the dominant other.

Potential Problems

When the therapist succeeds in making some headway in understanding the patient's psyche, several developments that must be promptly coped with may occur. The patient may become less depressed but angry, either at the dominant other or the therapist, whom he would like to transform into a dominant third. The anger and hostility toward the dominant other (most frequently the spouse) is at times out of proportion. Once the ideations which were repressed or kept at the periphery of consciousness come to the surface, the dominant other may be seen as a tyrant, a domineering person who has subjugated the patient. The therapist may have a difficult task in clarifying the issues. At times the dominant other has really been overdemanding and authoritarian and has taken advantage of the placating, compliant qualities of the patient. Often, however, it is the patient himself who, by being unable to assert himself and by complying excessively, has allowed certain patterns of life to develop and persist. Now, when he wants to change these patterns, he attributes the responsibility for them to the dominant other. No real recovery is possible unless the patient understands the role that he himself has played in creating the climate and pattern of submissiveness. Similarly, if the major pathogenetic constellation of his psyche is his concern with the dominant goal, he must understand why he gave such supremacy to the achievement of that goal. Did he see in such a goal the only possible meaning of his life, and why?

Helping the Patient Perceive New Ways of Living

The therapist must not only help the patient bring to full consciousness cognitive patterns and their effect on his life; he must also gradually guide the patient to visualize different patterns and to invest them with hope and desires. The patient who has understood the role he has played in the dynamics of his condition will lose a sense of passivity and will feel less helpless and less inclined to accept a feeling of hopelessness. He will learn to assert himself and to aim at what is really meaningful to him and gratifying.

Bemporad and I have elsewhere (4) described the varieties and details of the complicated psychotherapeutic approach to the severely depressed. However, even in a short presentation like the present one, a few more important issues cannot be omitted.

In a considerable number of patients, although a smaller number than used to be, the depression is accompanied by a profound guilt feeling. The patient feels responsible for whatever has happened that brought about his psychological collapse. If some energy is left, it must be used for self-punishment. He feels vaguely that enough punishment will restore a self-image acceptable to him. The guilt is often actually based on hate for the dominant other, in a way reminiscent of the sadistic trends of the depressed that Freud (2) described as directed against the incorporated love object. The guilt feeling has repeatedly helped the patient to choose a pattern of atonement and submission to the dominant other in order to placate him and to obtain his love and approval. If such approval is not obtained, the patient's guilt feeling increases because in his cognitive structure lack of approval means not having done enough to deserve it. Moreover, in accordance with his patterns of living he has learned to transform anxiety into guilt. As painful as the guilt feeling is, the patient is aware that the possibility of suffering, atoning, and placating is within his capabilities, whereas with anxiety he is at a loss; he does not know what to do about it. It is important to explain these mechanisms to the patient. However, the greatest relief from guilt feeling will occur when he has transformed his relation with the dominant other.

When the patient has somewhat improved and is able either by himself or with the help of the therapist to realize that alternative ways of living are available to him, he may state that he is afraid to embark upon them, even though the depression has lifted and the motor retardation is no longer present. The patient may be afraid of attempting new ways of living because they are unknown and unfamiliar. Other inhibiting factors stem from the fact that these new ways seem extremely remote and do not arouse in him motivation toward their achievement. The patient, who has been for so long entrenched in his complexes and enduring patterns of living, has never day-dreamed about or invested feelings in these alternatives. Again the therapist must help the patient gradually to develop the lacking motivation for different paths of living.

The patient who has partially recovered continues, nevertheless, to feel more at home with the feeling of depression than with other moods. He may exploit any negative event or thought to become depressed again. We must repeatedly show him how he uses every minor unpleasant event or thought to justify his depression, which initially stemmed from a deeper source.

At other times the patient entertains ideas half-way between these superficial depressive ideas that he exploits and ideas that are genuinely related to the deep problems of which he has now acquired awareness. He may brood over what he did not have or may have a feeling of self-betrayal. He has some realization that some aspirations have to be given up; he has the feeling that he cannot count on the recurrence of opportunities that he did not grasp, and so on. The therapist must guide the patient so that he can catch himself in the act of having these ideas or in an attitude in which he expects to be or to become depressed. If he becomes aware of these ideas and of expecting to be consequently depressed, he may be able to intercept the process and to avert the depression. He will become more and more receptive to alternative ways of living.

As a final point, I wish to mention that psychotherapy as described in this paper does not exclude concurrent drug treatment with antidepressants such as MAO inhibitors or tricyclics. I have resorted to such combined treatment with satisfactory results. I want to stress again, however, that psychotherapy alone can be an effective treatment in severe depression and that it may have to be used to the exclusion of other types of treatment in the conditions which I mentioned at the beginning of this paper.

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The Isaac Ray Award

The American Psychiatric Association invites nominations for the Isaac Ray Award for 1978. This award, in memory of Margaret Sutermeister, is presented to a person (psychiatrist, attorney, or someone from a discipline related to human behavior) who has made outstanding contributions to forensic psychiatry or to the psychiatric aspects of jurisprudence. The purpose of this award is to promote better understanding between the law and psychiatry. The award, which will be presented at the Convocation of Fellows at the annual meeting of the American Psychiatric Association in Atlanta, Georgia, in May of 1978, includes an honorarium of $2,000. The recipient agrees to present his or her work at an institution of higher learning (or other suitable location) and to present the manuscript for publication. The presentation will be located and timed to give maximum exposure to students and practitioners of law and medicine and to other professionals.

Nominations are requested by the deadline of September 12, 1977, from any interested professional, and may be submitted to: Naomi Goldstein, M.D., Chairperson, Isaac Ray Award Board, 15 West 81st St., New York, N.Y. 10024.

Continuing Education, Recertification, and Examination Anxiety

BY EUGENE B. FEIGELSON, M.D., AND WILLIAM FROSCH, M.D.

A substantial majority of the 645 psychiatrists responding to a questionnaire survey favored mandatory continuing education and evidence of continuing education as a requirement for relicensure. The respondents' overwhelming repudiation of recertification may be based on examination anxiety. The respondents recognized the value of continuing education, were even willing to document it in order to be relicensed, but did not wish to be examined. Those respondents who were younger, Board-certified, and had an academic appointment were more likely to favor continuing education requirements in order to be relicensed. Private practitioners were underrepresented among the respondents, highlighting the important role that APA and its district branches must play in developing meaningful continuing education opportunities for psychiatrists.

CONTINUING MEDICAL education following postgraduate training has been of concern to medical educators for many years. It has become a subject of increasing importance in the past few years. The number of courses registered by the American Medical Association increased from 1,105 in 1961 to 4,862 in 1976 (1, p. 589); similarly, the number of continuing education opportunities for psychiatrists has been on the increase (1). In spite of the increasing concern about continuing education and recertification in psychiatry, little is known about the practicing psychiatrist's attitudes regarding these matters. This is at least in part the result of a paucity of studies in the area. A computer search of the literature of the last 5 years using such code words as "continuing education," and "recertification" yielded just over 100 papers. However, 50 of these dealt with the education of nonpsychiatrists, 25 with residency training, and 10 presented pious platitudes but no data. There were 7 papers on peer review and only 9 substantive contributions to the continuing education of psychiatrists. We undertook this study in an effort to provide the public deliberative process with information concerning the perceived needs and desires of the psychiatric community.

Revised version of a paper presented at the 129th annual meeting of the American Psychiatric Association, Miami Beach, Fla., May 10–14, 1976.

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METHOD

We used a questionnaire designed to elicit the attitudes of different segments of the psychiatric community toward continuing education, recertification, relicensure, and curriculum.[1] The questionnaire was a modification of a questionnaire used by Naftulin and associates (2). The two-page questionnaire was mailed to the 1,500 members of the New York County District Branch of the American Psychiatric Association (APA). A total of 645 members (43%) responded to the single mailing with completed questionnaires. Demographic data and professional activity were correlated with the following key questions:

1. Should continuing education in psychiatry be required?
2. Should recertification in psychiatry be required?
3. Should evidence of continuing education be necessary for relicensure?
4. Should the APA Office of Education and the district branches become the accrediting agencies of continuing education courses?

An overall count was taken on each item in the questionnaire and then the respondents were separated into 4 groups representing the most common patterns of response to questions 1, 2, and 3 (i.e., yes, yes, yes; yes, no, yes; yes, no, no; and no, no, no). Analysis of the data revealed significant relationships between attitudes toward continuing education, recertification, and relicensure and age, Board status, and academic affiliation.

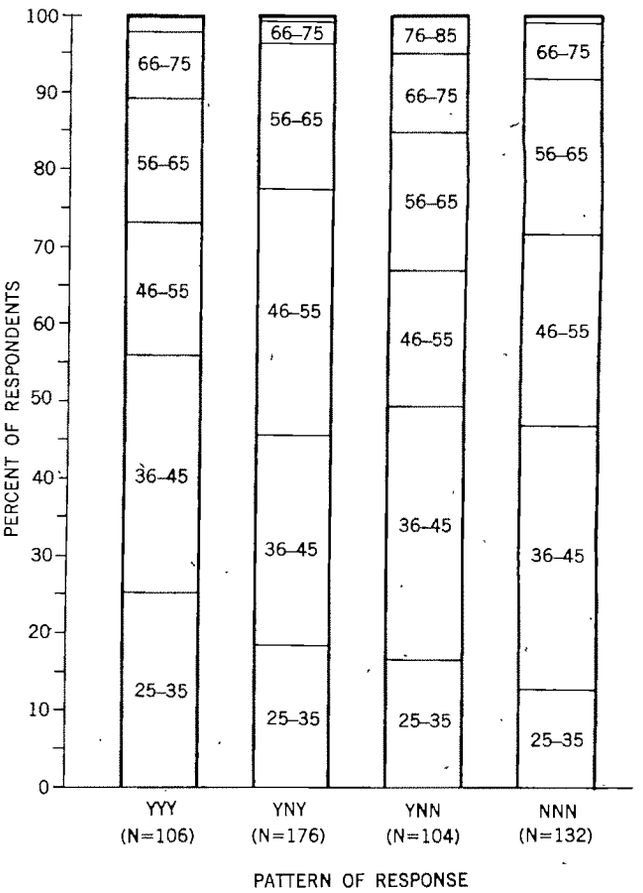
We compared the respondents with a representative sample of the New York County District Branch (N=400) by age, year of graduation from medical school, Board status, principal professional activity, academic appointment, and whether American or foreign medical graduate. The representative sample was obtained by selecting every fourth name from an alphabetical list of the district branch members and then obtaining the above information from the *Biographical Directory of the American Psychiatric Association*.

[1]The questionnaire is available from the authors on request.

TABLE 1
Comparison of the New York County District Branch Sample and the Questionnaire Respondents, in Percents.

Item	District Branch Sample (N=400)	Questionnaire Respondents (N=645)
Age		
25–35	9	17
36–45	31	29
46–55	22	25
56–65	21	19
66–75	13	8
76–85	4	2
Year graduated from medical school		
1900–1909	2	0
1910–1919	1	1
1920–1929	8	6
1930–1939	19	16
1940–1949	19	24
1950–1959	24	9
1960–1969	28	39
1970–	1	4
Board-certified		
Yes	58	67
No	42	33
Academic appointment		
Yes	53	70
No	47	30
Foreign medical graduate		
Yes	21	18
No	79	82
Principal professional activity		
Private practice	80	57
Institutional	13	22
Academic	7	21

FIGURE 1
Relationship Between Pattern of Response and Age



Statistical significance was determined by complex chi-square test and contingency coefficient.

RESULTS

Table 1 presents the findings from the comparison of the 2 groups. While there was no important difference in age between the sample group and the respondents, a lower percentage of the sample had graduated from medical school after 1959 (29% versus 43%). More of the respondents were Board-certified (67% versus 58%), had an academic appointment (70% versus 53%), and were principally involved in institutional and/or academic work (43% versus 20%) rather than private practice. American and foreign medical graduates were about equally represented in both groups. Perhaps the most important finding is that full- and part-time private practitioners, who constituted the largest subgroup of questionnaire respondents (80%), were the slowest to respond to the survey.

Regarding the overall response to the 4 key questions mentioned before, the respondents strongly favored mandatory continuing education in psychiatry (434 versus 151)[2] and roundly repudiated mandatory recertification (475 versus 135). A surprising majority (325 versus 272) favored evidence of continuing education as a requirement for relicensure. Most of the respondents (531 versus 71) felt that the APA Office of Education and the district branches should become the accrediting agencies of continuing education courses.

Having determined some general characteristics of the respondents, we attempted to demonstrate the relationship between the attitudes revealed by answers to questions 1, 2, and 3 and those factors shown by statistical tests to be significant.

Figure 1 employs response patterns as the basis on which to compare age groups. The overall relationship between these two is statistically significant ($p<.025$). Most readily apparent is the proportion of the YYY group aged 25–35. This proportion decreases as negativity of response increases. A second set of statistical analyses between each age group and the responses revealed a trend that is interesting in terms of the relationship between aging and negativity of response.

[2]The numbers given here and in other parts of the paper add up to less than the total sample because not all respondents replied to each question.

FIGURE 2
Relationship Between Board Certification and Pattern of Response

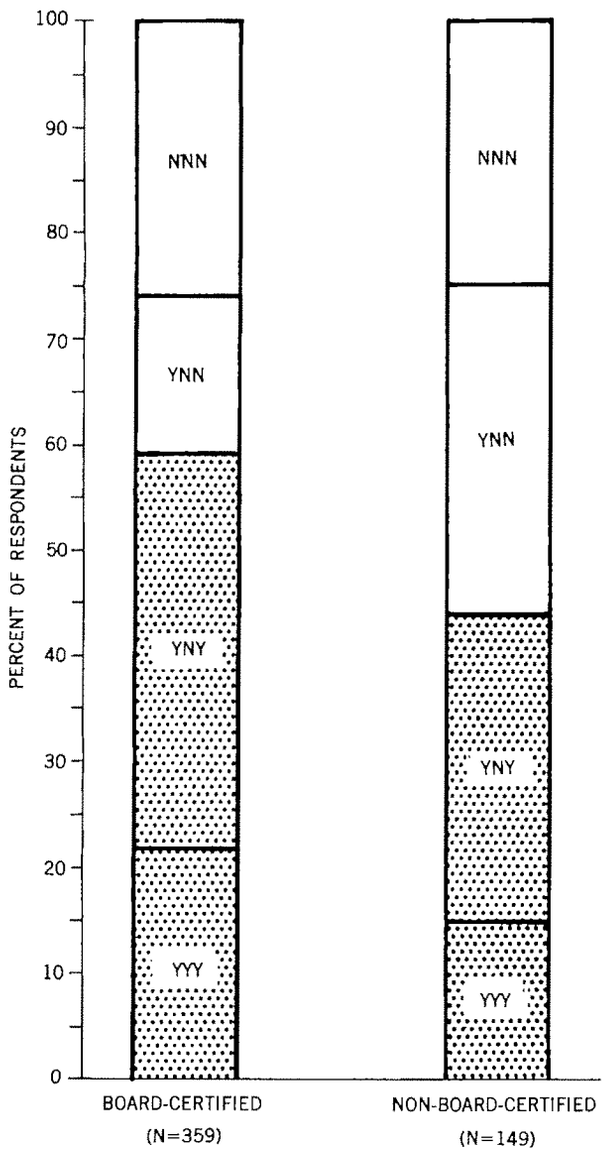
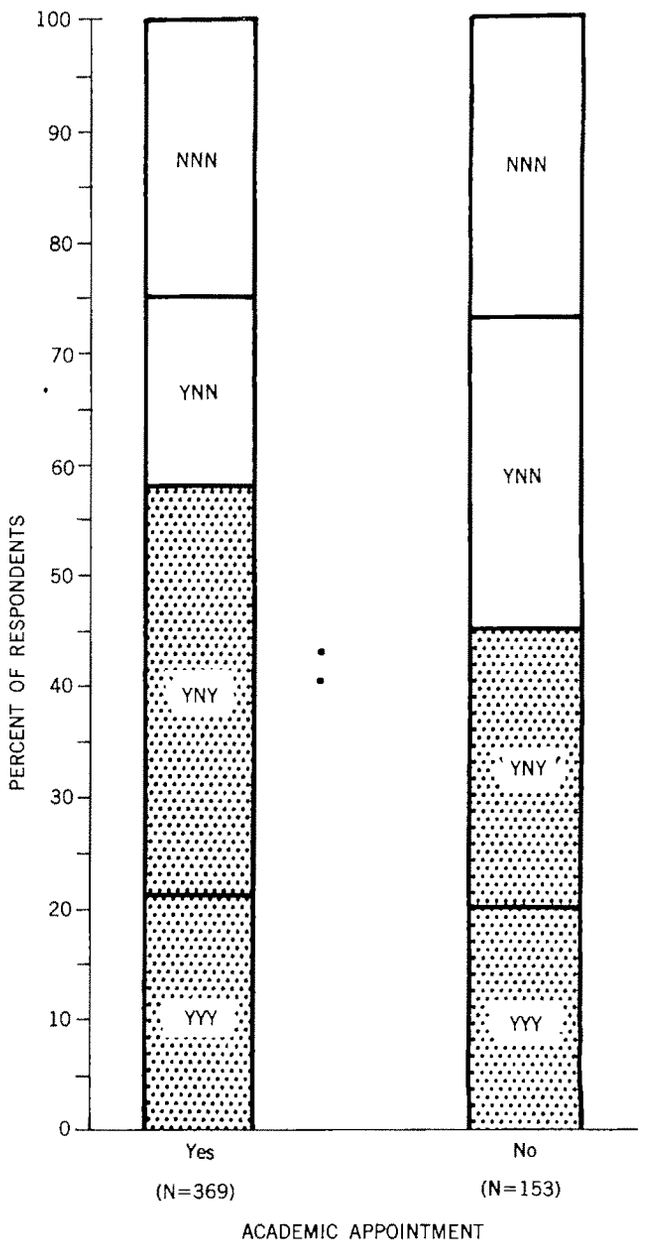


FIGURE 3
Relationship Between Academic Appointment and Pattern of Response



The results were as follows: 25–35, $p<.10$; 35–45, $p<.25$; 46–55, $p<.50$; and 56–65, $p<.90$.

Figure 2 compares the response patterns of Board-certified and non-Board-certified psychiatrists. The distinction between the two is best illustrated in the response to question 3 regarding the requirement of continuing education for relicensure. When the YYY groups are combined with the YNY groups, the result is to isolate question 3. Fifty-nine percent of the Board-certified respondents answered "yes" to question 3, compared with only 44% of the non-Board-certified respondents ($p<.005$). It is interesting to note that equal proportions of Board-certified and non-Board-certified psychiatrists fell into the NNN response group. One might speculate that those inclined to say "no" to all three questions did so out of a consistent philosophical stance.

The findings concerning the relationship between academic appointment and response pattern were similar to those regarding Board status. Figure 3 is nearly identical to figure 2; 58% of those with academic appointments answered "yes" to question 3, compared with only 45% of those without an academic appointment ($p<.01$). About equal proportions of academically affiliated and non-academically affiliated psychiatrists fell into the NNN response group.

Figure 4 illustrates the relationship between professional activity and response pattern. While there were no significant findings, it is important to remember that the respondent group was not fully representative of the district branch membership. As can be seen in

FIGURE 4
Relationship Between Pattern of Response and Professional Activity

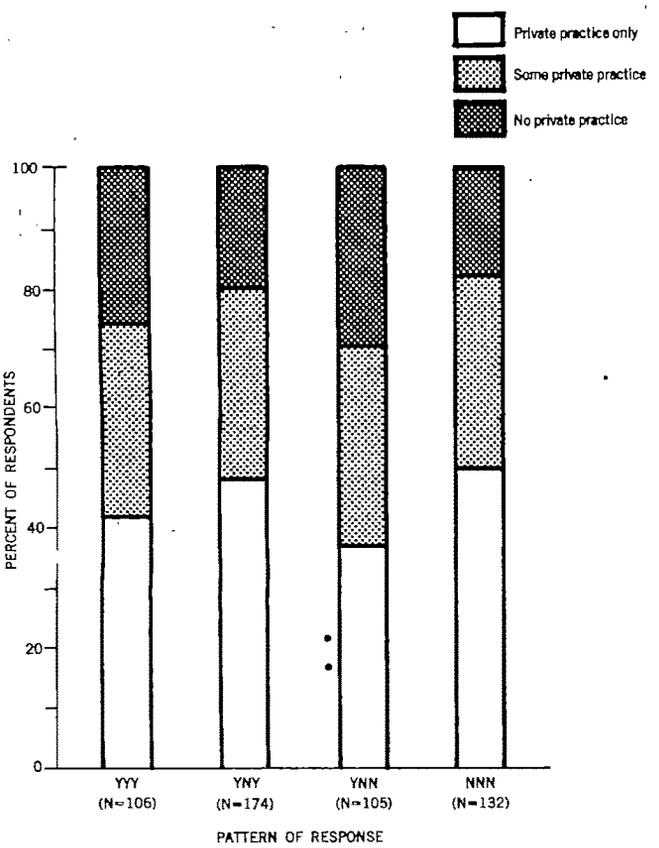


table 1, private practitioners were underrepresented among the respondents.

DISCUSSION

The test of the value of continuing education in psychiatry will be its effect on the quality of care, a difficult measurement. What is not difficult to measure is that a significant proportion of the outpatient psychiatric care in this country is provided by private practitioners, who, as a group, are probably the least responsive to the need for organized continuing medical education. Private practitioners comprised 80% of the membership of the New York County District Branch but only 57% of the respondents; conversely, institutional and academic psychiatrists comprised only 20% of the district branch membership but 43% of the respondents. Naftulin and associates (2) also reported the relative lack of interest and involvement of private practitioners in continuing medical education. Such lack of interest is surprising when one considers that academic and institutional psychiatrists work in atmospheres where utilization review and quality control are a fact of professional life; to the physician in private practice they loom as noxious threats on the horizon. Logic would suggest a larger response from the most threatened group, but this was not the case; one can only surmise that a combination of denial, avoidance, and professional style limited the response of the private practitioner.

Among the respondents the most noteworthy findings were that a substantial majority favored mandatory continuing education and even favored evidence of continuing education as a requirement for relicensure. The overwhelming repudiation of recertification suggests examination anxiety as its basis. As a group, the respondents paid high tribute to the value of continuing education, were willing to document it in order to be relicensed, but did not wish to be examined. That only 47.1% of the 22,205 members of the APA were Board-certified in 1976, in contrast to much higher percentages for other specialties, may be further evidence of examination anxiety (3). However, many psychiatrists in private practice question the relationship between continuing education programs, as well as certification, and the services they render patients; this argument may have merit but it may also be a rationalization for examination anxiety. Only 2% of the 1,062 psychiatrists responding to a survey in 1973 reported that they had ever been called upon to engage in a professional activity for which they felt ill-prepared (4).

Our attempt to correlate age, Board status, and academic affiliation with response revealed that younger members were more likely to support continuing medical education and recertification requirements; 25% of the YYY response group were 25–35 years of age; this same age group comprised only 13% of the NNN response group. Psychiatrists who were Board-certified or who had an academic appointment were more likely to favor documentation of continuing medical education activity as a requirement for relicensure.

As noted earlier, academic and institutional psychiatrists not only show more interest in continuing medical education and recertification (as indicated by their disproportionately high response rate to the questionnaire), they also are more likely to have educational experiences and quality control built into their work environment. Because the private practitioner seems relatively less interested, we feel that the organizational effort toward continuing medical education in psychiatry should have as its dominant thrust meeting the resistances and needs of private practitioners. As a group they do not tend to have substantive academic environments that provide ready access to educational experiences. In addition, there is evidence that this gap is not filled by more intensive use of journal and monograph material. Private practitioners read less than psychiatrists in academia, who in turn read less than psychiatrists in both academia and research (5). The report on recertification by the Group for the Advancement of Psychiatry discusses some of the resistances of psychiatrists to continuing medical education and recertification (6).

The findings of this study, as well as those of some of the other studies cited, suggest that for a variety of reasons the psychiatrist in private practice constitutes

the greatest reservoir or resistance to meeting any requirements, whether they be in continuing education or recertification, practice audit, or clinical competence exams. This places a greater burden on the APA district branches to develop solutions concerning continuing medical education, since they are the principal professional link between the private practitioner and the profession. Our study further suggests that an overwhelming majority of respondents were willing to vest responsibility for continuing medical education in the APA and the district branches. We must translate this vote of confidence into meaningful educational opportunities for the private practitioner. Almost every psychiatrist's fantasy includes his personal responsibility to stay abreast of his field. Some do it on their own; most do not.

The pathologist is the natural monitor of the surgeon; psychiatrists have only their conscience as their monitor. The APA, through the district branches, must assume this responsibility. In so doing, study and attention must be given to how psychiatrists learn. For example, continuing discussion groups and clinical demonstrations may be more useful than formal, didactic course work. It is important that we do not settle for what is easy to teach but do what is useful and meaningful. Cognitive knowledge and clinical skills are both important, but are not necessarily related. There must be further study of what constitutes meaningful continuing education for psychiatrists.

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Temporal Disorganization and Inner-Outer Confusion in Acute Mental Illness

BY FREDERICK T. MELGES, M.D., AND ARTHUR M. FREEMAN III, M.D.

The authors found that changes in temporal disorganization covaried substantially and significantly with changes in Schneider's first-rank symptoms in 58 acutely ill psychiatric patients. Schneiderian symptoms reflect a confusion of inner and outer events; therefore, the findings suggest that difficulties in indexing events in time are closely related to problems in distinguishing experiences that arise within the self from outer experiences.

TEMPORAL DISORGANIZATION refers to alterations in the rate, sequential ordering, and goal-directness of thinking processes (1–5). A person can become temporally disorganized without being disoriented to clock and calendar time. Examples of temporal disorganization include racing of thoughts, difficulty keeping track of sequences, and indistinction as well as interpenetration of past, present, and future. These processes have been observed in patients with "functional" mental illness (6–9), but their possible relationship to symptom formation has been largely unexplored. In this paper, we will report a study of temporal disorganization in acutely ill psychiatric patients who were not disoriented to clock time and who did not have any other classical sign of coarse organic brain disease.

The focus of this study was on the relationship between temporal disorganization and symptoms of confusion of inner and outer events. To represent symptoms of inner-outer confusion, we chose Schneider's first-rank symptoms, such as experiences of alien influence, thought diffusion, and auditory hallucinations (10). Although Schneider originally proposed them as being "first rank" for the diagnosis of schizophrenia in the absence of organic brain disease, these symptoms of inner-outer confusion have been found in other illnesses, such as acute mania (11–13). We were interested not in diagnostic categories but in processes, i.e., the relationship of changes over time in temporal disorganization and symptoms of inner-outer confusion. A finding that such processes change together through time would suggest treatment strategies, since altering one of the processes might then influence the other (14–16).

The hypothesis was that in a sample of acute psychiatric patients (excluding organic brain syndromes) changes in temporal disorganization are correlated positively with changes in symptoms of inner-outer confusion. The rationale for this hypothesis has been formulated in detail elsewhere (1–6), but the essential consideration is as follows: If inner events such as memories, expectations, and emotions are no longer temporally indexed as linear sequences embedded within the continuum of inner experiences, then such inner events could conceivably be experienced as current phenomena that come from outside the person, much like present-time outer perceptions. Thus, the question we asked was: Are experiences lost in time also dislocated in space?

METHOD

Testing was performed by research assistants blind to the hypothesis who evaluated consecutive admissions to the psychiatric inpatient units of the Santa Clara Valley Medical Center on the day of admission after explaining the procedures and obtaining informed consent. Patients answered questions about orientation to time, place, and person and performed calculations; they were also questioned about illicit drug use and alcohol abuse for at least three days before admission. Those with organic brain disease or a recent history of drug abuse were excluded.

After the screening procedures, the research assistants tested 58 patients on the day of admission and again at the time of discharge (or transfer to another hospital) in order to obtain change data. The mean age of the sample, which was evenly divided as to sex, was 28.9 years (SD=11.8) and mean education was 14.1 years (range 7–22). Other descriptive variables are listed in table 1. The diagnostic breakdown was as follows: schizophrenia, N=41; mania, N=5, other psychoses (e.g., paranoid state), N=7; and neurotic-character and adjustment reactions, N=5. These diagnoses

When this research was done, Dr. Melges was Chairman and Dr. Freeman was Chief of Unit II, Department of Psychiatry, Santa Clara Valley Medical Center, San Jose, Calif. The authors were also on the faculty of the Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Stanford, Calif. Dr. Melges is now Professor of Psychiatry, Duke University Medical Center, Box 3812, Durham, N.C. 27710, and Dr. Freeman is Professor of Psychiatry, University of Alabama Medical Center at Birmingham.

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TABLE 1
Descriptive Data for 58 Patients

Variable	Number	Percent
Marital status		
Married	19	32.7
Divorced	14	24.1
Single	25	43.1
Occupation		
Unemployed	3	5.2
Semiskilled	27	46.6
Skilled	25	43.1
Professional	3	5.2
Previous hospitalizations		
None	17	29.3
1 to 2	26	44.8
3 to 5	13	22.4
5 or more	2	3.5

were made independently of the researchers by the ward psychiatrists according to the broad *DSM-II* categories listed above. There was diagnostic concurrence between the primary ward psychiatrist and an independent assessment by a psychiatric resident or another ward psychiatrist in 91% of the patients. Before the present illness, 77.6% of the patients had been regularly employed or going to school. At the time of admission, all patients were acutely ill, i.e., they required admission to a locked facility for emergency management. The average hospital stay was only 5.8 days (SD=3.0), which reflects the acute treatment and triage functions performed by this unit for highly disturbed patients in Santa Clara County. At the time of admission testing, all of the patients were acutely ill and 25% had received initial doses of psychotropic medication. By the time of second testing, 88% of the patients were receiving antipsychotic medications, usually in moderate doses (e.g., 20–30 mg/day of fluphenazine).

The tests consisted of inventory statements taken from previous research and refined in pilot studies with structured interviews to make them as clear as possible for acutely disturbed patients. The inventories were presented in random order, and the statements were read aloud by the research assistant with the patient looking on and also reading. Patients were carefully instructed and reminded to reply in terms of their experiences on the day of testing on the following scale: not at all, occasionally (1–2 times per hour), often (5–10 times per hour), frequently (more than 20 times per hour). Patients' understanding of this scale and of the inventory statements appeared to be adequate, since ratings by the research assistants indicated all patients were at least "reasonably reliable" except 2, who were of "questionable" reliability. In the lists of items given below, the last item in each component except number 5 for temporal disorganization is phrased in the direction of normality (and scored in the opposite direction) to help avoid response sets.

Temporal disorganization was measured by a total of 20 statements, derived largely from previous research (1–6), with 4 statements in each subcomponent.

1. *Rate-duration changes*: My mind seems to be racing. My mind seems to be going slowly. I'm unsure how much clock time has gone by (unless I look at a clock). My mind seems to be going at its usual rate.

2. *Tracking difficulties*: I tend to lose my train of thought. I forget what I've just said or intend to say. My mind goes blank at times. I can keep track of what I'm thinking about.

3. *Temporal indistinction*: My past and future seem to have collapsed into the present, and it is difficult for me to tell them apart. My past, present, and future seem all muddled up and mixed together. Sometimes I feel absent from the present, swept into the past or future as if I were really there. It's easy for me to tell whether something is a memory, a perception, or an expectation.

4. *Impaired goal-directedness:* My sense of self-direction seems to be impaired. I lose control over my thinking. It's hard for me to direct my thoughts to what I intend to think or say. My thoughts and actions are organized toward what I want to do or say next.

5. *Desynchronization*: My mind switches between speeding up and slowing down. I am having two or more trains of thought at the same time. Some of my experiences seem to have happened before in the exact same way. My mind swings back and forth in opposite directions.

The symptoms of inner-outer confusion were derived from Schneider's first-rank symptoms of schizophrenia and refined from other studies (10–13), with 4 statements in each of the subcomponents below.

1. *Auditory hallucinations*: I hear my inner thoughts spoken aloud. I hear voices outside my head arguing or talking about me. Voices comment on my actions at the time that I do them. All the voices that I hear come from myself or persons I can see talking.

2. *Experiences of influence*: I am made to feel emotions that are not my own. Outside forces put impulses into me. Forces other than myself make me do or say things that I don't intend. I myself am in control of what I do, think, and feel.

3. *Thought diffusion*: Thoughts that are not my own are put into my mind. My inner thoughts are broadcast to other people even when I don't speak aloud. Some outside force or person takes my thoughts away. My inner thoughts are contained within my head and nobody can read my mind.

4. *Delusional perception*:[1] Something strange is going on in the world around me. Unusual coincidences are happening to me that seem to have special mean-

[1] Careful reading of Schneider (10) suggested that uncanny coincidences of outside events referred to the self were central to his phenomenological description of delusional perception.

TABLE 2
Correlations Between Changes in Temporal Disorganization and Symptoms of Inner-Outer Confusion*

	Total Temporal Disorganization	Rate-Duration	Tracking	Temporal Indistinction	Impaired Goal-Directedness	Desynchronization
Total Inner-Outer Confusion	.73	.53	.57	.52	.57	.70
Auditory hallucinations	.54	.51	.44	.28	.35	.58
Influence feelings	.62	.35	.51	.45	.52	.62
Thought diffusion	.58	.35	.43	.54	.49	.50
Delusional perceptions	.55	.44	.42	.35	.42	.57
Somatic passivity	.63	.49	.49	.46	.51	.55

*Correlation coefficients greater than .25 are significant at the .05 level, those greater than .35 at the .01 level, and those greater than .44 at the .001 level (df=56).

ings for me and my future. Something I see or hear suddenly takes on a profound significance for me and my future. The coincidences that happen to me are just ordinary chance happenings, with no special meaning for me or my future.

5. *Somatic passivity experiences*: Somebody or something touches me even when nobody is around. Unusual sensations are put into my body. My body is controlled by outside forces. My bodily movements seem normally under my control.

Although most of the Schneiderian symptoms listed above can be conceptualized as inner-outer confusion, some of Schneider's symptom descriptions (e.g., thought-echo and delusional perception) may not be readily interpreted in this way. For the purposes of this research, compared to constructs such as diffusion of ego or body boundaries these Schneiderian symptoms investigated questions relevant to inner-outer confusion that were specific enough to be measured by scaled subjective report methods.

The total temporal disorganization score and the total inner-outer confusion score consisted of the sum of their respective subcomponents, with higher scores indicating greater psychopathology. For the data analysis, each subject was used as his own control to obtain difference scores between the first and second testing results in each test category, and then Pearson product-moment correlation coefficients between the changes were computed.

RESULTS

The hypothesis that changes in the degree of temporal disorganization are correlated positively with changes in the degree of symptoms of inner-outer confusion was confirmed (see table 2). The change correlation between the total temporal disorganization and the total inner-outer confusion score was .73 ($p<.0001$). The change correlations between the subcomponents of temporal disorganization and of inner-outer confusion were all significant at least at the .05 level; most were significant at the .001 level.

DISCUSSION

The finding of substantial, significant positive correlations between changes in temporal disorganization and concomitant changes in symptoms of inner-outer confusion suggests that these two processes are closely linked with one another through time. Just how these are interrelated is open to interpretation and further inquiry.

The finding of covarying changes over time in a clinical investigation such as this does not necessarily imply that there is a causal or mutually influential interaction between the variables. To study the relationship further, manipulation experiments are necessary to test whether inducing or treating one process will bring about changes in the other. We have already studied the induction of temporal disorganization in normal subjects through the use of THC. One of the earliest and most distinctive effects of THC is the alteration of time sense (17). Low doses of THC have been found to produce mild to moderate temporal disorganization but few, if any, symptoms of inner-outer confusion, whereas high doses of THC produced marked temporal disorganization and definite symptoms of inner-outer confusion (1, 3, 5). None of these subjects was globally disorganized or delirious in terms of being disoriented to clock or calendar time, place, or person. Another type of disorganization induced by comparable doses of alcohol in the same subjects was not associated with marked temporal disorganization nor with symptoms of inner-outer confusion.

In the THC experiments, temporal disorganization preceded the emergence of symptoms of inner-outer confusion; thereafter, the two processes covaried. In longitudinal clinical studies (1, 9), temporal disorganization (e.g., racing of thoughts, confusion of sequences, and timelessness) has been found to occur prior to the onset of symptoms of inner-outer confusion.

The most useful experimental design for studying this relationship may be to find some way of specifically treating temporal disorganization in order to test whether symptoms of inner-outer confusion might be

prevented or diminished by such a treatment. Whether such a treatment strategy would be at the social, psychological, or neurochemical level, or a combination of these, deserves exploration. Because temporal disorganization has been found to be related to problems with immediate or "working" memory (4, 5), it would be interesting to see whether treatments aimed specifically at enhancing immediate memory would correct temporal disorganization. If so, one could also test to see whether symptoms of inner-outer confusion could be prevented or diminished by this approach.

An enhancement of immediate memory might help restore the patient to what Minkowski (18) termed the "I-here-now" position so that he/she can distinguish the *now* from the *then*. This may prevent the patient from being engulfed by his/her own inner memories, expectations, and imaginations as if they were telescoped into the present, and responding to them as if they came from outside the self, much like present-time outer perceptions. Psychotherapeutic strategies also could be developed to help patients differentiate the present from the past or future. To quote Arieti (19), "we must try to reenlarge the patient's psychotemporal field. . . . Whereas the world of psychosis has only one temporal dimension—the present—the world of reality has three: past, present, and future." By helping patients to distinguish and reorder their experiences as past, present, and future, their experiences of inner-outer confusion may also be corrected.

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Life Planning: An Educational Approach to Change

BY HUGH JAMES LURIE, M.D.

Life-planning methods that have emerged over the past several years offer alternatives to psychotherapy for the person experiencing the crises of mid-life and mid-career. The author describes several approaches to life planning and presents details regarding the implementation of life-planning methods.

> The daily pressures to act, to do, to decide make it difficult to stop and think, to consider, and to examine one's life goals, one's directions, one's priorities—the basic choices one faces in managing his own world.
>
> —Roy Menninger (1, p. 16)

NEW AND intriguing methodologies have emerged over the past several years which offer alternatives to the traditional therapeutic measures that have been used to help people cope with the stresses of mid-life. These stresses include a sense that one is on a treadmill in a career, that family and friends are growing stale, and that life is closing in.

Life-planning methods combine aspects of several disciplines, including education, educational psychology, and ego psychology. This paper will focus on life-planning formats that have been derived from these disciplines. In addition, I will discuss implementation strategies for life planning and the relevance of life-planning exercises for medical and other professional groups.

LIFE-PLANNING METHODOLOGIES

Management by Objective

Educational psychology has exerted a strong influence on organizational planning and development in recent years. This influence often takes the form of "management by objective" (MBO). MBO methods were taken from business administration theory, applied to the analysis of current organizational realities, and ultimately directed toward planning for short- and long-term goals. Some life-planning centers have translated these methods into a format that is "objective" and relevant to assessment and analysis of an individual's present life situation and establishment of new goals for the future. Although such methods were originally used primarily with women contemplating employment after years away from the labor market, they have been increasingly used with other groups, including physicians.

One life-planning center, the Individual Development Center of Seattle, uses a variety of exercises to illuminate different aspects of the individual's current life situation.[1] These exercises include detailed analyses in the areas listed below.

1. Influences that have shaped a person's self-image, including experiences in grade school, high school, and college; family and other interpersonal influences; introversion and extroversion; career; fantasies; and previous experiences in seeking employment.
2. A systematic analysis of work the participant has done (including volunteer work and hobbies) and work skills, specialized knowledge, natural abilities, and specific tasks accomplished.
3. Current life situation, involving assessment of activities that decrease or increase anxiety, destroy or build self-esteem, produce pleasure or pain, exhaust or energize, and narrow or expand horizons, as well as an analysis of values.
4. Current time expenditures, past goals, finances, friendships, health and energy, and potential or contemplated new goals.
5. Long- and short-term goals and an analysis of what they would mean for the participant and his/her family, economic situation, self-concept, and lifestyle.

A large amount of information is assembled through these exercises to use as the basis from which an individual can begin to change his/her life, e.g., career, time allocations, friendships, and finances. Each exercise is designed to be used in classes that are held weekly for six weeks; after the series, there is an individual counseling session.

"Students" are expected to complete weekly "homework assignments" that are labeled as such rather than as "therapeutic goals." Participants are asked to analyze in writing the issues assigned to them for each session; these responses are used as the basis for discussion, sharing, and peer consultation in small groups at the weekly sessions.

Revised version of a paper presented at the 129th annual meeting of the American Psychiatric Association, Miami Beach, Fla., May 10–14, 1976.

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[1]The center is located at 1020 East John St., Seattle, Wash. 98102; complete exercises and further information may be obtained from Ms. Alena H. Moris, Director.

A Personal Time Study

Roy Menninger has remarked that

> the important things in our lives are frequently deferred with some comforting but self-deceiving assumption that there will always be time tomorrow. . . . there is a high probability that your use of time and energy reflects serious imbalance within the life-space of each of you. . . . Time for one's self is discouraged, pleasure is deemed to be selfish, and one's own needs come last. (1, pp. 16, 17)

The need to allocate time and determine priorities—especially time for oneself—is crucial to successful life planning. This problem has been analyzed by different observers who (predictably) propose different solutions: Menninger (1) suggests that time allocation should be delegated to other people. An article in a popular magazine (2) suggests that individuals who have efficient personal filing systems and are organized will save time. The Individual Development Center believes that wasted and unproductive time often reflects failure to analyze and establish time priorities; this center has developed an exercise to facilitate establishment of time priorities in which the participant is asked to calculate the amount of time spent each week on a variety of activities, including sleeping, eating, paid work, volunteer work, driving and commuting, child care, family interaction, food preparation, entertainment, physical activities, education, and hobbies. Participants are then asked, "A week has 168 hours. What have you learned about your time scheduling?" Clearly, unless one gets down to specifics, talk about saving time is nonsense.

Values Clarification Techniques

Values clarification, a technique originally designed for teachers and their students (3), is a useful adjunct to life planning. One of the assumptions behind values clarification is that many people are unaware of their values and choices until they are forced to rank them in importance. "Practical people" like physicians often prefer to grapple with life planning by sorting out priorities in their favorite activities rather than struggling with more abstract concepts such as fantasies and roles.

Although values clarification exercises can be done on an individual basis, they appear to be particularly suited to groups in which a person can first assess his/her own values and then discuss them with a group of peers. I have used various values clarification techniques with groups of physicians, including psychiatrists, and I have been impressed with the degree of self-scrutiny and reassessment they often encourage within a brief period of time. Clarifying values is quite different from clarifying goals; many individuals can function adequately on a day-to-day basis despite being out of touch with their basic values. Values clarification is particularly useful before life changes such as retirement. The physician who is anticipating retirement in a Florida golfers' paradise may discover that he will seriously miss his friends, neighbors, and family, and that the allure of golf under sunny skies does not balance out the contemplated losses.

Projective Techniques

Life-planning exercises available from the National Training Laboratories of Applied Behavioral Sciences, Arlington, Va., include a variety of projective techniques designed to be used on an individual basis; results are then compared with those of peers. For example, in the "life on a line," the person is asked to draw the pattern of his/her life as if it were a fever chart. The person is then asked to label the highs and lows and to draw a line anticipating the pattern of the future. This exercise is particularly useful in identifying concerns and dissatisfactions with the current life situation (as well as the specific ingredients of "better times"). In another exercise, the individual is asked to write an epitaph for him/herself and then determine, with small-group consultation, how his/her current life situation supports or refutes these ultimate wishes.

Role and Goal Priorities

Some life-planning exercises, including those of the National Training Laboratories, ask the participant in a workshop to identify and rank his attributes and roles. One exercise is, "Think of all the answers you can to the question, 'Who am I?' " There should be at least 20 answers; often, there are more. The answers might include one's role in life, appearance, or one's beliefs and feelings. The individual is then asked to arrange the list in order of importance and to discuss his/her top choices.

In another exercise, the individual is asked to rank a list of 11 goals or life values in terms of the present, as well as what he/she would like the future to contain. Some of the items on the list are leadership, prestige, wealth, independence, security, and duty. The lists are then discussed in a group format. This exercise offers a more structured method of goal assessment than the "Who am I?" exercise.

Video Stimulus Materials

Middle-age crises seem remote and the need for life-planning seems nonexistent to some professional groups. This is particularly true for young medical students who face immediate, pressing goals such as graduate education, exams, and postdoctoral training. One effective way to stimulate medical students' recognition of the need for life planning is to show provocative videotaped vignettes that portray (with appropriate affect and anguish) physicians who are experiencing mid- or late-life crises (overwork, depression, disillusionment, marital conflict). This "visual assault" seems to be one of the only ways in which to capture the interest of such a difficult and well-defended group before a crisis actually occurs. Although many younger physicians today are obviously capable of elaborating comprehensive life goals, a number of physicians

of all ages become locked into narrow and limited goals that look more toward material acquisition than toward emotional needs and satisfactions. Videotaped vignettes, produced by the author, have been used successfully with students in a number of different medical schools as a basis for discussion and a prelude to life planning. These vignettes have also been used with a group of psychiatrists at an annual meeting of an American Psychiatric Association district branch to stimulate discussion and personal introspection about the life-styles of psychiatrists, which often include isolation, depression, and psychological morbidity. I have also used them productively for life-planning discussions at an APA colloquium on postgraduate education and at several national conferences focusing on practicing family physicians.[2]

Organization and Implementation of Life Planning

Life planning can be like New Year's resolutions: without a specific scheme for their execution, plans and dreams may just disappear. Methods of implementing life planning range from those which produce little anxiety to those designed to stimulate the individual to take immediate action. Several possible approaches are listed below.

Individual Counseling

By definition, individual counseling for life planning involves a meeting between a client contemplating a change in career or life-style and a skilled, informed counselor who can assist in discussing the options. Advantages include convenience and privacy; many individuals may be reticent about discussing fantasies and plans for change with peers for fear of ridicule or even retribution. Major disadvantages are the lack of peer feedback and support and the overreliance on the "expert" rather than the opinions of colleagues.

A felicitous compromise can exist when individual counseling for life change is an adjunct to a larger mental health training program. The Menninger Foundation training program on personality development often included individual elective life-planning sessions. Many of the individuals attending these programs, most of which were directed by Dr. Herbert Klemme (4), already had a high level of sophistication and understanding about the possible needs for change in their careers or life-styles (participants included physicians, corporation executives, and city managers). The life-planning counselor—in the context of a cognitive and affective learning experience about personality development throughout life—was able to help the participant identify and formulate his/her own life and career goals and changes and provide a helpful push toward implementation.

[2]The vignettes, titled "Human Experiences of the Practicing Physician" are available through the Mental Health Media Project, Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine, Seattle, Wash, 98195.

The Educational Course Approach

A group program with a classroom and course format may be seen as less threatening and more familiar to many individuals than would an experience with overtones of a therapy group. The unique advantages of life planning conducted within an educational format include the structure of cognitive experiences and exercises, the support of fellow students, the period of time between assignments, and the greater likelihood that the participant will carry out his/her plans because they have been put in writing with peers as witnesses.

In any counseling format, the skills of the leader in organizing the group and conducting exercises to encourage individuals to share their concerns and hopes is critical to success, as is the model of openness the leader presents. The teacher may use a variety of "games," such as three-person group consultations, collage techniques (cutting out magazine pictures describing different aspects of one's life and showing them to the group), or cognitive methods. The educational course approach encourages a climate of self-inspection, cognitive understanding, and structured fantasy and can provide the best entry into life planning for many individuals. The setting can be either a classroom (with comfortable seating) or an atmosphere that provides more flexibility in seating to accommodate the ebb and flow of small and large groups and the need for each group or individual to be separate at times.

Therapy and Support Groups

Therapy groups rarely include specific goals for life planning, but some support groups do include an opportunity for individuals to discuss and receive feedback from other members about current life and career goals and problems and their evolution. Some therapy groups evolve into life-planning and reassessment groups; in others, this aspect is part of the original design. Such groups can be included in professional meetings. The Network for Continuing Health Educators (a group originally organized under APA auspices) includes support and life reassessment activities as part of its semiannual training workshops. Another small group of mental health educators who met initially with a highly skilled leader to assess how they were managing their mid-life "transitions" now meets from time to time on a leaderless basis to ask the same questions and to give each other support and feedback.

There is always the danger that the group will become coercive or that there will be an expectation of change in an individual member that he or/she may not be prepared to make. Despite these potential difficulties, when a group of individuals is willing to invest in the undertaking, the creation and continuation of a support group to mediate career and life-style change is feasible. A relaxed setting, away from the distractions of the city and career and conducive to self-revelation, listening, and planning, is optimal for

groups meeting for several days of life-planning activities.

One-Day Consciousness-Raising Sessions

One-day workshops can often be helpful for professionals who are in a rut but are not sure what the problem is. Workshops can help the participants to crystallize issues and can raise affect and mobilize curiosity and interest in the process of problem solving and self-scrutiny. A particularly successful workshop, entitled "What You Have Always Wanted to Know About Doctoring But Were Afraid To Ask," was held in western Pennsylvania several years ago for physicians and their families. The workshop, guided by a cadre of skilled group leaders, moved physicians and their spouses and children, separately and with other family members, through a series of rap group experiences. There was a great deal of intense discussion by the physicians and their families about the hazards of medical life and of being part of a doctor's family: the isolation, the harassment, the lack of opportunity for recreation, and the progressive alienation of spouses and children from the excessively busy doctor. Wives often confessed that this was the first time they had ever really "let their hair down" and acknowledged their pain and frustration. As a result of this experience, several families made a commitment to examine their life-styles and to implement specific changes in current activities and future goals.

Atmosphere is a crucial factor in the one-day consciousness-raising session. The setting must be comfortable and relaxed, and recreation should be available either before or after the conference. Although they resemble encounter groups in some ways, such workshops raise more focused issues; thus, the affect that is mobilized is less frightening to participants than an experience without explicit expectations, time limitations, and agenda. On occasion, consciousness-raising sessions have led women's auxiliaries of medical societies to have more intensive life-planning experiences for themselves and their spouses. The desired outcome is for the participants to take more responsibility for acknowledging, sharing, and using their perceptions of their own needs rather than simply submitting to the desires of the "professional" in the family.

Life Planning for Couples

Studies suggest that half of physicians have been in "bad" marriages (5). An interview in the *Hospital Tribune* in 1974 with several knowledgeable psychiatrists—Drs. James Evans, Herbert Modlin, and James Karding—suggested that doctors' wives often feel second rate and neglected by their busy husbands and believe that most of the important marital decisions are made by the "important," i.e., professional, spouse with little reference to the needs of the "less important" spouse. The advantage of involving couples in life planning is that it may provide a means to preserve relationships and marriages while allowing both members of the couple to change and grow. A couples group can be an extended experience (e.g., weekly meetings) or an intensive experience for a weekend or week. Couples workshops can use the traditional format of a therapy or encounter group or include more cognitive or structured experiential exercises. The motivation for attendance at such a group is often a recognition that the marriage is floundering or intimacy is dying. The group may focus on ways to enrich the marriage or increase intimacy between the partners. As is the case in other group experiences, there is a danger that "pseudo-intimacy" may result, and that a "group high" may be seen as a substitute for working out the basic problems in the relationship.

Life-Style Experimentation

Some individuals discover their goals after they try them out. This may include experimentation with different ways of relating, with various roles, and with new styles of professional practice. Opportunities to experiment with a new life-style are available through intensive and often rather lengthy human growth experiences such as those provided by some of the summer programs at the National Training Laboratories of Applied Behavioral Sciences at Bethel, Me., or at growth centers such as Esalen in Big Sur, Calif. Some participants, during the experience, can adopt new identities, new ways of relating, and even new names.

There are several means of career experimentation. One method is the sabbatical: for a physician this works best if he/she is associated with an institution such as a health maintenance organization or a prepaid health plan. For example, one physician changed from a primary care role to teaching at a local medical school for six months. At the conclusion of this trial career change, he was able to assess whether the new job provided enough compensation, e.g., less pressure and more time with his family, to compensate for the decrease in income. Another method involves a new commitment to an intensive volunteer or consultative experience for a specified period of time, following which the individual can evaluate the satisfactions and drawbacks of the new career and decide whether it should be pursued in earnest. It is often easier for an individual to reach conclusions about implementing change if he/she can discuss the meaning of the experience with someone else and develop a cognitive understanding rather than simply mulling it over without feedback.

CONCLUSIONS

Life planning is available in a variety of formats, ranging from classes to individual counseling and from highly structured exercises to projective experiences.[3] Life-planning activities may be tailored to the individ-

[3]A national organization of life-planning facilities is Catalyst, 14 East 60th St., New York, N.Y. 10021.

ual needs of the person, to his/her particular learning style, to the time available, and to his/her goals (e.g., career change or reestablishing marital intimacy). Many factors about the ways in which individuals grow and change are still unknown; Sheehy's book (6) about individuals experiencing life change is a beginning attempt to describe some of these variables. The person's profession may be relevant to these factors. Does a family physician who often has to compartmentalize his day into brief segments learn best through a life-planning experience that is similarly compartmentalized (7)? Does the architect who often must mull over a project and ideas for weeks at a time learn best if his/her life-planning experience is correspondingly deliberate, reflective, and gradual? Despite our lack of knowledge, one thing is clear: life planning for many people caught in a developmental crisis offers an alternative to psychotherapy and can be a useful tool for helping the individual who does not wish to define himself as "sick" to get better.

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Evaluation of a Psychiatric Clerkship By Videotape

BY ARTHUR T. MEYERSON, M.D., ALAN WACHTEL, M.D., AND JOHN THORNTON, PH.D.

The authors used a videotape-based objective examination in general psychiatry to assess the clinical competence of medical students who had completed their third-year clerkship. The test has been shown to be a reliable, valid measure of clinical competence. Subjective faculty grading related positively to test scores. Students on one ward had significantly lower overall test scores, with particularly low scores on clinical judgment questions. However, faculty grades for these students were not significantly different. This suggests that the faculty was unable to assess the relative efficacy of its own teaching. The test can provide objective assessment of student clinical competence and is a means of program evaluation.

THE NEED FOR standardized, objective measures of clinical competence in psychiatry has been well documented (1–3). Such measures are necessary to objectively evaluate clinical competence of students and to assess the efficacy of student training programs.

The use of audio and visual aids to examine the clinical psychiatric competence of medical students gained prominence in 1958 and 1960 with the work of Gurtsma and Stoller (4, 5), who used filmed interviews as a clinical stimulant for a series of written, scaled descriptions of the patient's behavior and psychodynamics. In 1961 Saltzman and Goldstein (6) used an audiotaped patient interview and had students rate 303 psychiatrically relevant statements on a 7-point scale. The subjects (faculty, residents, interns, and four classes of medical students) demonstrated the same correlation of performance with level of training as previously reported by Gurtsma and Stoller (5).

Goldstein and Saltzman (7) concluded that "performance on clinical judgment tasks was significantly related to [the] student's overall academic performance, but showed no relationship . . . to rating of clinical competence made by his supervisors . . ." Therefore, they concluded that "one cannot accurately predict the student's actual clinical performance on the basis of his scores in the clinical judgment test."

Using written case material, Langsley (8) in 1962 compared performance of second-year medical students from four different schools on a multiple choice examination.

Monroe (9) in 1965 used filmed interviews with a multiple choice examination, dividing his test into three categories: psychotherapy, clinical evaluation, and knowledge. He divided clinical clerk performance into upper, middle, and lower thirds and "in a rough comparison, it was reassuring to find that in only 10 of 85 students was there marked disparity between clinical performance on the second-year examination and their performance during the third-year clerkship."

Turnblad and associates (3) developed an examination using a videotaped psychiatric interview followed by a series of statements designed to measure "a combination of observational skills, understanding of psychiatric knowledge and theory, and (on some items) a degree of empathic understanding." In four classes of medical students Turnblad and associates found a significant change between the preclinical medical students, freshmen, and sophomores but found no change when they compared the sophomores (i.e., before clinical experience) with juniors and seniors who had completed clinical clerkships. Although they had planned to develop an examination "to simulate a clinical task and measure clinical skills," Turnblad and associates (3) concluded that they had found "a useful instrument for evaluating the knowledge and observational skills of preclinical teaching."

Miller and Tuppin (10) used a video-based examination to evaluate performance of students taking the SAID (Systems Analysis Index for Diagnosis) program. This was an evaluation of a preclinical course and involved pre- and posttesting. Thus, program evaluation, as well as student evaluation, has been a goal of this developing field.

Recent work by Dowaliby and Andrew (11) in 1976 showed a positive correlation between National Board Examination performance and faculty evaluation of clinical competence. They suggested that, on the one hand, "faculty members are able to make discrete judgments about students on more than one dimension" and that, conversely, an objective exam can correlate with faculty clinical performance rating. The National Board of Medical Examiners Examination, Part II, uses no audiovisual aids.

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The authors wish to thank James Ryan, M.D., Department of Psychiatry, Columbia University College of Physicians and Surgeons, for the use of some tape material and questions.

The validity of these instruments is frequently established by faculty agreement on correct answers to the test questions. Test reliability has been largely unexplored. Langsley (8) did use a split-test reliability to assess his instrument.

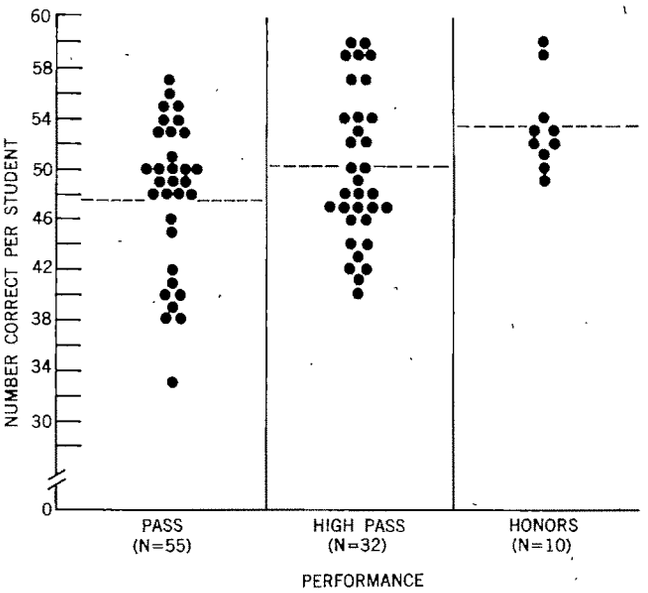
This paper focuses on the nature of the study examination, its reliability and validity, a comparison of the test results with the subjective evaluation of the clinical clerks by the faculty of the department of psychiatry, and the use of test results for program evaluation.

METHOD

A videotape-based test of clinical competence was given to 77 third-year medical students at the end of each of their 6-week clerkships in psychiatry. The test consists of four major taped student-patient interviews and four clinical vignettes. The patients interviewed suffered from the following illnesses (diagnoses from *DSM-II*): psychotic depressive reaction; schizophrenia, paranoid type; organic brain syndrome, acute—secondary to toxic ingestion; organic brain syndrome, chronic—secondary to senile dementia; and chronic alcoholism. The test consists of 73 short-answer, multiple choice questions. Among the several types of questions are true or false, best answer, and best combination of answers. Questions are directed toward three major areas: observation (e.g., Which of the following signs is displayed by this patient?); clinical judgment (e.g., Which of the following statements are correct in regard to criteria for this patient's hospitalization?); knowledge (e.g., Which of the following are side effects associated with phenothiazines?). Informed consent was obtained from all the patients who were interviewed. Students were informed that this test was experimental and were assured that the results would in no way influence their clerkship grade. They were asked not to discuss the procedure and content of the test with classmates who were to take it at a later date. No time limit was imposed and all students completed the test. Only research personnel were aware of the students' test grades and the content of the test.

The student's grade for the clerkship was derived from the unit chief, the unit chief resident, all inpatient and outpatient preceptors, and all course instructors, all of whom were required to globally rate several major items (knowledge, interest, participation, attitude, curiosity, etc.) and to fill out a comprehensive and detailed rating form of more than 30 items on a 5-point scale, including amount of reading, personality, ability to relate to staff and patients, etc. A meeting of all major teaching staff was held at the end of the clerkship and verbal reports were made and recorded. The director of the clerkship wrote a summary report and grade based on the aforementioned sources. A student was assigned one of four grades—honors, high pass, low or routine pass, and fail. One student failed during the study.

FIGURE 1
Relationship of Subjective Faculty Grades to Students' Test Scores*



*F=3.7; p<.05.

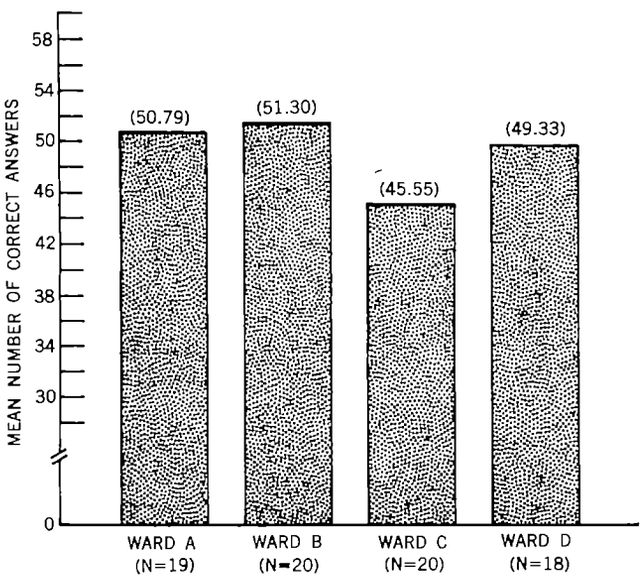
RESULTS

Test reliability as calculated by the Kuder Richardson 20 (KR-20) (12) on the results of 77 students was .6651. All other statistical analyses do not include 3 students who either failed (N=1) or received low or routine pass (N=2) for the clerkship. However, no reported results change significantly if these 3 are included. Figure 1 shows the relationship of subjective grades assigned by the faculty to test scores. Out of a possible 73 correct answers, the mean scores were 47.88 correct answers for the low-pass group, 50.16 for the high-pass group and 53.30 for the honors students. As shown, these results are significant ($p<.05$; Fisher exact probability=3.70).

Figure 2 displays the students' test scores by ward assignment (labeled A, B, C, and D)—19 students were assigned to ward A, 20 to B, 20 to C, and 18 to D. These assignments were totally random. Each clerkship group is listed alphabetically and then assigned a ward in rotation. Figure 2 demonstrates that the test performance of the ward C students was below that for the entire group of low-pass students ($F=3.89$, $p<.025$). This remains true even if the one student who failed is excluded. An examination of ward C students' performance on the three subsections of the test reveals that this group of students did not significantly differ from the others on knowledge or observation questions, although a trend appeared ($p<.1$). However, on clinical judgment items, ward C students differed significantly from the whole group ($p<.025$).

Table 1 represents an examination of the subjective faculty grades given by all four wards to their students. There is no significant difference between wards.

FIGURE 2
Analysis of Students' Test Scores by Ward Assignment*



*F=3.89; p<.025.

TABLE 1
Subjective Faculty Grades for Students on Four Wards

Grade	Ward A (N=19)	Ward B (N=20)	Ward C* (N=20)	Ward D (N=18)	Total (N=77)
Honors					
Expected**	2.47	2.60	2.60	2.37	
Given	1	4	2	3	10
High Pass					
Expected**	7.90	8.31	8.31	7.48	
Given	10	4	8	10	32
Pass					
Expected**	8.64	9.09	9.09	8.18	
Given	8	12	10	5	35

*Ward C, the ward with low test results, came closest to the expected grades (χ^2=7.90, n.s.).
**Obtained by taking 10/77 of students on ward for honors, 32/77 for high pass, and 35/77 for pass.

Ward C, in particular, came closest to the expected values (χ^2=7.90, n.s.).

DISCUSSION

The reliability of the test instrument is a significant issue in all studies such as this. Few references to formal assessment of reliability of test instruments of student clinical competence in psychiatry are available (8, 11). Dowaliby and Andrew (11) recently reported a study in which faculty grades were compared with test scores on the National Board Examination, Part II, after a training clerkship for paramedics. In that study the results of four of the six Board exam subsections correlated significantly with faculty evaluations of the students. The KR-20s of two of these four were .61 and .49. These are well below the level achieved by our test (KR-20=.6651). Test reliability is probably not as high as one would prefer for a test of objective knowledge. However, the KR-20 is a function of the number of questions and videotape, which reduces available time, is a limiting factor. The KR-20 provides an estimate of the reliability of a test. The equation has been derived in various ways by using sets of assumptions and different techniques (12–16). A basic assumption used to develop the KR-20 is the homogeneity of the function being measured by the test items. Because this assumption is intentionally not satisfied by the present test, with its focus on observation, knowledge, and clinical judgment, one cannot place too much emphasis on the apparent value obtained for the KR-20. The .6651 value may be viewed as a lower bound (lower limitation) of the true reliability of the instrument (16).

Validity, as stated before, is often equated with faculty agreement on an instrument's correct answers—"the test was also given to a group of faculty to test its validity" (3). Our instrument was submitted to several senior faculty members (not directly involved in the clerkship), and correct responses were verified. However, the use of faculty assessment of the correct answers provides no measure of the internal consistency of an examination or whether it measures what it was intended to measure. It merely provides a statement that the faculty agrees on the correct response to any one question.

The correlation of student test grades with faculty assessments ($p<.05$) strengthens the proposition that the current test is a valid reflection of the faculty's goals for the clerkship. A similar correlation was noted previously by Dowaliby and Andrew (11), although not with medical students. However, Goldstein and Saltzman (7) found no such correlation. Turnblad and associates (3) approached the question of the usefulness of an objective instrument independent of the clinical setting that used media and not real patients to measure medical student clinical competence. Turnblad's study did not attempt to correlate faculty assessment with test results. Our study does not directly compare students at various levels of experience. However, the instrument's capacity to distinguish student performance levels on questions of clinical judgment after different clerkship programs (the 4 wards) ($p<.025$) further strengthens its validity as a measure of clinical competence.

The notion that a test can substitute for some aspects of faculty evaluation, particularly of attitudes and ability to relate to patients and staff, is not the focus or conclusion of this study, nor is it a belief of ours. In this study direct observation of students offers invaluable insights. However, evaluation procedures involving direct observation of students can become cumbersome and may require using the same patient for many students to achieve standardized results,

multiple faculty observations, and a great deal of time; also, it would depend on questionable interrater reliability. This videotape-based test allows for distinctions in student performance on clinical judgment items. Thus, our data do not support Turnblad's critique of the capacity of videotape tests to assess clinical judgment based on the student role as a passive observer of a videotape as opposed to performance in the actual clinical setting.

The low test performance of the students on ward C ($p<.025$) is not reflected by any lowering of their faculty evaluations. Thus, the test permits a singularly objective assessment of faculty effectiveness regarding both training and student evaluation. This finding is further supported by ward C students' low performance on clinical judgment questions ($p<.025$) in contrast with the other teaching units. Thus, major support for the hypothesis that a test offers an opportunity not offered by subjective approaches for program and faculty assessment lies in the lack of a significant difference between ward C students' subjective faculty grades and faculty grades given students on the other wards. The fact that many schools train student clerks in a variety of hospitals, not just different wards in the same institution, suggests the possibility that variance in program success may be a virtual inevitability. Thus, testing as in this study seems clearly indicated.

CONCLUSIONS

Our use of the videotape-based test continues the efforts of others toward establishing a statistically reliable clinical examination in psychiatry. The use of analysis of variance and a standard test of reliability (KR-20) permitted a comparison of our findings with those of others, which has proven difficult in this area in the past. In addition, the instrument fulfills Turnblad's goals: "it is important that our assessment methods be reliable, that they validly measure desired objectives, and that they be efficient in the sense that they can be applied to large groups in a limited period of time" (3). The increasing pressure for objective evaluation of teaching programs and certification of professional competence renders further efforts in this direction essential.

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In Defense of Community Mental Health

BY JACK ZUSMAN, M.D., AND H. RICHARD LAMB, M.D.

Initial enthusiasm for community mental health has given way to a barrage of criticism, much of it deserved. Originated to deal with abysmal conditions in state hospitals, community mental health focused instead on primary prevention, political activism, and the mildly mentally ill. The authors propose a return to the original fundamentals, stressing that the highest priority be given to community treatment and rehabilitation of the severely mentally ill.

THE ENTHUSIASM OF THE 1960s about community mental health has given way to a barrage of criticism from many different quarters, particularly from senior academicians (1–7). Further, there have been severe cutbacks in funding of training and services, especially at the federal level. Although the Community Mental Health Center Amendments of 1975 expanded the scope of community mental health centers and the services they should provide, this legislation has not been accompanied by any significant increase in funding.

Disillusionment is evident, even among former enthusiasts. Few among the leadership of the mental health professions are now inclined to defend, in whole or in part, the programs and theory embraced so enthusiastically just a few years ago.

Many criticisms of the community mental health movement have been well deserved, particularly those related to the failure to serve the many thousands of severely disabled long-term psychiatric patients who have been discharged from state hospitals (8–10). It has become increasingly apparent that society has chosen the easy and cheap way out: instead of providing these patients with the community treatment and rehabilitation they need, they have been placed in community settings where they live impoverished lives that resemble their experiences in the state hospitals. Community mental health has also been accused of pursuing its own interests, namely, meeting the financial, prestige, and power needs of the professionals in the field, while allowing the seriously ill patients to whom it promised so much to suffer neglect, poverty, and disability. Only recently have psychiatry and community mental health leaders begun to express concern about the problems attendant on deinstitutionalization.

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There has been much said and written by those working in community mental health about the value and potential of prevention. It was often implied (and sometimes promised) that the techniques of prevention would result in a significant reduction of mental illness and would eventually drastically reduce the need for conventional treatment because there would be so few patients (11, 12). Although preventive efforts in the form of consultation to community agencies such as schools, welfare departments, the police, and other caretakers have been popular and probably useful, the promised eradication of mental illness and/or social problems has not been forthcoming. Elaine Cumming (1) pointed out that the effectiveness of popular strategies for the primary prevention of the major mental illnesses has not been tested and that these strategies remove needed manpower from the treatment of the mentally ill. The overselling of prevention has probably been a major factor in causing people to become disillusioned with community mental health.

Still others (2, 3) have criticized community mental health for turning away from psychodynamics, psychotherapy, and meaningful interactions with patients. Although such criticism overlooks some of the basic origins, strategies, and components of community mental health, it is true that the importance of individual psychodynamics is frequently neglected in community mental health. For instance, aftercare programs consisting primarily of medication and a minimally stimulating environment are often provided routinely to every patient regardless of individual characteristics and needs. Even when relationships between staff and patient are good and there is close involvement, the understanding of the patient and his/her problems is often superficial and not based on individual study of the patient.

Some have attacked community mental health because "it has branched out well beyond mental illness into problems that it is not especially qualified to handle—community, national, and international affairs, poverty, politics, and criminality. In each of these areas, we have responsibilities as citizens and human beings; we have yet to demonstrate any special competence as psychiatrists" (4, p. 962).

A number of observers have attacked the whole policy of deinstitutionalization. Arnhoff (5) cites studies which show that community treatment is no better than hospital treatment for acutely psychotic patients,

either in short-term or long-term effects, and that community treatment has the additional disadvantage of placing a burden on relatives and the community; this burden is rarely negligible and in some cases intolerable. Arnhoff is concerned about the effects on children who are coming into greater contact with psychotic patients, both acute and chronic, who formerly would have been hospitalized. He points out that follow-up studies tend to reveal distressingly high percentages of marginal or poor adjustment, unemployment, and, for many, a subsequent need to return to the hospital and remain there. Arnhoff also worries that psychotic patients in the community will have a higher rate of procreation, produce more schizophrenics, and in many cases will be unable to provide adequate parenting for them.

In the early days of community mental health, there was much talk of the usefulness of blurring of professional roles and reducing the differences in both function and status among various staff groups. Psychiatry has suddenly awakened to find much of its power, prestige, and prerogatives being forcefully taken over by professionals from other disciplines and even by paraprofessionals. Community mental health gets the blame for this, and, in fact, such an egalitarian philosophy has been most widely articulated and put into practice in community mental health.

Still another criticism that is widely held but rarely aired in print is epitomized by the following:

> The [Federal] Office of Management and Budget is discovering inefficiency, waste, and vacuous promises, all right—not in good laboratories and clinics (although those will suffer from the cutbacks) but among the neighborhood storefront clinics, the funded "advocates," and other strange ad hoc "institutions." In the ballyhoo of the power struggles between minority political cadres and medical school administrators, nobody is served and potentially serviceable facilities have been destroyed. Lincoln Hospital, the Temple University Community Mental Health Center, the Boston City Hospital Drug Program and the Harvard Psychiatric Program at Boston City Hospital are just a few famous examples. Although the radical concerns are not as hot as they once were, and the street activists have quieted, they left a heritage of egalitarianism and complex jerry-built bureaucracies. The clamor for communities to determine their own health care delivery needs and to control the services still reverberates across the land. I, for one, hold the community psychiatry movement in no small way responsible for the current debacle—armchair philosophy and vague, untested notions are of no use in designing and running programs that cost hundreds of millions of dollars. (6, p. 1138)

Program evaluation has been proposed as one of the means of solving the problems of community mental health, but meaningful evaluation and quality control are rare in community mental health (13). Where such evaluations have been completed, administrators disregard them because of strong political, professional, and bureaucratic pressures to resist change.

Having acknowledged that these criticisms are not without merit, we must ask ourselves three crucial questions: What is the primary mission of community mental health? Has it really failed? What, if any, should be its future course?

THE PRIMARY MISSION OF COMMUNITY MENTAL HEALTH

In our view community treatment of the severely mentally ill is the raison d'être of community mental health. Perhaps the most important factor leading to the development of the community mental health movement was the concern about the abysmal conditions of treatment and life for patients in large state hospitals. These conditions were studied, documented, and publicized by Albert Deutsch in *The Shame of the States* (14) but were also of great concern to mental health professionals and their organizational leaders. A determination to bring about major improvements resulted.

Community mental health, to the extent that it grew out of the report titled *Action for Mental Health* by the Joint Commission on Mental Illness and Mental Health in 1961 (15), was intended to shrink large state hospitals by developing community alternatives, reduce the need for new admissions by the same means, and cover the country with community mental health centers that would provide a wide variety of services for patients who either had been in or would have had to be sent to state hospitals.

Even where hospital conditions were adequate, psychiatrists realized that most patients were capable of more than a life characterized by custodial care in an institution. A basic tenet of community mental health is that the treatment and rehabilitation of the long-term and seriously mentally ill can best be done in the community and not in large hospitals far removed from home (15). Implicit in such an approach is the concept of continuity of care not only between hospital and community but also within the community services.

A basic concept in community mental health is that behavior is affected, and in many cases determined, by the psychosocial-cultural milieu. In addition to understanding the intrapsychic conflicts of patients, we must understand their sociocultural backgrounds, adaptive capabilities, community supports, and social and family pressures. Armed with this knowledge, we can then provide treatment, rehabilitation, and support to deal with problem areas and to build strengths. Thus the community psychiatrist is involved not only with his patient but with his patient's milieu.

It is useful and important to consider which aspects of the contemporary community mental health movement were not among those originally propounded and therefore lack the foundation of years of concerned demonstration and testing. Among these latecomer elements are the ideas that many forms of mental illness are preventable, particularly by social changes, that effective comprehensive treatment and rehabilita-

tion of the seriously mentally ill in the community is less expensive than residential treatment, that the handicap of minimal theoretical and clinical training in treatment of the mentally ill can be overcome by membership in the same minority sociocultural group as the patients, and, finally, that mental illness inevitably results from, is tied to, or even is synonymous with deplorable, undesirable, or impoverished social conditions.

HAS COMMUNITY MENTAL HEALTH REALLY FAILED?

In our view, there has not been a failure of community mental health per se, but rather a failure to focus on the basic mission of community mental health. Only limited aspects of the original conception have been implemented, while the interest of most workers has gone off in other directions.

The major emphasis on reducing state hospitalization by administrative fiat without a corresponding effort to implement community treatment and rehabilitation of the long-term, severely mentally ill has not resulted from a lack of knowledge—effective community treatment techniques have been demonstrated and reported for a number of years (16). Instead, community mental health has tended to focus on treating the "healthy but unhappy." Considering the amount of manpower and funding available, an inordinate amount of effort and publicity have been put into primary prevention and social and political activism rather than direct service.

THE TURNING AWAY FROM COMMUNITY MENTAL HEALTH

We believe that community mental health has not been given a fair trial. The needs, originally so pressing that it was felt the structure of the national system of mental health services had to be revamped, are now just as pressing, but few political or professional leaders have made an all-out effort to meet them. A reasonable course of action would be for mental health professionals in general and psychiatrists in particular to move to meet these needs by putting into practice the basic concepts that still seem so promising. Instead, we see psychiatrists, especially those in academic settings, turning away from community mental health. A tendency exists to see mental illness as a collection of symptoms for which we need only select the right drug or as a biochemical disorder awaiting the discovery of the magic bullet that will cure the otherwise hopelessly ill (as if mental illness did not manifest itself in complex distorted interactions among human beings—interactions that can then lead to severe, permanent disability despite the disappearance of the original illness).

Why has this happened? Although we do not presume to analyze the motivations of any of our colleagues or of entire professional groups, we think the following factors are involved. First, professionals in community mental health settings have run up against the demands, not always reasonable, of community groups. These groups often see professionals as adversaries and as nonresponsive to the needs of the mentally ill. A similar situation exists in the attacks on psychiatrists as enemies of civil liberties. Having acquiesced to pressures to take on a social guardianship role, psychiatrists are surprised to find themselves accused of depriving persons of their civil rights. Thus many psychiatrists have been stung by such criticisms, which are at variance with their own self-images as physicians primarily concerned with the welfare of their patients, and have withdrawn to private practice or the academic ivory tower.

Another factor has been our disillusionment with the results of social and political activism and our realization that not everyone, and at times almost no one, regards us as the experts we think we are. There has been keen disappointment both within the professions and on the part of the public in our inability to reduce mental illness significantly by preventing it.

A further source of frustration has been the challenge to psychiatry from other disciplines and a feeling that in the area of community mental health the psychiatrist runs the risk of losing his status, his power, and even his high income. It is no wonder then that having ventured forth and tested the water in the community and having found it cold and uninviting, many of us have retreated to more scientific and academic pursuits.

RECOMMENDATIONS

Despite the difficulties of working in community mental health settings, our profession must not shrink from the task. It is time for a major rethinking of the community mental health movement and a return to the original fundamentals. There should be an emphasis on and high priority given to community treatment and rehabilitation for the severely mentally ill. A reasonable balance should be struck between direct and indirect services. Thus while still providing preventive and consultative services (we hope those with effectiveness that has come closest to being unequivocally demonstrated), we must retain a major emphasis on direct services for those groups in greatest need of psychiatric skills.

There has been important and exciting research in recent years in the biochemical and physiological bases of human behavior. Psychiatrists have every reason to be enthusiastic. Likewise, new drugs such as lithium have given new meaning to the refinement of psychiatric diagnoses; with drugs specific for affective disorders there is added reason to diagnose them correctly and differentiate them from the schizophrenias, and with the recognition of the serious side effects of

the antipsychotic drugs we have a mandate to develop our knowledge about these drugs and find more effective and less harmful ways to administer them. However, this research does not mean that we should decrease our interaction with our patients and no longer be concerned about the social elements and consequences of their illnesses. We are beginning to see cases in which almost no attention is paid to the specific kinds of interpersonal situations that have proven consistently overwhelming to patients, and no attempt has been made to identify the family, social, and vocational stresses that have immobilized patients and caused them to decompensate. The result is that little is done to help patients resolve these stresses, modify pathological patterns of behavior, and work with community resources.

Psychiatrists must become more involved in community mental health and should reassert their leadership. As the group of mental health professionals with both the broadest and most intense training in relevant areas, they have an unmatched overall perspective. Furthermore, the burden of ultimate responsibility for patient care that both law and tradition place on them makes any arrangement in which they serve simply as "team" members strained and cumbersome. They should not be deterred by accusations that they want to "perpetuate an outmoded medical model" or that they are "motivated by their power needs." Where there is truth in such indictments, they must be alert to it and correct their orientation and attitudes. Realistically, there is no other professional group that can adequately fill the gap if psychiatrists abdicate leadership; the result will be a chaotic battle with no rational means of settlement. We do not mean to imply that possession of an M.D. bestows administrative or social expertise or that nonmedical professionals have no leadership role. We simply assert that, in general, psychiatrists are better prepared for leadership than other professionals.

Finally, the training of psychiatric residents should be geared to meet the needs of the public and not the needs of a relatively small group of private psychotherapy patients. Residency programs should involve service to the needs of the total population (including the long-term severely mentally ill); training in crisis intervention; operation of a follow-up system for all patients; experience with rehabilitation, with resocialization programs, and with 24-hour care in graduated levels of intensity; and information and education programs for the public and professionals (17). The psychiatrists of tomorrow need to be oriented to the importance of making deinstitutionalization work as well as learning the techniques of treatment and rehabilitation that make this possible.

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Freedom of Inquiry and Subjects' Rights: An Introduction

BY DAVID REISS, M.D.

The author introduces the Special Section papers to follow, pointing out that two new issues have attached themselves to the issue of the physical safety and civil dignity of human subjects. One new issue is an emphatically legal defense of the concept of subjects' rights; the other is the evolution of political characterizations and caricatures of many forms of research.

DURING THE LAST FEW YEARS we have become much more sensitive to the issue of the rights of human subjects in medical studies. In my view, this accomplishment arises partially from the civil rights movement of the 1950s and 1960s. That movement made us all more sensitive to the intricate network of overt and covert social processes that offend human dignity and constrain human freedom. I think that our understanding of the medical experiment as a human encounter can only be enriched by applying what we have learned from other areas in which the civil rights of individuals have been championed. Another reason for the increased sensitivity to the rights of human subjects is the fact that during the last decade a series of painful and mortifying examples of the abuse of human subjects by scientific investigators has come to light. This has led us to redouble our efforts to prevent the abuse of human beings in research.

The story has not ended here, however. Those of us who spend our days engaged in research on human beings are becoming aware of additional problems relating to the human nature of medical research. These problems were at first hard to detect. In the beginning, it seemed that they too arose from the intense and impassioned defense of the dignity of human subjects; we heard more strident and urgent demands for the protection of human subjects, demands with which we could only agree. As time went on, however, particularly in the last two to three years, we seem to have been dealing with something new. As I see it, two new issues have attached themselves to the issue of the physical safety and civil dignity of human subjects. This theme is treated in more detail in the papers to follow. I will mention them only briefly here.

The first additional issue is an emphatically legal defense of the concept of subjects' rights. An increasingly complex set of government regulations is being elaborated to protect the experimental subject. This is vastly increasing the administrative complexities of conducting research. More important, it is crystallizing and formalizing an adversary relationship between subject and researcher.

The second issue is perhaps more ominous. It is the evolution of political characterizations and caricatures of many forms of research. A number of highly vocal groups are conceptualizing some psychiatric research as a political process that by its very nature threatens human freedom. The most conspicuous object of attack is behavioral genetics; this line of research has been caricatured as based on a creed that harkens back to Nazi Germany and that threatens to provide a rationale for weeding degenerates out of the human community. Similar political concerns have been raised about other research areas, including behavior modification, narcotics addiction, and psychopharmacology.

Our concern is that current efforts to protect subjects' rights, intensified by legal and political tactics, are providing serious obstacles to the conduct of research. I wish to emphasize that this by itself is *not* grounds for dismay. If subjects have actually been abused and if heroic measures are necessary to protect them, then it may be legitimate to accept curtailment of some research. However, we cannot accept such a conclusion without careful and concerned review. Indeed, in my view, the present threat to some areas of psychiatric research is so real that another form of hu-

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man freedom and dignity is severely threatened: our right to know and understand the forces that affect our lives; in short, the freedom of inquiry. Today we may be approaching a highly ironic, if not tragic, impasse where the freedom of inquiry comes into serious conflict with forces defending subjects' rights.

The first five papers in this Special Section of the *Journal* were presented in a session at the 1976 APA annual meeting. I chaired the session. The papers serve two functions. First, they survey the problem. They assess the obstacles that political and legal forces have erected against research and estimate the damage already done. Dr. David Musto, an eminent psychiatrist-historian, sets the matter in historical perspective; he notes the major forces that have arisen to support or retard laboratory research during the last few centuries. The focus of the papers then shifts to the severe problems in the present. Many sectors of psychiatric research are suffering and could have been selected for review; these sectors include behavior modification, behavioral genetics, and mental retardation. I chose three particular areas that have been hard hit to serve as examples of the growing emergency in psychiatric research. For each of the areas selected, I asked one of its most eminent and experienced investigators to review the current state of affairs. Dr. Jonathan Cole reviews problems in psychopharmacology, Dr. Roger Meyer reviews problems in narcotics addiction research, and Dr. Lee Robins reviews problems in follow-up studies. Drs. Cole and Robins focus on the intended and unintended destructive consequences of the newly constructed legal maze surrounding human subjects. Dr. Meyer comments on the disturbing problems arising from political opposition to research.

The second function served by these papers is to explore practical solutions for reconciling subjects' rights and the freedom of inquiry. The papers by Drs. Cole, Meyer, and Robins contain a number of practical suggestions; the paper by Dr. Bradford Gray focuses exclusively on the problem. Specifically, Dr. Gray focuses on research review committees (often called institutional review boards). These committees are now required by the U.S. Department of Health, Education, and Welfare in all institutions in which it funds research. The alert reader should not mistake these institutional review boards as just another harmless bureaucratic cog in the academic research machinery. They are a harbinger of a whole new process in psychiatric investigation as well as in other areas of biomedical research. They represent a systematized and powerful mechanism by which scientists and laymen can decide what research with human subjects may or may not be done. If this review process goes awry it can cause endless mischief and incalculable damage. If it functions properly, however, it bears the promise of reconciling subjects' rights and freedom of inquiry.

Taken as a whole, the papers in this Special Section are addressed to our colleagues in research and, more importantly, to our colleagues in teaching and practice. Beyond that they speak to legislators and to the general public, who are the beneficiaries of mental health research. I think we can all agree that a new age is dawning in psychiatry. During recent decades diligent and skillful practitioners have defined a set of major problems in psychiatric assessment, etiology, and treatment that require painstaking research of the highest caliber to solve. For their part, psychiatric researchers have developed exciting new theories and methods and have started to help solve some of these urgent problems. This productive cycle continues as practitioners and teachers, testing the new methods, have facilitated new advances in our field. This precious cycle of practice and research is one that we have hoped for and, as have other branches of medicine before us, seem finally to be achieving. Our concern is that this cycle may be severely interrupted just as it is becoming firmly established. Thus we ask the reader to examine the issues raised in these papers for their broad implications for psychiatry as a whole. We invite observations, comments, and suggestions.

Freedom of Inquiry and Subjects' Rights: Historical Perspective

BY DAVID F. MUSTO, M.D.

The author presents an historical overview of the various attitudes toward animal and human experimentation. He cites advocates (Bacon, Welch) and opponents (Johnson, Shaw) and traces the debate from the Graeco-Roman era, through the rise of the scientific establishment, to the present. Controversy over the fundamental rights of individual research subjects versus a future-oriented freedom of inquiry has been and continues to be a dilemma for clinicians and research scientists.

GEORGE BERNARD SHAW had a seemingly inconsistent scorn for what we would hail as some of the medical profession's finest achievements. He regarded bacteriology "as a superstition" and compulsory vaccination on the same level as compulsory baptism. His full fury, however, was aimed at the laboratory technique almost synonymous with Pasteur, Koch, and Sherrington: experiments on living vertebrates or, more broadly defined, vivisection. Shaw saw no sharp demarcation between animal and human experiments and no justification for either. In 1911 he argued that "the exemption of the pursuit of knowledge from the laws of honor is the most hideous conceivable enlargement of anarchy . . . a curious devil might destroy the whole race in torment, acquiring knowledge all the time from his highly interesting experiment" (1, p. 56). Shaw liked to enrage his enemies and astound everyone else, but this sentiment was no casual jab at medicine. More than one of Shaw's readers have wondered how such a self-advertised advanced thinker could be under the spell of an antiscientific sentimentality.

We find less puzzling the testimony in 1900 of Dr. William Henry Welch to a Senate committee considering an antivivisection bill. First Dean of the Johns Hopkins Medical School and a world-renowned bacteriologist, Dr. Welch spoke against restrictions such as Shaw would impose on the freedom of inquiry:

> Our own contributions to [medical] progress may now be small, but America is destined to take a place in this forward movement commensurate with her size and importance. We today should be recreant to our great trust, did we not do all in our power to protect our successors from the imposition of these trammels on freedom of research. Our appeal to you is not only in the name of science, but in the truest and widest sense in the name of humanity. (2, p. 101)

Backed by influential and educated Americans defending medical research, Welch won an overwhelming victory against the antivivisectionists. Shaw's crusade to stop animal experiments was no more successful than his campaign for a new English alphabet.

Today we are again questioning whether medical research on living beings should be severely restricted or perhaps even prohibited. I would like briefly to sketch some history of the strife so eloquently represented by Shaw and Welch and still divisive in 1977.

EARLY VIEWS ON RESEARCH AND PROGRESS

Human experimentation involving drugs, diet, regimen, and surgery has existed as long as one person has treated another, although institutional and methodical experimentation on humans was uncommon in the Graeco-Roman era. At the height of Athenian excellence scientific inquiry seemed merely a strange pursuit and gave Aristophanes' audiences a good laugh. His portrait of Socrates in *The Clouds* as headmaster of a school for pure research did not reveal Athenian fear of any tangible evil resulting from scientific research or, for that matter, recognition of any practicality. Interestingly, Aristophanes linked pure research to an atmosphere of sophistry and an attack on old values.

The Western classical world did not expect that scientific inquiry would bring radical and quick improvement in life-style, transportation, communication, and health and felt that only more of the traditional elements of the good life might be obtained through wealth, power, or luck. The rise of Christianity devalued even the material rewards wrought by temporal success: progress would be spiritual and measured externally by charitable actions. For both Christian and pagan, each believing the world in decline after a distant Golden Age, progress itself was an uncommon concept. Conflict between freedom of inquiry and subjects' rights required establishing experimental science

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and medicine as social institutions. Until science could wield practical knowledge with a beneficial impact on everyday life, it would tend to yield when in conflict with ethical and cultural traditions.

Francis Bacon at the beginning of the 17th century held a larger vision of science. He minimized exaltation of truth for its own sake, feeling that science must be utilitarian. As Lord Chancellor, Bacon urged King James to support scientific academies and intensive experimentation into practical problems, but it was too early in the scientific revolution for Bacon's ideas to appeal much to practical minds. King James listened incredulously and then, with an Aristophanic wink, remarked that Bacon's ideas were "like the peace of God, which passeth all understanding."

RISE OF A SCIENTIFIC ESTABLISHMENT

Bacon's desire for a practical, experimental, scientific enterprise would be gratified later in the 17th century when experimenting became a fad for advanced thinkers, scientific minds, and practically anyone looking for a new hobby. Restoration playwrights ridiculed experiments of the Royal Society as much as any Attic comedian had mocked Socrates. Telescopes, microscopes, vacuum chambers, and timepieces became, depending on the observer's perspective, tools or toys for the curious. Squints at fleas and microorganisms could amuse or instruct, but crude operations on birds, cats, dogs, and horses disgusted some observers and experimenters. The pain apparently felt by animals was one criticism, but the effect of experimentation on the investigator's virtue was as often invoked against the practice. One is reminded of Thomas Jefferson's warning that the most damaging effects of slavery were on the slave-owner's character.

Bacon's contemporary, William Shakespeare, portrayed this negative aspect of medical research in *The Tragedy of Cymbeline* (c. 1610). A malicious Queen eager to investigate the "most poisonous compounds" applies to a court physician offering what must be one of the earliest research protocols:

> . . . I will try the forces
> Of these thy compounds on such creatures as
> We count not worth the hanging—but none human—
> To try the vigor of them, and apply
> Allayments to their act; and so by them gather
> Their several virtues and effects.

The physician, moral more than curious, warns her that this research will harden her heart, and then confides to the audience:

> I do know her spirit
> And will not trust one of her malice with
> A drug of such damn'd nature. . . .
> . . . first, perchance, she'll prove on cats and dogs,
> Then afterward up higher. . . .
>
> Act I, scene v

To those shocked by a vogue for animal experimentation the question recurred, Does curiosity require cruelty for satisfaction? The carefully timed drowning of animals, cutting living dogs open to study respiration, and asphyxiating kittens in a bell-jar evacuated of air could have such specific and important scientific goals as learning whether the lungs needed ventilation in order to support life. As for allegations of cruelty to animals, even troubled researchers could reply that the Bible had given man mastery over animals. In these investigators' own time Descartes submitted that because only man had a soul, only man could feel pain; an animal could be damaged, but not hurt.

Questions persisted into the 18th century over the morality of animal experiments. Dr. Samuel Johnson, an ardent antivivisectionist, complained bitterly that in animal experiments "knowledge is not always sought, and is very seldom attained." Like Shakespeare, Johnson saw experimentation hardening the heart of the experimenter, extinguishing "those sensations which give man confidence in man, and make the physician more dreadful than the gout or stone" (3, p. 56).

What would Dr. Johnson say in 1850 or 1900? Could discoveries in bacteriology, immunology, surgery, and pharmacology have been made without animal experiments? What would be Johnson's reaction to the possibility that our generation may see the last case of smallpox? All modern societies willingly offer influence and prestige to medical researchers for the practical benefits they provide. The power given research, however, has created a deeply felt dilemma over the rights of individual research subjects. Is it simply a tough law of nature, as one defender of human experimentation stated in 1882 (4), that "the few must suffer for the many"? Dramatization of contemporary research would require Euripides, not Aristophanes. We have descended from Cloud-cuckoo land to the cancer ward.

FEAR OF AN UNCHECKED SCIENTIFIC ESTABLISHMENT

Two kinds of fear about science coexist. On a mass scale, there is the Frankenstein spectre that science will learn through experimentation some destructive secret. We already confront an atomic bomb; could we also acquire an epidemic of an untreatable, fatal disease as a result of genetic manipulation with viruses?

Shaw and Dr. Johnson did not address the catastrophic possibilities of science's freedom of inquiry; they feared small-scale effects as this imperial institution came into contact with one person, then another, and another—isolated individuals before a juggernaut. This is the other fear underlying current efforts to safeguard subjects' rights. Johnson worried about the experimenter who becomes a cog in the juggernaut's wheel when he regretted, "he surely buys knowledge dear, who learns the use of lacteals at the expense of his humanity" (3). Shaw was outraged on behalf of the

promptly jailed "conscientious objector" to vaccination.

The antivivisectionists believed experiments on living beings destroyed sensitivity to charitable relations between people and between people and animals. Was the new world promised by science worth sacrificing a fragile and precious form of moral development? Understandably attracted to this zealous movement were protagonists of other struggles who shared resentment toward repressive and indifferent social institutions. Women fighting for equal rights and angry defenders of deprived social classes, who suspected physicians used poor patients as human guinea pigs, combined to protest against the depersonalizing trends of industrial and scientific progress.

By the 1870s animal experiments began providing practical, at times almost miraculous, medical advances. As a series of remarkable discoveries quickly increased chances for a healthy and comfortable life, the public naturally assigned great responsibility for these startling advances to animal experiments. Antivivisectionists could not compete with the enormous hopes held for future medical research. Support for all kinds of scientific research has built a gigantic social institution. The result is that most of the scientists who ever existed are alive today, and they create an unparalleled demand for human research subjects. The consequent routinizing of human research on a grand scale may be related to Professor Bernard Barber's claim that human experimentation of questionable ethics reaches its highest percentage in run-of-the-mill "mass producers" of scientific papers (5).

COUNTERFORCES TO THE SCIENTIFIC ESTABLISHMENT

If we compare present criticism of research on humans with the heyday of antivivisection activity in the last century, we find similar pressures against experimentation and in favor of strict, perhaps prohibitive, safeguards for subjects' rights. In the 1870s a sizable minority could still question whether the spiritual costs of vivisection—including human experimentation—were balanced by hope of practical benefits. Now, having gained dramatic victories over childhood and infectious diseases, facing apparently slow progress on diseases of later life, and suspecting an excessive busy-work quest for human research subjects, critics argue that many experiments on humans could be reasonably eliminated. Perhaps we have reached, on a higher plateau of practical knowledge, a new equilibrium of lowered expectations from science and its research methods. This new balance may be found in demands that now is the time to consolidate what we already possess in scientific advances, distribute health services more equably, and develop sensitive ethical relations with one another. The spirit of conservation, so prominent in the environmental movement, has weakened a once simple faith in the benefits of untrammeled research by emphasizing the dangers of new (and old) technology to the quality of life. Further, some disadvantaged groups find the complex technology of experimental scientists repressive rather than evidence of a mysterious power over illness; they feel it excludes them from a higher social status while they are being used as subjects to sustain the scientists' prestige. Contemporary echoes can be heard in the warning, over 80 years ago, of an English antivivisectionist and opponent to medical research on humans:

> In its relation to science and her operations, the working class occupies, and is beginning slowly but surely to realize that it occupies, a position perilously more near to that of the vivisector's subject than to that of the patient of the fashionable physician. . . . And whatever gains may be achieved by the process will be achieved, not for its benefit, but at its expense.(6)

COMMENT

A similarity worth consideration between current opposition to experimental procedures and earlier strenuous opposition to vivisection is a turn among some thoughtful people toward a primary emphasis on the style or quality of each individual's life. For them, justifying risks today to obtain possible future scientific miracles has become less attractive, and unrestricted human experimentation has become an unnecessary hazard. Critics will point out that this emphasis on "quality of life" is possible because the scientific achievements of the last two centuries have created a relatively satisfactory quality. Isn't the internal meaning of life easier to contemplate when external risks are less threatening? Or is the declining enthusiasm for medical research a valid reawareness of our common mortality, which has been obscured by a century of unique, but still inevitably limited, medical progress?

The skeptical attitude toward research on humans so confounding to investigators may announce resurgence of modest expectations from utilitarian science, an attitude of near indifference mankind found congenial and proper until, historically speaking, quite recently. Those questioning the wisdom of seeking unlimited material progress must be sympathetically understood if an accommodation is to be made between future-oriented freedom of inquiry and our here-and-now rights as living beings.

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Research Barriers in Psychopharmacology

BY JONATHAN O. COLE, M.D.

The author discusses barriers to psychopharmacological research, including attacks by vocal human rights groups, regulation by local review boards, and Department of Health, Education, and Welfare restrictions. He suggests that those patients least able to give informed consent are most in need of the benefits of research on new drugs.

OUR CURRENT ARRAY of drug therapies for psychiatric patients is often helpful but is inadequate for the needs of many patients. Antipsychotic drugs improve the conditions of only two-thirds of schizophrenic patients treated with them. Even patients who improve often continue to be socially ineffective and to suffer from delusions, hallucinations, and thought disorder. In addition, some drugs have undesirable early and long-term side effects. Antidepressants, for example, take one to three weeks to act, have anticholinergic and cardiovascular side effects, and improve, at best, one-half of the patients treated. The most effective antianxiety drugs can cause psychological and physical dependence. Drugs used to treat senile dementia are either ineffective or can be distinguished from placebo only with difficulty. Mental retardation and severe personality disorders lack drug therapies almost entirely. Current drugs are a vast improvement over the electroconvulsive therapy, insulin coma therapy, and barbiturate sedation that were the only treatments available in 1950, but many patients are still chronically distressed, dysphoric, and nonfunctional, despite our best efforts at drug therapy and psychosocial therapies.

It seems to me that public policy over the last few years has been making the development of better drug therapies in psychiatry more and more difficult. Well-meaning regulations have caused the costs of drug development at the preclinical level to sky-rocket and, at the clinical level, have placed a growing array of bureaucratic hurdles between the investigator, the patient, and the acquisition of new knowledge. Regulatory agencies and treatment facilities are getting deeper in red tape, much of which is alleged to be in the interest of patients' rights and safety. Pressure for these escalating rigidities seems to come, however, from a small group of very vocal laymen who are hypersensitive to a range of risks—e.g., to privacy, safety, humanity—but seem totally uninterested in the benefits of better therapies. As one reads attacks on clinical drug research, one gets the feeling that the attackers believe that mental illness and suffering do not exist and that all mental patients would be better off totally untreated or that a little love and moral support can make everyone happy.

It is ironic that at the same time there is public pressure to prove that drug therapies are really effective, something which cannot be done without controlled clinical drug studies. Current restrictions make studies of drug therapies very difficult and make more basic studies that use drugs as tools to study the biological bases of psychiatric disease even more difficult.

PRESSURES TO STOP ALL RESEARCH

Recent attacks on psychiatric drug research by small groups of enraged citizens have caused some public mental hospitals to abandon all drug research. The attack on the Missouri Institute of Psychiatry by the Scientologists is one alarming example; the attack

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by a legal aid group on research at the Lafayette Clinic in Detroit is a second. The killing of a controlled study of drug therapies for disturbed children in Boston is a third instance. In all of the above cases the research attacked was dropped even though it had met all Department of Health, Education, and Welfare (DHEW) and local institutional requirements. A study of a narcotic antagonist using paroled drug addicts in Boston was stopped in the same way.

Research in a number of prison systems—most recently in the Federal Bureau of Prisons—has been stopped by fiat in the past couple of years. Research in public mental hospitals may well be the next to go. Although one can argue the pros and cons of drug research with prisoners (I personally believe that prisoners can give informed consent, that payment for their trouble is legitimate, and that they can in this way partially repay a real debt to society), the actions on the part of prison administrators, at least in Massachusetts, have been in response to pressures from vocal minorities; it's easier to stop all research than to be hassled by angry groups. The social need for research and the rights of the many prisoners who want to participate in research have not, I believe, been considered at all seriously.

Although the overall threat to psychiatric drug research is ominous, in many settings it is only a cloud visible on a distant horizon. The current DHEW regulations, however, are a real, present, and pervasive difficulty.

THE REVIEW GROUP

Present regulations require an institutional review board (IRB), which must have totally disinterested (but unpaid) citizen members. This latter requirement sounds reasonable but presents grave practical difficulties. How can one find mature, sensible citizens who are willing to put in a lot of time without pay to review and monitor studies? At one of the two institutions at which I do research all such members come for two or three meetings and then gradually disappear, despite our best efforts to retain them. When present, they are usually helpful and reasonable.

Such committees, like all committees, can get incredibly hung up over minutiae. For example, one IRB spent five hours of time over several weeks considering the problem posed by offering schizophrenic patients in an aftercare program $20 to participate in two two-hour sessions of electroencephalographic recording. After the issue of too much versus too little pay had been beaten to death, the possibility that patients in marginal job situations might jeopardize their jobs by volunteering for the study was brought up. The issue of harm from research versus individual rights of self-determination was argued vigorously and interminably. To make matters worse, it was never clear that any of the available patients actually had jobs to begin with. What I am trying to say is that committees can become embroiled in personality conflicts and can reflect institutional schisms; these problems can lead to agonizing deliberations that are generally irrelevant to issues of human safety or patients' rights. All bureaucratic remedies are likely to have undesired and sometimes unanticipated side effects.

In addition, with the current review process required by the National Institutes of Health (NIH) and the Alcohol, Drug Abuse, and Mental Health Administration, investigators face double jeopardy. A research design acceptable to a local IRB may be vetoed at the NIH level. All multilayer systems of this sort increase the likelihood that someone will say "no." Nowhere in current regulations is there any provision for appeal of a restrictive ruling made at any level to a review group at a higher level. I'm not even sure I want an appeal mechanism—it would tie up even more time and create more paper work; however, the more elaborate our safety review systems get, the less progress will be made in clinical research.

INFORMED CONSENT

A larger and more unique barrier to psychiatric drug research is the requirement that full, informed consent be obtained in writing from all patients. At one level, I doubt that most nonpsychiatric medical patients are capable of really understanding all the issues and risks involved in consenting to participate in either drug studies or conventional therapies. The risks in most drug studies are less than those associated with elective surgery or even with receiving penicillin for a streptococcal infection. Patients asked to participate in drug studies have a lot more freedom of choice than do patients who go to their own doctor for routine treatment. In neither case can one ever be sure that full information is imparted and understood.

A more serious problem for psychiatric research is that the patients needing better drug therapies the most—treatment-resistant chronic schizophrenics, patients with senile dementia, mental retardation, or marked behavioral disturbance—are often the least able to give informed consent at all. The appointment of legal guardians for such patients is expensive and time consuming. It resolves the legal issue but may not resolve the ethical one. To deny such patients any research drugs may leave their rights intact but will also deprive both the individual patient and such patients as a class of any chance to benefit from new therapies. At worst, it leaves them treated with older therapies which themselves have been inadequately studied in such patients.

It seems to me that degrees of risk exist and that degrees of consent should also be recognized. I find it hard to believe, for example, that a new technique for monitoring an old drug therapy—drawing blood to determine drug blood levels—requires any more "consent" than drawing blood to monitor diphenylhydantoin or lithium levels, providing such a proce-

dure is done only occasionally. The physical risk is nonexistent, the discomfort is slight. In cases in which the patient's privacy might be violated—a very unlikely event—the IRB could handle this problem without escalating the whole issue to the signed-consent level. If a series of blood samples or a venous catheter is required, informed consent is desirable but should not be mandatory.

With studies involving several blood drawings, several hours of polygraphy or EEG recording, or participation in psychological testing, it seems to me that the patient's willingness to participate (acquiescence) should be sufficient "consent." Most of these research procedures can't be done with resistive or uncooperative patients anyway. The patient's willingness to go along with the procedure—as he might with a clinically indicated EEG or psychological testing—seems to me adequate consent. Blowing up benign procedures into major issues seems unwarranted. Of course, the patient should be told about the procedure and its purpose and any attendant minor discomforts, but patients who are so impaired that it is unlikely they will fully understand the procedure should still be available for study without court-appointed guardians being required. The IRB should approve the study, including its provisions for protecting privacy and the manner in which cooperation is elicited.

Comparisons of standard marketed drugs used for an FDA-approved purpose also entail no greater risk than nonresearch treatment; use of patients unable to give full informed consent should also be possible in these studies.

I would also like to see a mechanism developed which would permit the study of very benign drugs of dubious clinical value in comparison with placebo in conditions for which no established treatments exist. Some of our current geriatric drugs (Hydergine, papaverine) would qualify for study under such a procedure, as might studies of established antipsychotic drugs in mentally retarded patients. In such cases consent from the next of kin might be the appropriate level of consent.

Obviously, either full informed consent or consent by a court-appointed guardian would be necessary for studies with new drugs or for procedures with greater potential risks. At even higher risk levels—where the potential for benefit is equally high or higher—special monitoring of consent procedures and of the patient's progress during the study may be desirable. I believe that acceptance of a range of degrees of acquiescence or consent appropriate to the risk or lack thereof is needed. Without this flexibility, minor research procedures become encumbered with complex mandatory procedures that were designed with psychosurgery in mind.

CONCLUSIONS

In my own limited experience, the "informed consent" issue has interfered with research in several ways. A study of cyproheptadine (Periactin), a marketed antihistamine, would have been carried out in chronic psychotic patients with tardive dyskinesia at Boston State Hospital if patients unable to give informed consent could have been asked to consent to take the pills. I believe that the risk would have been minuscule; a pilot study in a private-practice setting had been favorable. A study of the dose management of loxapine, a marketed antipsychotic, in agitated patients with senile dementia will probably not be done at McLean Hospital because informed consent by the patient or a legal guardian is required by our IRB. I am sure that this drug is being used unsystematically in such patients without the care involved in a careful clinical trial. A study of chlorpromazine blood levels in newly hospitalized schizophrenics has been greatly retarded by the requirement of prior informed consent; obtaining acquiescence for blood sample drawing during the early days of hospitalization and informed consent for use of the data later, when the patient had improved, would have tripled the rate at which the study was done.

It is interesting to note that the original purpose of informed consent was to protect the investigator from future tort actions; this role has been declared improper, and the procedure now serves a growing range of regulatory and bureaucratic purposes.

Barriers to research are being erected by pressure groups and regulatory agencies. They impede or threaten to destroy all research, but are particularly difficult in the area of clinical psychopharmacology. We are in real danger of preventing the development of any new drug therapies for mental illness unless more flexible procedures are developed. Many, many patients will suffer needlessly if we are compelled to continue to use only our current drug therapies for the next 20 years.

Subjects' Rights, Freedom of Inquiry, and the Future of Research in the Addictions

BY ROGER E. MEYER, M.D.

Since the recent passage of regulations concerning subjects' rights and freedom of inquiry, opposition by the public and others to some areas of research in the addictions has prevented its implementation or continuation. Research investigators in the biomedical and behavioral sciences have been placed in the position of defending their work in an adversary climate. The author points out the importance of transmitting to the public, the scientific community, and legislators the investigators' concern that "subjects' rights" not be viewed only in a legalistic context, but also in the context of not harming the patient.

TO THOSE OF US with a humanistic orientation who were educated in the medical schools of the late 1950s and early 1960s, the movement to apply human studies reviews and an ethical brake on research is understandable and justified. To those of us with a concern about the advancement of knowledge and the importance of free inquiry to our profession and our society, the present rush by some to condemn and restrict clinical research (and other scientific studies with human subjects) suggests a dangerous trend that may have moved beyond its initial noble impulse. The scientific community has responded by defending the enterprise on the basis of previous achievements and the continuing need for the struggle against diseases that afflict humankind. The failure of this defense was suggested by a recent editorial in the *New York Times* (1) equating this position with the defense mustered by our intelligence agencies on behalf of practices that were said to serve the interests of national security. In this context, our political system has seemed to be moving to set up countervailing forces and adversary procedures designed to "equalize" the interaction between research scientists and research subjects.

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At the time this work was done, Dr. Meyer was Associate Professor of Psychiatry, Harvard Medical School, Boston, Mass.; Director, Harvard-Boston University Center for Biobehavioral Studies in the Addictions, Boston, Mass.; and Associate Director, Alcohol and Drug Abuse Research Center, McLean Hospital, Belmont, Mass. He is now Chairman and Professor, Department of Psychiatry, University of Connecticut School of Medicine, Farmington, Conn. 06032.

If the concern about research with human subjects were limited to a concern about inadvertent or unavoidable harmful consequences of innovative procedures or research methods, then one could realistically restrict potentially harmful procedures while providing a mechanism to compensate subjects who have participated in research and who have sustained inadvertent complications consequent to their participation in the experiment. This would be consistent with the medical ethic *primum non nocere*, "above all do no harm." We have, however, moved into an area where the ethics of human experimentation are increasingly being defined on the basis of "imperatives" that are more consistent with the principles, practices, and beliefs of civil liberties attorneys. It is in the context of these ethical imperatives that individual research investigators have been unable to defend their work and approved research projects have been stopped. To some, this situation is reminiscent of the legitimate anguish of the oppressed against the oppressor elite. To others, the situation is but one more repetition of the classic struggle between scientists and those who hold political power as to whether science shall serve or be independent of political ideology.

The distinguished philosopher Charles Frankel has observed that it is not technology per se that threatens mankind or a humanistic view of man; rather, the real threat lies in the tendency of some people to superficially link technological data to support for ideologies and movements (2). Indeed, on the issue of censorship of biomedical research, Frankel wrote,

> For while technology is equivocal in its consequences, one thing is not—the damage that ensues, almost always very quickly when a field of inquiry is banned because it is said to conflict with higher moral laws. The number of subjects that can come under this axe is indefinitely large. The character of the people who have ever wielded this axe is not reassuring. (2)

The future of clinical research seems least secure in areas of experimentation into the causes and treatment of nonlethal conditions, particularly in fields where treatment and prevention programs are polarized and scientific advances "appear" to offer little and may threaten philosophical positions. By these criteria, research into the causes and treatment of substance abuse should be controversial, and it is. There are two general areas of controversy that can be highlighted by

case examples. A third general area (confidentiality and privacy) has been described by Robins (3).

FREEDOM OF INQUIRY VERSUS RESTRICTED INQUIRY

The Study of Alcoholic Mothers

Data from a study by Jones and associates (4) suggest that offspring of alcoholic mothers may have a greater likelihood of premature birth and various levels of neonatal distress. The neonatal opiate withdrawal syndrome in the offspring of heroin-addicted mothers was described a number of years ago and has been updated by the work of Finnegan and associates (5). The question of neonatal distress in the offspring of alcoholic mothers is in need of more careful delineation.

A team of psychiatrists, obstetricians, and pediatricians at a municipal hospital undertook a study of neonatal behavior and development in the offspring of mothers who were being seen in the prenatal clinic and who were identified as having problems of alcohol abuse. The physicians offered a program of psychotherapy coordinated with the prenatal care to assist the expectant mothers with their drinking problems. Preliminary data suggested that the offspring of mothers who drank heavily were at high risk as compared with the offspring of patients who drank moderately or rarely. On the basis of these preliminary findings, a grant was prepared and submitted to the National Institute on Alcoholism and Alcohol Abuse (NIAAA). Because the protocol also included the support of a treatment and prevention program, the review group included persons who could consider all aspects of the study. One of the reviewers was a community activist on the staff of an agency affiliated with the medical school at which the work was to be done. Despite the fact that this institutional affiliation should have excluded him from the review, he participated in the review of the grant application and a subsequent site visit.

The NIAAA staff then demanded that, in addition to the already obtained institutional reviews, the protocol be considered by a locally constituted group called the Community Research Review Committee. After reviewing the protocol, the committee felt that the research might draw defamatory conclusions regarding the competency of black mothers, since 55% of the clinic population was black. The committee insisted on monitoring the personnel practices and conduct of the research and previewing any formal or informal reports emanating from the project. In order to effectively monitor the work, the committee members stated that they would have to receive a portion of the budget as overhead. The principal investigator was told that since it was already the responsibility of the NIAAA and the institution to monitor the project, federal funding for a third group of monitors could not be budgeted. While it was clear that the NIAAA staff had erred in requiring that the research investigator obtain the sanction of this locally constituted group, it was also clear to the research investigator that the full-scale program was not possible in the face of determined political opposition.

Narcotic Antagonist Project 1

The Community Research Review Committee has made similar budgetary demands upon a number of other nonpolitical research investigators whose research projects involved studies into the causes and treatment of nonlethal conditions. The committee members have been relatively selective in their approach and apparently have not made these demands upon all research investigators in the geographical area. On one previous occasion they approached a team of research investigators who proposed to study the effects of narcotic blocking drugs in a population of heroin addicts at the same municipal hospital. Their demands at that time included 10% of the budget, 50% of the positions, and the appointment of an associate director of the project from among the membership of their committee. They also demanded censorship of reports.

The thrust of the committee's concern regarding studies of narcotic antagonist treatment of heroin addicts seemed to be that the research investigators were taking a simplistic, biomedical view of a complicated social problem that (in its view) really required a solution to the problems of poverty and other social ills in the community. While the causes of addiction may be multifactorial, the committee was concerned that the biomedical orientation of the research project conflicted with its own view regarding the causes of the problem and its own definition of funding priorities. Faced with powerful opposition that undermined the support by the hospital's trustees, the research investigators chose to transfer this project to another community.

While individual communities will continue to be able to exercise a political veto over research within their midst, the real question concerns the application of this principle on a larger scale. At least one monograph prepared for the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research (6) proposed to sanction "community consent," which it defined as "informed consent negotiated with the community in which it is planned to conduct research or from which it is planned to draw prospective subjects." If this recommendation is adopted in this form, its consequences for the future of research could be extremely restrictive. As the Edelin trial has demonstrated, such political pressure can come from diverse sources and can scapegoat ethical practitioners and scientists.

Federal Opposition to Behavioral Research

Political concern about behavioral research with relevance to addictive drugs has not been limited to ad hoc community groups. Comments by Senator Proxmire and the threat by several congressmen to cut off

funds from the National Institute on Drug Abuse prevented the implementation of an approved and funded research project at Southern Illinois University on the effects of marijuana on human sexuality. The researcher had previously obtained local lay approval for his study, in addition to required institutional reviews.

The penultimate federal document challenging the ethics of behavioral experimentation was a report prepared by the Senate Judiciary Committee on Individual Rights and the Federal Role in Behavioral Modification (7). The report alleged,

> The behavioral technology being developed in the United States touches upon the most basic sources of individuality, and the core of personal freedom. . . . While psychosurgery under certain forms of electrophysiology is perhaps the most . . . sophisticated method of behavioral control, there are now being tested a number of other techniques based on more traditional, psychological principles [that] pose similar questions with regard to individual liberties. (7)

The implications of this report suggest the need to restrict behavioral research in the interests of the civil rights of individuals in the future, as well as the present. It is curious that alcoholism and drug addiction are major behavioral disorders whose alteration will require one or another form of behavioral modification. Indeed, methadone maintenance, narcotic antagonist treatment, and self-help residential programs all seek to modify behavior. The issue would seem to be whether clinical research in these areas can proceed in a variety of voluntary and nonvoluntary settings with appropriate regard for adequate and voluntary informed consent.

SUBJECTS' RIGHTS VERSUS THE RIGHT TO TREATMENT; SUBJECTS' RIGHTS VERSUS SOCIETY'S NEEDS

There is a recurring notion that research subjects must be protected from researchers by an adversary process (consent committees versus review committees, and patient advocates versus research physicians). The adversary process is a legal construct that works effectively in the courts and the political system. When applied outside this framework to the practice of medicine and research, it may run counter to the medical ethic of "do no harm." Indeed, as in the field of mental health generally, the arbitrary imposition of "individual rights" in a legal context may impose medical and social risks for individual patients. The following examples highlight the problem in addiction research.

Narcotic Antagonist Project 2

Vaillant (8) has carried out extensive follow-up studies on heroin addicts discharged from the U.S. Public Health Service Hospital in Lexington, Ky. From his work it is clear that patients who are on probation or parole do better in all forms of treatment. Indeed, many of the self-help residential "concept houses" have increasingly restricted admission to patients who are court referred because the dropout rate of volunteers in these hierarchical, authoritarian environments was too high. Vaillant's findings are clear: in the absence of some system of control, the addict is not able to modify his own behavior. As I have described elsewhere (9), that control can be manifested by methadone maintenance; an authoritarian, hierarchical residential community; or the criminal justice system.

The recidivism rate of former heroin addicts discharged from prison is quite high. This population presents unique problems in terms of the relatively effective treatments available to narcotics users. Since they have been drug free for a period of time in prison, they are not considered appropriate candidates for a program of opiate substitution (e.g., methadone maintenance). Since they have been living in a prison for a period of time, they are not anxious to volunteer for a 9- to 24-month residence in a "concept house." While parole or probation support may be helpful to the individual in his efforts to seek a job or obtain some sort of counseling, the presence of many stimuli associated with previous addiction cycles (familiar friends and "drug copping areas") may encourage relapse. The National Institute on Drug Abuse recognized that this population might be ideal for narcotic antagonist treatment. They funded one such project in one community in association with that city's drug treatment program. The principal investigator of the project described its course as follows:

> The project as approved and funded by HEW proposed to offer treatment to prison inmates shortly prior to their release from prison and during a parole period subsequent to release. A condition of the project . . . stated that under no circumstances could participation in the project be a consideration for parole. Plans were finalized for the conduct of the project in such a way that patients would not be approached to give informed consent until after the parole board had met and voted for parole. Despite the fact that a joint medical school and hospital based human studies committee approved of this project, the study attracted widespread local attention when the research protocol was stolen in a burglary of the office of the principal investigator. [The study was described in the local media in such a way that prison inmates were seen as guinea pigs and parole was described as dependent on participation in the study.] As a consequence of the widespread circulation of these allegations, community based and other agencies' human study committees requested review of the project. In a long series of reviews, multiple meetings resulted with four additional human studies committees, and the project met with final approval shortly before the expiration of the project period. Only two subjects entered the research study and HEW finally made the judgment that extension of the project with refunding was unwise. The principal investigators viewed the episode as

one of continuous harassment by an ill-informed media and a series of well-intentioned but unsophisticated and at times ad hoc human studies committees.

When one considers the number of "rehabilitation measures" that are routinely employed in the context of parole, probation, and other aspects of the criminal justice system, one is struck by the fact that these measures are seldom evaluated. Indeed, if informed consent is ruled out for this population in an a priori manner, then the population will be exposed to all sorts of unevaluated interactions with undefined risks while being prevented from participating in rehabilitation programs with a research-oriented design.

The Addiction Research Center

Forty years ago the Public Health Service set up the Addiction Research Center at Lexington, Ky., in order to screen new drugs for their potential to cause addiction, to explore the biomedical and behavioral aspects of the addiction process, and to evaluate potentially promising pharmacological approaches to the treatment of addiction. Over the years the mission has evolved consistent with evolving federal priorities. The addiction liability of many new analgesics became known before they could be advertised as "nonaddicting." The human pharmacology of methadone, *l*-α-acetylmethadol, cyclazocine, naloxone, and naltrexone were all delineated at Lexington. Over its 40-year history there have been no deaths or serious disabilities consequent to this research. With the development of locally constituted drug treatment programs in nearly all parts of the country, the need for a "treatment farm" for volunteers in Kentucky became unnecessary. Instead, in 1973 the Federal Bureau of Prisons relocated its facilities for known heroin addicts to Lexington in proximity to the Addiction Research Center.

It is now four years later. The staff at the Addiction Research Center was told that they could not begin any new studies using federal prisoners after January 1, 1977. The distinguished team of pharmacologists and behavioral scientists has been perplexed. Dr. William Martin testified before the National Commission for the Protection of Human Subjects in March 1976 concerning the importance of the work and the safety of the procedures. Within a month of Dr. Martin's testimony, the Bureau of Prisons acted independently in response to congressional inquiry and unilaterally ordered the end of research in prisons under its jurisdiction. This order came out before the conclusion of deliberations and the preparation of the final report by the national commission. In his testimony, Dr. Martin wondered how society's needs for safe analgesics and new pharmacological approaches to the treatment of addiction could be reconciled with the closing of Lexington. He reviewed the safety of the procedures at Lexington, and he argued that the prisoners were capable of giving informed consent.

COMMENT

Four issues have been raised with regard to informed consent in research involving the mentally disabled, children, and prisoners: 1) the question of the capacity of individuals in each group to give informed consent, 2) the question of coercion, 3) the question of a need for an adversary procedure to defend the rights of research subjects, and 4) the question of community consent. While all four of these questions have been applied to the mentally disabled, only one has been applied to research with prisoners and persons in the criminal justice system—the issue of coercion within an institutional setting. Persons living within institutions are thought to be at risk for coercion. One of the monographs prepared for the commission, which dealt with the mentally disabled, stated, "When proposed research is considered therapeutic, an institutionalized individual may be under more pressure to participate insofar as the possibility exists that the anticipated benefit may relieve the mental illness, and thus increase the likelihood that the patient would be released" (10). So far as research with prisoners is concerned, the problem of coercion has been presumed to exist in all phases of the criminal justice system.

Regarding research with drug-dependent patients, there may be the presumption of mental disability, and all four issues of informed consent have been raised in different contexts. There has been a tendency in some quarters to assume that diminished capacity exists, that coercion by the researcher is likely, and that adversary procedures and some form of community consent are essential to protect the individual rights of research subjects.

The resolution of these issues will be different if primary concern is given to the health and safety of the individual and society's needs as opposed to individual rights. It is paradoxical that the case for "health and safety," "society's needs," and "individual rights" is usually argued for hypothetical individual patients and subjects by elitists who presume to know more than the patients themselves about what is "good" for them. Indeed, the struggle between objectivism, intellectualism, and the pursuit of knowledge on the one hand, and subjectivism, anti-intellectualism, and equality[1] on the other, is a political conflict with implications beyond the limits of biomedical and behavioral research. Daniel Patrick Moynihan has quoted the political philosopher Joseph Schumpeter in words that seem to have some relevance: "It is an error to believe that political attack arises primarily from grievance and that it can be turned by justification. Political criticisms cannot be met effectively by rational argument" (11).

[1] "Equality" has recently come to mean that all points of view are equally valid in any situation involving two or more persons, that the power to veto is distributed widely and across many issues, and that executive power (literally the power to execute, implement, or develop) must be reduced until progress proceeds only by consensus.

In the present climate, appeals to the need for scientific advancement will not, and are not, convincing those with power and authority to let the enterprise continue in a number of communities. The research projects that have been stopped were defended (it seems to me) in a characteristic way. The research investigators met repeatedly with their most militant adversaries. They did so at the urging of their colleagues, who let them enter these meetings without significant support. The principal investigators and their colleagues believed that reason would prevail and that they would be able to convince their most ardent opponents. At the least, they expected that they could effect a compromise that would enable them to continue to work and would enable their adversaries to feel that they, too, had accomplished their ends. The institutions that had previously supported the research now supported the mediative process, thereby invalidating the previous support of the project that had been granted by the human studies committee and other relevant structures. Committees were set up to assist in the mediation; the committees usually consisted of the more militant opponents of the research and some individuals who were deemed friendly (although not emotionally committed) to the work of the research investigators.

It is untenable for the research investigator to defend his work on his own. It is also probably untenable for the scientific community to defend itself without major support from lay organizations. Previously, lay groups, like some of their scientific counterparts, have been organized to support categorical research. This has increased the risk of "salami tactics" by opponents of research, since attacks on individual projects have not been considered a threat to broader categories of biomedical and behavioral research. A coalition of lay persons and biomedical and behavioral scientists may be essential at this juncture to present to the public, the scientific community, and legislators a concern that includes freedom of inquiry *and* subjects' rights. Subjects' rights should not be stated only in a legalistic context, but also in the traditional context of "do no harm." Scientists have a major educative role relative to the public and the surface has barely been scratched. The adversaries of science are doing much better.

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Problems in Follow-Up Studies

BY LEE N. ROBINS, PH.D.

Recent regulations concerning consent procedures and protection of privacy fall most severely on follow-up studies of children. Indeed, rigorous sample selection, nearly complete follow-up, and objective assessment of outcome are virtually impossible now. Further, compliance *with current "subjects' rights" regulations sometimes seems potentially more harmful to the subjects than is the research itself.*

RECENT REGULATIONS by the Department of Health, Education, and Welfare severely limit direct access to certain groups as research subjects (i.e., prisoners, children, and the mentally disabled). At the same time, new laws passed to protect privacy make indirect access by review of records virtually impossible for the researcher. These two changes have created a situation in which the ability to use follow-up studies as a research tool for understanding the causes and consequences of psychopathology and childhood deviance and to evaluate the effectiveness of the prevention and treatment of these problems has been seriously compromised. Indeed, most of the research tools I have used in the past 15 years to locate and evaluate the long-term outcome of child patients and normal control children have now been declared off limits to the researcher. I would like to argue that the researcher's access to a variety of records is necessary and not excessively dangerous—indeed, a good deal less dangerous to research subjects than is compliance with the new regulations.

The techniques that I have used and have encouraged other researchers to use are generally considered a good way to produce unbiased results in the very difficult area of separating the causes of deviant behavior and psychopathology from their effects. In simplest outline, to study factors in the risk of developing psychiatric disorder, the researcher chooses a general or high-risk sample from a population roster established before the age of risk for onset of the problems to be studied. He uses records or case histories dating from that early period to establish levels of familial, social, and behavioral variables that may influence the occurrence of the problem. He then follows the sample subjects after they have been at risk for the problem and evaluates their outcome by interview and record research. This method allows the researcher to 1) locate almost all of the subjects for follow-up, 2) test the importance of predictors without allowing the good or bad outcomes to bias either entry into the study or the assessment of the presence or absence of the predictors, and 3) verify the interview findings by comparing them with records. Work using these epidemiological methods has produced important new insights into the causes of psychiatric disorder and childhood problems and into the effectiveness of various treatments, but much work remains to be done.

Further research into the causes of childhood problems and mental disorders and the effectiveness of prevention and treatment is not superfluous. Those opposed to the continuation of research on children's psychiatric and emotional problems say that further research is unnecessary because the causes of children's problems are already known (they are due to lack of good schools and adequate health care, and so forth). Yet every careful evaluation of efforts to provide the services that are supposed to prevent children's problems has shown that they do not produce any significant long-term benefits. Children have not stopped developing psychiatric problems, school failure, or delinquency when they are offered early childhood educational programs, reading clinics, dental care, special education, school lunches, and counseling. Either our explanations are wrong or our remedial techniques are ineffective. It would be a pity to call a halt to the effort to understand causes and to find effective preventive and treatment techniques. The question is whether we can continue to do the necessary research without harming our research subjects.

The protection of human subjects of follow-up studies can be achieved without the severe current regulations that obviate the best kind of scientific evaluation of preventive and treatment efforts. Rigorous research on social and familial factors in psychopathology and on treatment *can* be done with the most minimal risks to the children or patients involved.

SAMPLING SOURCES

How do current regulations interfere with good research? Let us consider trying to determine the causes of deviance and psychiatric problems in children. Studies of this kind must begin with a general sample

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so that it will include children both who will and who will not develop problems. Entry into that sample should be unbiased with respect to the chance of having problems currently or in the future. This means that it should also be unbiased with respect to the chance of coming from families with problems, since we know that problems tend to be passed on within families. A treated sample is not useful because *all* of its members have problems, and thus we cannot learn how those with problems differ from those not affected. We cannot rely on comparisons of treated children and normal children because we know that treated children are not a random sample of all children with these problems. Entering treatment depends on the facilities available, social status, and family attitudes as well as on the severity of the disturbance.

The best sample to use to seek answers about causes is one selected from rosters representing a general population—rosters prepared *before* the population being investigated had any difficulties. Excellent rosters for such purposes are birth records or early school records, since the subjects' chances of appearing in these rosters will not have been affected by later psychiatric or behavior problems. Much of the research I have done has been based on random samples from elementary school records. With the passage of new laws and regulations, these records are simply no longer available.

In the course of a study of Viet Nam veterans and matched nonveterans (1), my associates and I wrote to schools located in the same zip code areas in which the veterans had lived at the time of induction, requesting the school principal to choose a control subject from among his alumni of the same age and level of education as the veteran at the date the veteran was inducted. The letter pointed out that we were *not* requesting school records. All we needed was the name, last address, and parents' names to help in locating the man in case his address was no longer current. We would then write the selected man and invite him to participate in the study. He would be free to do so or to refuse.

Most of the small town schools cooperated. Most of the big city school systems, however, declined to cooperate with our project—not because they disapproved of the study or feared that it would violate the privacy of their alumni, but because they feared that if they cooperated they would lose federal funding. One principal explained, "We know you are all right, but we can't even give out addresses from school records. After all, you might be trying to collect a bill from somebody." I pointed out that *he* was providing the name, not I, so there was no way I could be tracking down a "skip." That made sense to him, but he still did not help because there was a regulation and he could not violate that regulation regardless of the fact that my purposes were not the ones the regulation was written to protect students against.

With school and birth records unavailable as sampling sources, one alternative is to conduct area surveys. In the typical area survey, a representative sample of dwelling units is selected, the occupants enumerated, and one occupant chosen at random for interview. This is a poor choice when doing studies of psychopathology, deviance, or serious illness. In the first place, people with these problems have higher death rates than does the general population. Area surveys cover only survivors, thus omitting the most serious cases. In the second place, deviant and some psychiatrically disturbed people have lifestyles that make them relatively less accessible for such surveys. More are in hospitals and jails, and more do not have fixed addresses. The researcher is unlikely to find them at home by ringing doorbells. Thus the area surveyor gets a sample biased in favor of normal or neurotic people and loses the psychotic and socially deviant subjects.

FOLLOW-UP

The researcher needs access to records for reasons other than choosing an unbiased sample. Records permit him to assess possible causal predictors objectively, locate persons for interview, and objectively evaluate outcomes. People with problems who are interviewed about their childhoods are likely to remember selectively or to exaggerate memories that make sense of their current situation. Using records collected before their problems occurred avoids "retrospective falsification." In evaluating treatment or preventive programs, the researcher must compare persons who have and who have not had contact with them in order to determine whether their participation in the programs made a difference in the course of their illnesses.

Treated subjects and controls must be contacted sometimes years after exposure to the programs. In the past, skilled researchers have been able to find almost everyone designated. (Our own location rates have been 92–98% after 25 or 30 years.) It is important to locate almost everyone because *ease of location* is highly correlated with outcome. Subjects with the worst outcomes will be dead and thus locatable in vital statistics; subjects with the best outcomes will be listed in the telephone directory. The rest will be difficult to find. To locate subjects we used hospitals, police, drivers' license bureaus, credit rating corporations, school alumni offices, and a host of other formerly public records. All of these sources are no longer available without written permission. The difficulty with obtaining written permission is that not only is it difficult to anticipate all of the record sources for which permission ought to be sought, but permissions given are now valid for only *6 to 9 months*. Clearly, this makes long-term follow-ups impossible. A second problem is that the researcher often has no contact with his control subjects before the time he is ready to contact them to evaluate their outcomes, and thus he has had no opportunity to request their permission to examine records helpful in locating them.

STUDIES OF CHILDREN

The problem of long-term follow-up studies is particularly critical in research on children. As an example, it is important to know whether prescribing amphetamines for hyperactive children might be associated with illicit use of this or other drugs later on. The opposite possibility, and perhaps one just as likely, is that treatment of hyperactivity prevents the development of delinquent behaviors of all kinds and thus reduces the risk of drug abuse in adulthood. A reasonable study design to answer these questions calls for following hyperactive children treated with amphetamines into later life and comparing their outcomes with those of hyperactive children who did not receive drug therapy. At the present time school and police records could not be used either as address sources to help locate these children as adults or as measures of their outcomes. Permissions do not remain active long enough, and certainly a parent's permission will not be valid once the child is adult. Having to obtain permissions after the children have grown up is almost certain to mean a high rate of refusals and probably a seriously biased sample, since cooperation *after the fact* may well vary with the favorableness of the outcome.

THE FAMILY STUDY

Another valuable kind of study that has become virtually impossible under current regulations is the family study. Starting with parents with and without problems, for instance, the research wants to learn whether children separated early from a problem parent grow up to have higher problem rates than children from normal families, and whether they have lower problem rates than children reared by their problem parents. Formerly, it was not difficult to check school, hospital, and police records for the offspring to answer these questions without having to intrude on the families at all. Under current regulations this study could not be done without obtaining consent from each family member to check his records. Given this requirement, the chances of surveying the records for *all* members of a family become remote. Obtaining the consent of all family members is important if those without problems are more (or less) likely to consent than those with problems. And what if one of the offspring has been committed to an institution? Under current regulations it is not even clear that he is allowed to give his permission.

When families have split up and parents remarried, the problem of locating the child to request his consent becomes not only difficult but potentially upsetting to the people involved. We did such a study before the enactment of regulations about the privacy of records (2). We interviewed fathers and asked for the names of their wives and children. We then sought the names of the whole family in school and police records 10 years later and are now tabulating similarities between parents' and children's records under various conditions, such as when children did and did not live with the parent at critical ages. Some of these men were divorced when we first interviewed them, and many more have been divorced since then. Some of the children about whom they told us 10 years ago were illegitimate. Consider the relative risk to the people involved of what we actually did, i.e., making statistical tables from their records, versus what would have happened if we were to attempt that study now. We would have to appear at the man's home, where his current wife may not even know about his previous marriage or illegitimate children, to ask how to locate these grown children in order to seek their consent to examine their records. Future divorces might be partly *our* responsibility!

DISCUSSION

Much of the research on psychopathology and deviance for which records are needed carries no risk to the individual studied *unless* confidentiality is breached. Yet researchers are burdened with consent regulations designed to protect human beings when their participation in research requires taking potentially dangerous drugs or undergoing painful procedures. For such studies, the ability of children and the mentally disabled to consent *is* a difficult ethical question. The privacy regulations were not designed with research in mind but rather to protect persons from losing jobs or being sued when unauthorized persons read discreditable things about them in records. Unfortunately, these regulations fail to make a distinction between the researcher, who is interested in a person only as a representative of a class of persons, and employers or creditors, who are interested in him as an individual. These regulations, which are supposed to protect human subjects, have reached the point where the researcher's very *act of compliance* with the regulations while doing follow-up studies is likely to do the subjects more harm than are the research procedures themselves. Surely something is amiss.

We need to preserve access to records for research purposes; it is also important to combat the increasing tendency to destroy records. In the study previously described, in which my associates and I examined delinquency rates across generations, we waited until the children were past the age of risk of becoming delinquent before examining their records, to ensure that all cases of delinquency that would ever occur could be located. It turned out that the computer tape containing juvenile records was erased as soon as the child passed the age at which he could be arrested as a juvenile, in order to "protect" him. Luckily, one old-fashioned member of the juvenile court could not bring himself to destroy the cards on which the computer record had been based and had thrown them into the bottom of a file drawer, so the project was saved.

Such destruction of records is not simply a matter of conserving space, but has been recommended as a way of reducing the chance of records getting into the wrong hands. This view ignores the possibilities of using records for scientific as well as administrative and treatment functions. The record destroyers think they know when records have outlived their usefulness. As Dr. Otto Marx, a psychiatrist and medical historian, warned at an American Psychiatric Association conference in 1974:

> let us . . . stop the destruction of records wherever we can. The problem of confidentiality will not be solved by having no records of a confidential nature and nothing confidential in our records. . . . When fear of abuse leads to the destruction of record information, we are cheating future generations out of their heritage. Let us spend our energies on devising the means to maintain confidentiality within the new technology so that our own and future generations can benefit from technological advance (3).

Recent regulations have been offered in the name of ethics. But there are ethical problems other than those of consent and confidentiality. Sick children and the mentally disabled have a right to treatments and preventive health measures of proven effectiveness so that their lives and futures will be safe. There is no way to be sure that new treatments or preventive efforts are effective and safe without rigorous research and long-term follow-up studies. The issue of confidentiality is not insuperable. Researchers must be required not to disclose data about individuals, but most researchers want very much to preserve the privacy of the subjects who make their research possible. There are already a number of rather simple methods to do so. Let us find ways of protecting privacy without depriving future generations of the knowledge that could spare them unnecessary anguish.

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The Functions of Human Subjects Review Committees

BY BRADFORD H. GRAY, PH.D.

After summarizing the literature on the functioning of committees set up to review research involving human subjects, the author describes three latent or emerging functions of these committees. These include using the committees to protect institutions, to judge proposed research in terms of possible social policy impact, and to consider proposed research from the standpoint of "community acceptability." The author expresses concern about the possible politicization of review committee decisions.

WE ARE ALL AWARE of the great interest that has developed in recent years in the complex issues raised by research involving human subjects. Many slips can take place in the application of general principles or guidelines to particular situations. Thus there is general agreement—reflected in the regulations of the U.S. Department of Health, Education, and Welfare—that when a research project entails risk to subjects the judgment of the investigator regarding the rights and welfare of subjects should be reviewed by people who have no vested interest in the research. However, very little has been written about the specific mechanism—human subjects review committees—that has been invented to play this role. The literature contains some information on the background and development of

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this regulatory approach (1, 2), descriptions of the procedures and problems of a few committees (3–6), and some limited empirical studies of such committees (7–10).

Rather than discussing the well-known duties of human subjects review committees as set forth in DHEW regulations (see appendix 1), I will examine some functions that might be described as unintended, latent, or emerging. It might be useful to begin with a brief summary of what we know about human subjects review committees, although the committees whose experience has been shared in the literature hardly comprise a representative sample. (This and many other weaknesses of earlier studies should be corrected in a study that is under way for the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research.[1])

The literature suggests that the major issues being dealt with by review committees pertain to the risks of research and to informed consent; I believe that the latter is often dealt with too narrowly by focusing on consent forms and overlooking the setting in which consent is obtained (11). Melmon and associates (5) reported that the medical school at the University of California, San Francisco, found agreement among independent subcommittees in the rejection of a small number of proposals on the basis of excessive risk or inadequately evaluated risk. I know of no other information on the reliability of committee decisions.

Disagreement has been reported between committees and investigators over the legitimacy of review committees being concerned with adequacy of scientific design. It appears that in some cases investigators are beginning to accept the view that faulty research design is a legitimate matter of concern to human subjects review committees, particularly when subjects are to be put at risk, but this remains a thorny topic.

Finally, to conclude this brief survey of what review committees do, I should mention two problem areas. First, although it is often cited as being very important, continuing review or monitoring is not very extensively developed in either regulations or practice. There seems to be little general agreement on what sort of activity by a review committee would be appropriate in this area. The second problem is suggested by anecdotal reports of special difficulties created when research from one field is reviewed by a review committee accustomed to different research models. This indicates that there may be too much rigidity in carrying out the review function at some institutions.

One must be very careful in talking in evaluative terms about the functioning of review committees because there is no absolute standard against which to measure performance. Elsewhere (8) I reviewed the evidence for the impact of human subjects review committees. For this study my primary sources of data were the survey by Barber and associates of almost 300 review committees (7) and my own intensive study of a single review committee (11). My conclusions were not reassuring with regard to the general adequacy of the performance of review committees. However, I would prefer not to go into those data again, both because they are fairly well known and because they are several years old. The results of the study currently under way for the National Commission for the Protection of Human Subjects should enable us to discuss these matters more intelligently.

If one judges from what review committee members have said and written elsewhere, it appears that at least some committees have been growing in sophistication about the issues involved in human experimentation, in the stringency of their review, and, interestingly, in their acceptance by investigators. It is difficult to say whether their acceptance by investigators reflects a positive response to being helped with difficult issues, a perception that their interests are being protected, or simple resignation in the face of an unavoidable intrusion.

There seems to be general agreement now that review committees have a proper and important role in research involving human subjects. Current discussions tend to be about how they can be improved rather than about whether they are legitimate. The legitimacy of these committees is an important problem that is central to their successful functioning. The committees must have legitimacy in the eyes of investigators if compliance with the spirit of their activities is to be expected. There must also be legitimacy in the eyes of outsiders, whether they be the general public or legislative bodies, if current doubts about the propriety of research involving human subjects are to subside.

It is from the perspective of a belief in both the importance of the role of review committees in protecting human subjects and the importance of a high degree of legitimacy in the eyes of investigators and the public that I want to express some concerns about three unintended, latent, or emerging functions of these committees.

UNINTENDED FUNCTIONS OF REVIEW COMMITTEES

My first concern pertains to the uses to which the review procedure is put. A safe assumption with any control procedure in a bureaucratic structure is that it will tend to be used for purposes other than that for which it was designed. The review procedure can be used for its manifest purpose of protecting human subjects, but it can also be used to protect investigators or to protect institutions. These functions are not necessarily inconsistent: true protection of subjects would also protect the investigator and the institution. However, actions that protect the investigator or the insti-

[1]Although I have been closely associated with this study, which is being conducted under contract by the Survey Research Center at the University of Michigan, this paper was presented before the completion of the study and is not based on it.

tution may have little to do with protection of human subjects. (There is an element of this in the use of consent forms in some instances.)

In universities, the research activities of faculty have in the past been under rather minimal control of the institutional administration, but there are countervailing "rationalizing" pressures to curtail such independence. It would be understandable if institutional officials used their influence on or control over review committees to have them consider proposals from the standpoint of what is to be gained or lost by the institution if the research is done. This may have little to do with the protection of human subjects and a great deal to do with avoiding controversy. The extent to which such criteria are now applied to proposals is unknown; only anecdotal evidence is available. This problem, however, relates more to the way that review procedures are made operational than to their theory or concept and could perhaps be controlled through appeal procedures.

My second concern pertains to whether review committees should consider in their deliberations the implications of proposed research, particularly their social policy implications. Stated another way, should review committees weigh, as a negative factor, the uses to which the results of research might be put? This issue has been labeled the social risk issue because the words "social risk" in DHEW regulations have on occasion been interpreted as requiring or legitimating the consideration of such factors by review committees. In two situations in recent years such criteria were applied in National Institute of Mental Health study sections and produced a flurry of spirited correspondence. Although letters of clarification were eventually sent from NIMH and the National Institutes of Health's Office for Protection from Research Risks stating that study sections had no authority to apply such criteria, the letters indicated that there is considerable pressure to enter such considerations into the review process.

It is obvious that the application of such criteria would in many (perhaps all) situations be antithetical to established conceptions of free and open inquiry. Thus it requires justification. (This, of course, does not suggest that such considerations should not enter into decisions regarding allocation of public funds.) In addition, there is no reason why such social consequences of research should be considered only if human subjects are involved. Put another way, there is no reason why such considerations should enter into the human subjects review process, even if a public policy decision were to be made that research should be done only if the anticipated consequences of the study have some proper risk: benefit ratio. Such consequences have nothing to do with the presence or absence of human subjects in the research.

The third emerging function I wish to raise is actually found in DHEW regulations, although its meaning there is not very clear. The regulations require that the membership of human subjects review committees be able to determine whether research is acceptable in terms of "community attitudes." Over the past 10 years there has been a trend in the regulation of human experimentation away from a peer review model toward a review model that includes more lay representation. Robert Veatch, a bioethicist, characterized this as a movement from a professional model toward a jury or representative model (12). Veatch argued that confusion exists over what human subjects review committees are supposed to do. Certain tasks call for professional expertise and suggest one type of committee composition. On the other hand, there is, in Veatch's words, a "growing recognition" that committees must "have the ability to judge community attitudes and community acceptance," tasks that call for a membership that is representative of the community. Veatch did not examine the desirability of these developments.

As far as I can determine, the idea that human subjects review committees should judge whether research is acceptable in the light of community attitudes and the implications that follow from this idea have received remarkably little serious comment and analysis. The purpose of community representation on review committees is not clear. DHEW regulations, for example, do not specify which judgments should involve community attitudes; this leaves doubt about whether a shift has occurred in the functions review committees are to serve. If the function of community representatives is to keep the process honest, to look at consent procedures from a layperson's perspective, and to broaden the viewpoint from which judgments of acceptability of risks and benefits are made, no shift may be involved and lay representation may strengthen the process. However, if the function of community representation is to decide whether a community would find the study immoral, to judge the acceptability of policy implications of proposed research, or to determine whether the interests of certain organizations or groups are affected by the research, then a shift will indeed be involved. These decisions are essentially political, and their inclusion in the human subjects review process would fundamentally change its nature and purpose.

CONCLUSIONS

The politicization of human subjects review procedures may be inevitable, but it may weaken their effectiveness in functions related to the protection of human subjects, either by drawing attention away from human subjects issues or by reducing the legitimacy of review procedures with investigators. Thus there is reason for concern about developments that move the process in the direction of a political model of review, whether the impetus comes from the government or from the research community itself, which may seek to use the process to defend the researcher who has become involved in controversy.

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APPENDIX 1
U.S. Department of Health, Education, and Welfare Regulations on Human Subjects Review Committees

The DHEW regulations (45 CFR 46) require that the following determinations be made by review committees:

[The] review shall determine whether these subjects will be placed at risk, and, if risk is involved, whether:

(1) the risks to the subject are so outweighed by the sum of the benefit to the subject and the importance of the knowledge to be gained as to warrant a decision to allow the subject to accept these risks;

(2) the rights and welfare of any such subjects will be adequately protected;

(3) legally effective informed consent will be obtained by adequate and appropriate methods in accordance with the provisions of this part; and

(4) the conduct of the activity will be reviewed at timely intervals.

Federal Constraints: Earned or Unearned?

BY DONALD T. CHALKLEY, PH.D.

The author discusses the evolution of federal constraints on medical, behavioral, and social science research. There has been only one court decision related to behavioral research and none in medical research. The burden of consent procedures can be lightened somewhat by careful consideration of the potential risks and nature of the research; questions are presented that can be used to determine whether constraints apply. The author notes that although there are good reasons for regulations in both behavioral and medical research, the appropriateness of current and proposed constraints is still a matter of debate.

IN THE ACCOUNT that follows, I have attempted to be factual, with no imputations of profession-wide insensitivity, lack of ethical probity, or actual guilt. No court could convict any of the research professions as a whole on the basis of the few examples of individual misconduct that have disgraced the pages of investigational history. In the 200-year history of medicine in the United States, no physician has ever been found guilty of "malresearch," although malpractice decisions number in the few thousands annually. In fact, there has been but one malresearch decision under all of English common law. This case, *Halushka v. University of Saskatchewan* (1), involved a student who volunteered for a trial of a new anesthetic.

There has been only one decision in the behavioral sciences, but this was a United States case in the Federal Court for the Eastern District of Pennsylvania. The case, *Merriken v. Cressman* (2), has medical overtones since it was concerned with drug usage, but the research techniques were purely behavioral.

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Editor's Note: All of the preceding papers in this Special Section were presented at the 1976 annual meeting of the American Psychiatric Association in a session titled "Freedom of Inquiry and Subjects' Rights." In the interest of a balanced presentation, the Editor invited Donald Chalkley, Ph.D., Director of the Office for Protection from Research Risks, National Institutes of Health, to write a paper presenting some background information about why constraints on behavioral science have been imposed by the government.

Michael Merriken was an eighth-grade student in Norristown, Pa., and Wilmer D. Cressman was head of the Montgomery County, Pa., Board of Commissioners. The Board planned to introduce a drug addiction prevention program to aid the local school district in the identification of potential abusers and "to change the cognitive and affective domains of potential drug abusers and other forms of deviant behavior." Candidates for the program were to be identified by questionnaire, and student participation was to be required in the absence of positive parental objection. Many of the questions were directed toward familiar and sensitive issues: religion, race or skin color, political differences, differences in aggressive tendencies, unusual or odd behavior, and so on. The information was to be compiled in a "massive data bank" available to school employees, social workers, PTA officers, and school board members.

The Court's opinion discusses Constitutional guarantees of privacy, noting that "the right to privacy is on an equal or possibly more elevated pedestal than some other individual Constitutional rights and should be treated with as much deference as free speech." The "right to investigate" is sometimes equated with free speech, so this comment by the Court takes on an unusual significance. The Court then turns to the issue of consent, emphasizing that "in the case at Bar, the children are never given the opportunity to consent to invasion of their privacy; only the opportunity to refuse to consent by returning a blank questionnaire. . . . Whether this procedure is Constitutional is questionable. . . ." Citing the Supreme Court in *Brady v. United States* (3), "Waivers of constitutional rights not only must be voluntary but also must be knowing, intelligent and done with sufficient awareness of the relevant circumstances or likely consequences," the Pennsylvania Court concluded that attempts by the defendants to obtain consent would have been adequate if they had been comparable to the consent "ideally obtained by a physician prior to the performance of surgery," which does not quite equate the risks of behavioral research with those of clinical investigation but comes uncomfortably close to it.

Despite its potential significance in the hands of a plaintiff's attorney, *Merriken v. Cressman* has had little impact so far on the development of policy, regulation, and law with respect to behavioral research. Historically, concerns appear to have arisen out of public clamor that is often communicated through the Congress in terms of statements to the media, interaction with federal agencies, or the passage of legisla-

tion. The original version of the present Department of Health, Education, and Welfare policy, now codified as Part 46 of Title 45 of the Code of Federal Regulations, was a Public Health Service policy document, "Clinical Investigations Using Human Subjects" (Policy and Procedure Order 129, dated February 8, 1966) that was concerned solely with clinical investigation. On February 9, 1966, Representative Cornelius Gallagher, Chairman of the Special Subcommittee on Invasion of Privacy, wrote to Philip R. Lee, M.D., Assistant Secretary for Health and Scientific Affairs, asking for documentation of an earlier assurance by the Public Health Service that any federally supported research project using personality tests, inventories, or questionnaires would be done only with informed consent by or on behalf of the subject. Similar assurances had also been sought by Representative Reuss of the Committee on Government Operations (4). This, among other matters, led to a revised version of Policy and Procedure Order 129 on July 16, 1966, which made the order broadly applicable to all investigations involving human subjects, including clinical research. On September 13, the involvement of nonclinical investigations was made even more explicit by the issuance of a supplement entitled "Considerations in the Behavioral Sciences." This was followed on December 12, 1966, by a clarifying memorandum reflecting discussions with the American Psychological Association and other organizations representing the behavioral sciences. Much of the language of this memorandum was later incorporated into DHEW policy published in 1971 (5).

Here the matter stood until 1973. In the latter part of the 93rd Congress, unfavorable publicity surrounding the so-called Tuskegee case (6) led to a rash of Senate bills designed to establish controls on human experimentation. One of these bills (Senate Bill 878) dealt with human experimentation generally, another (Senate Bill 934) was concerned solely with medical experimentation, and a third (Senate Bill 2072) was designed to "assure maximum protection for human subjects of biomedical and behavioral research." This phrase was incorporated into the title of the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research, established by the National Research Act (P.L. 93-348).

The foundation of behavioral studies against which Representatives Gallagher and Reuss and Senator Edward Kennedy based their concerns is none too consistent. Committee prints issued by Representative Gallagher's special subcommittee in 1965 (7) are concerned principally with data gathering and the privacy issue. The 1967 Reuss committee was concerned with social science studies in such areas as crime and law enforcement, education, poverty, welfare, and social aspects of medicine and health (8). The latter specifically touched on behavioral research supported by NIH and NIMH. In 1970, Representative Gallagher explored the use of drugs in treating hyperkinesis (9). The Kennedy hearings touched on behavioral issues only peripherally in connection with psychosurgery and drug use (10). Separately, none of these hearings or studies provided a broad basis of fact for regulation of the behavioral sciences.

There is, however, reason to believe that such evidence could have been found had these hearings and studies been designed to look for it. A continuing study of the confidentiality of social science research sources and data sponsored by the Russell Sage Foundation[1] has identified a substantial number of cases involving advertent and inadvertent breaches of confidentiality by researchers in the behavioral sciences. Most of the cases cited are abstracted from the American Psychological Association's publication "Ethical Principles in the Conduct of Research with Human Participants" (11). Many are clearly recognizable and one could identify the institution and persons involved, but the implicit confidentiality of the study and of the "Principles" should be honored. As one member of the confidentiality study staff noted (12), "Not all involve litigation . . . although most of the nonjudicial cases *could* have involved such interests" (emphasis in the original).

These case citations are indefinite as to time, type of institution, nature of support, and prior collegial or review board consideration. A more general source of evidence on the frequency of problems involving human subjects can be found in the files of the NIH Office for Protection from Research Risks. For calendar 1976, some 15,437 applications were referred to NIMH and NIH for review at the March, June, and October National Advisory Council meetings. Of the 12,988 referred to NIH, 265 (2%) were criticized by reviewers in terms of the proposed handling of human subjects; 62 of the 2,449 applications referred to NIMH (2.5%) were similarly criticized. The difference is not statistically significant.

This is, of course, a soft statistic. There are differences in approval rates by institutions, in sensitivity to problems (a group of oncologists is undoubtedly less sensitive to certain issues in the use of narcotics to create euphoria in cancer patients than would be a group of psychopharmacologists), and in the types of issues raised. In addition, NIMH reviewers appear to be far more sensitive and active with respect to criticisms of informed consent procedures. Nevertheless, it is interesting that judgment of the problems in their respective fields by separate if not distinct groups of peers has resulted in about the same level of criticism.

There is a principle of the law that states simply *respondeat superior*—literally, "let the master answer," which originated in a challenge in the Roman courts to a master to account for the wrongful acts of his ser-

[1]This information is contained in an unpublished February 25, 1975, progress report to the Russell Sage Foundation of the American Political Science Association study "Confidentiality of Social Sciences Research Sources and Data." It should be noted that most of the publications emerging from this study have been concerned with the problems encountered by investigators in trying to protect confidentiality.

vants. Today it establishes the responsibility in certain circumstances of an employer, i.e., an institution, for the acts of its employees, staff, or faculty. The original Public Health Service policy, laid down in 1966 and continued roughly through 1973, established a formal system of institutional review boards to assure that this responsibility was recognized when an institution proposed to apply for a research grant or responded to a request for a contract proposal. The areas of concern were spelled out, the institution was required to develop or adopt its own set of principles and to adhere to its own regulation, to standards of professional practice, and to the applicable law, and to be alert to the concerns of its community. Details were sparse.

Unfortunately, old policies seldom die—they just become more complex. Initially, the lack of a formal position on research involving human subjects was looked on as a missing link in our policy chain. Once that link was forged, it was immediately evident that there were now missing links between that link and the rest of the chain—links with respect to different classes of subjects, different types of experimentation, etc. Each critic can find a link absent in his immediate area of concern; each investigator can find a reason why his immediate area of research should not be unduly restricted. The potential beneficiary of research can only wait for the resolution of the dispute.

The existing regulations were designed to permit institutional review boards a substantial amount of latitude. Before assuming the "constraints," any board should ask itself several questions, some of which are listed below.

1. Is the project really a research and development proposal? Or is it simply a service or other activity using techniques commonly associated with behavioral research but not intended in a particular instance to serve a research function? If the answer to the first question is no, the regulations do not apply.

2. Does the project involve risk? If the project involves the analysis of data already in existence that cannot be linked to personal identifiers, if it involves public observation of unidentified and unidentifiable persons, if the methods are entirely standard and accepted and applied solely to meet the needs of the subject—even if supported by research funds—no risk is involved and the review can be curtailed.

3. Does the project involve only minimal risk? Would full written or oral consent invalidate the study's objectives? Would any alternative means for obtaining these objectives be disadvantageous to the subject? If these conditions apply, the consent requirements can be modified or omitted altogether. Certainly, in many behavioral studies in the areas of psychosomatic medicine, clinical psychology, etc., these conditions do apply, with a consequent reduction in the burden of consent.

There are as many good reasons for constraints in behavioral research as there are in medicine. Whether the constraints currently imposed or to be imposed in the future on either field of investigation are truly justified is a matter for continuing debate. At the moment, we are busy dismantling the wall of protection we seek to build around those in need of the benefits of research while we devote much time and effort to erecting protections for the subject. The time and effort of any person is limited. Some compromise may be necessary.

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Effect of Diet on Urinary MHPG Excretion in Depressed Patients and Normal Control Subjects

BY GIOVANNI MUSCETTOLA, M.D., THOMAS WEHR, M.D., AND FREDERICK K. GOODWIN, M.D.

The authors studied the effect of a catecholamine-controlled diet on the urinary level of 3-methoxy-4-hydroxyphenylglycol (MHPG) of 6 depressed patients and 6 normal volunteers. The normal subjects showed no change in MHPG levels on or off the diet; the depressed patients showed a significant increase in MHPG while off the diet.

THE PURPOSE of this study was to examine the effect of dietary alterations on the urinary excretion of 3-methoxy-4-hydroxyphenylglycol (MHPG), an important norepinephrine (NE) metabolite, in depressed patients and in normal control subjects. Initially on the basis of indirect evidence suggesting the involvement of NE in the mechanisms of action of mood-altering drugs, it has been hypothesized that NE, a catecholamine neurotransmitter, may be involved in the pathophysiology of major affective disorders (1–3). To evaluate this hypothesis directly in patients, a number of groups have studied the urinary excretion of amine metabolites, principally that of MHPG, the major metabolite of NE in brain. A clinical study from our laboratory using isotope dilution techniques indicated that most of the MHPG in urine originates from brain NE metabolism, whereas virtually all of the 3-methoxy-4-hydroxymandelic acid (VMA), the other major metabolite of NE in urine, comes from the periphery (4).

In studies of affective disorders, one group (5) reported that urinary MHPG, but not VMA, was low in patients with endogenous depression compared with control subjects; another group (6) did not find this difference. Another study (7) reported that urinary MHPG, but not VMA, increased on the patient's recovery from depression. In addition, MHPG has been reported to be higher in the manic phase than in the depressed phase of bipolar affective illness, although there is disagreement as to whether the increase in MHPG precedes or follows the onset of mania (7–11). Because of these discrepancies and also because of the possibility of subgroups with different patterns of MHPG excretion (6, 12, 13), it has become important to evaluate such sources of variance as diet (14).

A variety of foods, particularly some fruits and vegetables, are known to contain preformed catecholamines (15–17). Although a 1959 study showed that ingestion of large amounts of bananas produced an increase in total urinary catecholamines in normal control subjects (18), subsequent studies in normal subjects have indicated negligible dietary influences (17, 19–21). To our knowledge the effect of diet on urinary catecholamine metabolites in depressed patients has not been reported.

METHOD

Six depressed patients (5 women and 1 man) and 6 normal volunteers (all women) were studied on a metabolic research ward at the National Institute of Mental Health. All of the subjects adhered to a special low-monoamine diet (see appendix 1) from the time of their admission; after at least 3 weeks all of the subjects were taken off this diet for 7 days, during which time they were encouraged to eat the previously restricted foods (with the exception of alcohol, which was excluded from the entire study). Following this off-diet period they were returned to the low-monoamine diet. Both diets were specially prepared and supervised by a trained National Institutes of Health dietician. The 6

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TABLE 1
Effect of Diet on Urinary MHPG in Depressed Patients: Individual Patient Data

Patient	Age	Sex	Diagnosis	Drug Treatment	Urinary MHPG (mg per 24 hours)								
					On Diet			Off Diet			Diet Reinstituted		
					N*	Mean ± SEM		N*	Mean ± SEM		N*	Mean ± SEM	
4129	18	Female	Unipolar	Placebo	5	1.24	.08	7	2.01	.12	4	1.40	.09
4130	42	Female	Bipolar I	Haloperidol; imipramine	4	1.61	.14	5	2.05	.05	1	2.16	
4134	39	Female	Bipolar I	Lithium	4	1.10	.13	5	1.85	.38	1	1.10	
4135	22	Female	Bipolar I	Chlorpromazine; lithium	5	1.12	.15	6	2.04	.14	4	1.44	.17
4137	24	Female	Schizo-affective	Chlorpromazine	4	1.78	.24	6	2.95	.16	2	1.87	.30
4138	33	Male	Bipolar I	Lithium	3	1.92	.43	7	3.48	.06	4	2.76	.12
Overall mean						1.66	.14		2.40	.27		1.79	.25

*Number of 24-hour samples.

normal volunteers were free of psychiatric symptoms or history. Of the patients, 5 were moderately depressed and 1 was severely depressed. One patient was receiving a placebo, and 5 were receiving various drugs as indicated in table 1; no medication changes were made during the study. Global ratings of depression, anxiety, psychosis, anger, and mania were obtained twice daily by consensus of a nursing research team using a modified Bunney-Hamburg Scale (22).

Twenty-four-hour urine samples were collected throughout the study. Completeness of urine collection was monitored by a urine loss recording system; any questionable collection was discarded. Urine was refrigerated within 30 minutes of voiding, and aliquots were frozen at −20° C within 3 days; urinary MHPG has been shown to be stable under these conditions (unpublished observations by F.K.G. and C.C. Jones). Samples were selected from 3 to 5 days during the week preceding the change in diet, 5 to 7 days during the period off the diet, and 1 to 4 days during the week after resumption of the low-monoamine diet. MHPG was analyzed by electron capture gas liquid chromatograph using a modification of the method of Dekirmenjian and Maas (23).

RESULTS

No overt behavioral or mood changes could be detected in either group during the period of the study, either as reflected in the global ratings or by clinical observation. The MHPG data for individual patients are shown in table 1, and the overall results are summarized in figure 1. For the normal control subjects there was no change in urinary MHPG during the period off the controlled diet. On the other hand, the mean urinary MHPG in the depressed patients increased by 80% during the off-diet period ($p<.01$ by paired t test). Two typical patterns of daily MHPG excretion throughout the study are shown in figure 2. There was a large variability in the time course of the individual patients' responses to discontinuation of the low-monoamine diet; 2 patients showed a dramatic increase after only 24 hours on the high-monoamine diet, while 3 other patients took a longer period of time to show this increase. When the low-monoamine diet was reinstated, the urinary excretion of MHPG of all patients returned to baseline levels in 1 to 3 days.

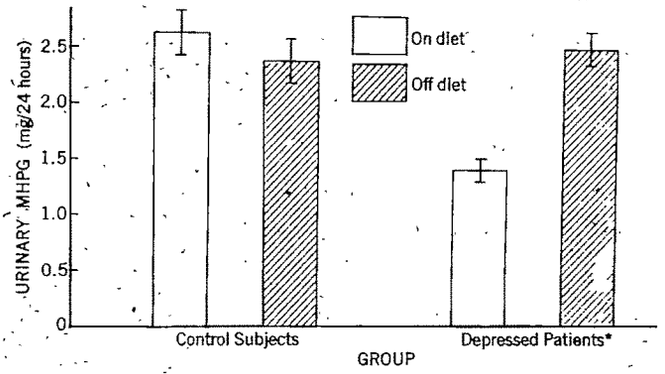
DISCUSSION

It is obvious that the amount and kind of food intake as well as the ability to cooperate with dietary restrictions can vary widely in different phases of affective illness in the same patient. Individual patients may also vary considerably in the duration and severity of appetite disturbances associated with their illness. Therefore, state-dependent differences in urinary MHPG excretion reported to exist in affective disorder must be cautiously interpreted until the relative importance of variations in monoamine ingestion—even in patients on restricted diets—has been determined.

The unique responsiveness of urinary MHPG to changes in dietary amines in depressed patients compared with normal control subjects is intriguing; we have no satisfactory explanation for it. Behavioral factors thought to be associated with MHPG excretion, such as mood state and anxiety, did not appear to change during the course of the study. However, we cannot exclude the possibility that the dietary change had a different meaning for the patients than it did for the control subjects; there may have been some subtle alteration in the patients' emotional state accompanying the experience of being off the diet. Alternatively, we might hypothesize that these patients have some impairment in noradrenergic "buffering" mechanisms, rendering them more vulnerable to dietary changes.

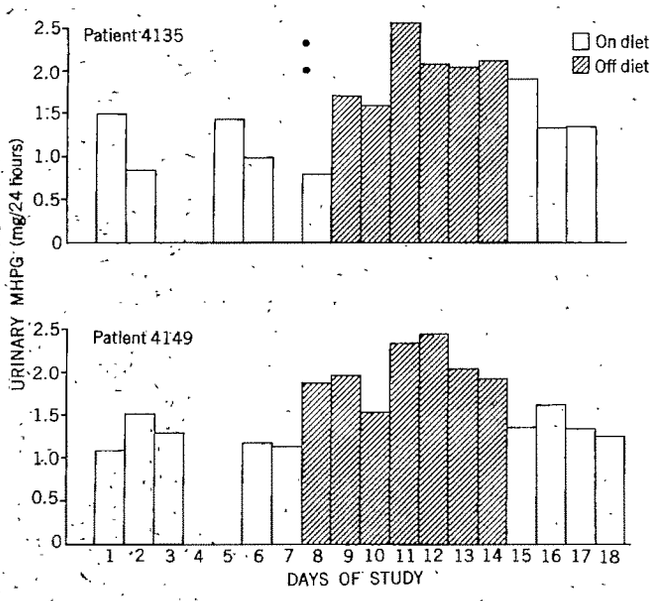
Whatever the explanation we are left with the conclusion that normal control subjects may not be suitable for evaluating the contribution of some potential

FIGURE 1
Effect of Discontinuation of Catecholamine-Controlled Diet on Urinary MHPG of 6 Depressed Patients and 6 Control Subjects



* The difference between MHPG levels on and off the diet is significant for patients ($p<.01$ by paired t test).

FIGURE 2
Effect of Discontinuation of Catecholamine-Controlled Diet on Urinary MHPG in 2 Depressed Patients



sources of variance in amine metabolite data in patients whose amine responses may not be "normal."

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APPENDIX 1
Low-Monoamine Diet

Excluded beverages: chocolate, cocoa, wine, beer, alcohol, orange juice

Excluded fruits and nuts: banana, avocado, pineapple, plum, prune, orange, walnut, raisin, fig

Excluded vegetables: tomato, broad bean, eggplant

Excluded meats, dairy products, and fish: chicken liver, herring, smoked fish, brain, aged cheese, sour cream

Community Psychiatry in Tutuila, American Samoa

BY WILLIAM E. WALTERS, M.D.

The author hypothesizes that the underreporting of mental illness in Tutuila, American Samoa, is a direct result of the social system, which provides a means of "curing" emotional disorder by family group process and shamanistic ritual, by making the disorder less disruptive, and by absolving the affected individual of personal guilt.

REFERRING TO MENTAL HEALTH problems in developing countries, Ari Kiev (1) said that what is needed is practical research on such questions as why patients with recognizable disorders fail to obtain available treatment and what makes indigenous treatments so effective. These questions have plagued the mental health staff of the LBJ Tropical Medical Center in Tutuila, American Samoa. According to the size of the population on this island (30,000 people) we should expect much more mental illness, but the mental health clinic's patients load remains light (see table 1). In an attempt to qualitatively identify the reasons for the population's underuse of mental health facilities we examined two factors: 1) family life, which is the sociocultural setting of all activity in Samoa, and 2) bush medicine, the traditional system involving belief in ancestor spirit (*aitu*) causation of most illness, a folk healer, or *taulasea*, and treatments that involve family prayer and certain herb remedies combined with the *taulasea's* intercession with the *aitu*, or spirit.

The basic sociocultural unit in Samoa is the *aiga*, or family. Samoans live in extended families of 6 to 30 members. Living space is crowded, and young people are aware of the facts of life early. Given this setting, one might think that there would be an accentuation of oedipal conflict and problems involving overclose, incestuous sibships. However, the large family group, in contrast to the Western nuclear family, seems to defuse the oedipal conflict. Brother-sister incest is not common in Samoa.

Fifty years ago Margaret Mead (3) noted that there were few cases of social unrest or mental problems in Samoa, except among people separated from their family by the stress of Western culture. Statistics are incomplete and unreliable, but the best indications point toward the fact that the major psychoses occur in about the same frequency in Samoa as they do in the Western world and in primitive cultures. This, despite rapid acculturation of Samoa to Western society, is consistent with Dunham's hypothesis concerning the independence of major psychoses from cultural variables (4).

The second feature of Samoan society that militates against recognizing mental illness and blurs the reporting of such problems as obsessional and hysterical neuroses is the concept of causation inherent in the *aitu* belief, a kind of ancestor worship holding that spirits return to haunt particular locations, where they may enter the bodies of unsuspecting victims. The offense to the spirit does not involve individual guilt. Rather, the family of the person who is stricken has offended the spirit. Thus the son or daughter may suffer for the transgressions of an elder, or *matai*.

This particular view of spiritual or mental trouble as a family problem has two important consequences: 1) it unites the family in support of the affected individual with a view toward expiating the offense and breaking the spell of the *aitu* through family prayer and visiting the *taulasea*, and 2) it offers a kind of built-in confessional whereby the guilt associated with mental illness is diffused over the entire family. The responsibility for disturbed behavior is removed from the patient and ascribed to the whim of an angry *aitu*.

Thus etiology, treatment, and prognosis of mental illness are all viewed in the context of family problems. Mental illness is a strange concept to Samoans; the disturbed person is seen as troubled rather than sick. The three following case examples illustrate Samoan attitudes.

CASE REPORTS

Case 1. A 15-year-old Samoan girl from a large family was brought to the medical center's emergency room because she had taken an overdose of 20 acetaminophen tablets. Her family disapproved of her 18-year-old boyfriend; her mother had forced him to leave the house, which resulted in a violent argument in which she struck the girl. The girl was very angry with her mother and developed a sudden headache. At the clinic the girl said that she took the overdose to rid herself of the pain.

The girl told us that this was not her first bad headache. She had been beaten by her brother in a similar quarrel two months before this episode and had been very angry at him. At that time she was seen by the *taulasea* for her head pain. He interpreted that the *aitu* of two girls who had died in a family feud had infected her. The *aitu's* name was

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TABLE 1
Comparison of Projected Versus Actual Number of Mentally Ill Patients Seen in Tutuila

Diagnosis	Projected Number of Patients with Psychiatric Diagnoses 1973–1974*	Number of Patients Seen at Mental Health Clinic 1973–1975
Character disorder, personality disorder, adjustment reaction	366	105
Victim of child abuse	—	56
Neurosis, psychophysiological reaction	1,832	99
Schizophrenia, other psychosis	595	52
Mental retardation, organic brain syndrome	275	42
Alcoholism	137	6
Major affective disorder	320	—
Other, undiagnosed, deferred diagnosis	—	56

*According to the statistical survey of the South Pacific Commission, cited in reference (2).

Sauma'iafe. In order to expunge the spirit, he said, it would be necessary for the girl to drink some medicine and for relatives to pray over her. This the family had done. However, after the girl took the overdose of acetaminophen, the family concluded that she had not profited by their treatment. They had a large meeting and determined that the girl should be beaten for her recalcitrance. She determined to accept her fate.

Case 2. A young Samoan woman had been hearing alien voices for about two years; she had tried to ignore them. At the time she first heard the voices she was living in Michigan with her husband. When the voices became threatening, she phoned her mother, who went to Michigan and brought her back to the island. Here they consulted a *taulasea*, who prescribed that the bark of *fue sina* should be mixed with water and rubbed on the patient's body. The diagnosis was *ma'i ole mafaufau*, or acute psychosis caused by an *aitu*.

After undergoing the prescribed treatment the patient became worse; she was convinced that the *taulasea* was part of a plot against her. At that point her sister urged her to visit the medical center. When we first saw her this patient was delusional with very fragmented thinking, ambivalence, and a sly, fugitive manner. After a brief hospital stay and intensive use of chlorpromazine and fluphenazine, she was able to speak coherently. She showed marked deference to her father and the *matai* system but denied any belief in *aitu*. She wanted to return to her husband in Michigan but expressed great fear about leaving her family in Samoa.

Case 3. A young, single Samoan man was brought to the medical center by his brother. The patient had become ill two years before, while he was living in San Francisco. His present symptoms consisted of a wandering life-style, attempting to find "the answer," praying a great deal, and having the delusion that he was the messenger of strength, wisdom, or purity. He exhibited a flattened affect, showed thought blocking, and stared into space. He appeared in the interview to be hearing something and admitted to an auditory hallucination, coming from the Lord, which admonished him to see the truth.

Before this patient was brought to the medical center his family had consulted a *taulasea*, who pronounced him afflicted with an *aitu*. There had been a family council in which the *matai* adjured the village to admit which individual had cast the spell. The patient continued to exhibit religious delusions and was brought to the pastor, an important man in the village. This man recognized the symptoms of schizophrenia and suggested medical care. The family seemed rather cynical about the religious content of the delusion, and the patient was universally regarded as *ma'i ole mafaufau*.

A HYPOTHESIS

In response to Kiev's questions regarding why some patients with recognizable disorders fail to obtain available treatments in developing countries I would like to offer the hypothesis that these patients are often sheltered by their family and that there are cultural sanctions for some types of mental illness. According to 1973–1974 statistics gathered by the LBJ Tropical Medical Center, an estimated 595 cases of schizophrenia were projected for our island of 30,000 inhabitants (see table 1). This figure contrasts with an incidence of 34 cases of schizophrenia and 18 of other psychoses seen in the mental health clinic during 1973–1975. McCuddin (5) indicated that patterns of referral were often from one *taulasea* to another rather than to the medical center.

The fact that the *taulasea* is able to effect a cure when modern methods fail to is traceable to the cathartic effect and group spirit aspect of the *aitu* cure. The group ritual is also practiced in exorcism, as noted by Lambert in Samoa (6) and by León in Colombia (7). Garrison (8) also noted the universality of primitive medicine and ascribed its success to group cohesion and social expectations. Another aspect of this success is intrapsychic; the patient might possibly identify with the power and magic of the shaman's personality. Several treatments are usually necessary in this kind of medicine. The cure is often obtained by the combination of coercion and group support and abetted by the suggestibility of the patient. Thus the patient may remain symptomatic but nondisruptive, as in case 3.

CONCLUSIONS

The underreporting of mental illness in primitive cultures noted by many authors is present in the developing society of American Samoa. This is a direct result of the social system, which tends to view underperformance and quiet insanity with calm disdain. The success of the *taulasea*, or bush doctor, in treating mental disorders can be attributed to the group process, the cathartic effect of the patient's participation in family prayer, the absolution of guilt, and faith in the magic of the healer.

An interesting unanswered question remains: Was

the gentle, stable, and permissive culture of old Samoa less productive of mental symptoms than our Western culture? Incidence studies (8, 9) and records of our medical center would so indicate. In 1973–1975 the medical center admitted 24 native Samoan patients with a final diagnosis of psychosis. Twenty of these patients were schizophrenic (12 were catatonic or hebephrenic, 2 were simple, chronic, and undifferentiated, and 6 were paranoid). Two patients had dementia caused by organic brain syndrome, 1 had paranoia, and 1 had psychotic depression. All of the 6 paranoid schizophrenic patients became symptomatic while living off the island. Recent trends indicate that drug and alcohol abuse, suicide rates, and delinquency are all on the increase in Tutuila and the other Samoan islands (2, 9).

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A Comparison of Outcome in Adolescent- and Late-Onset Bipolar Manic-Depressive Illness

BY GABRIELLE A. CARLSON, M.D., YOLANDE B. DAVENPORT, M.S.W., AND KAY JAMISON, PH.D.

The authors hypothesized that childhood- and adolescent-onset bipolar manic-depressive patients might have a more severe or a different type of affective disorder; early-onset patients were therefore compared with patients whose illness onset was after the age of 45. The results indicate that early age of onset is not a factor in the variable course and prognosis of manic-depressive illness.

ALTHOUGH THERE ARE references in older literature to the definite existence of manic-depressive illness in late childhood and adolescence, the problem of affective disorder in this age group has received little attention until recently, and its occurrence has been considered rare. However, one-third of Winokur and associates' bipolar patients became ill before the age of 20 (1); 40% of Perris' bipolar patients had onsets between the ages of 15 and 25 (2); and 20% of 2,000 manic episodes recorded by Wertham (3) began before age 20. The availability of definite treatment for depression and mania has made recognition of these disorders of more than theoretical interest, and efforts are being made to identify affective disorders in children and adolescents.

Criteria for establishing the diagnosis of bipolar affective illness in childhood were noted by Anthony (4) in 1960 and are similar to adult criteria (periodic, biphasic illness, family history of affective disorder, extroverted personality type). The existence of a depressive syndrome in children is still controversial, but there are investigators who advocate its existence and its protean manifestations (5–8). The question of childhood mania is even more unclear, although efforts are being made to identify either mania or a "manic equivalent" in childhood. For example, there has been some success in the use of lithium to treat episodic behavior disorders in children (9, 10).

This study was undertaken to add to our understanding of bipolar affective disorder in children and

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adolescents by examining the natural history of their disorder. It was proposed that the presence of a major psychiatric illness at a time when identity issues are being formulated and resolved might alter the adjustment of these patients and/or the course and prognosis of their illness, i.e., those who become ill during adolescence would have a poorer prognosis than those with later onset, either because the disease is worse or because they are more vulnerable.

METHOD

The diagnosis of bipolar affective disorder, using the criteria of Feighner and associates (11), was made in over 100 patients admitted to the NIH Clinical Center as patients of the National Institute of Mental Health Affective Disorder Study. They had either been admitted with a manic episode or had a manic episode during their Clinical Center hospitalization. The follow-up study of Carlson and associates (12), which ended in 1974, and admissions since then have included 28 patients whose first episode of affective disorder was recorded to have been before age 20. These patients were compared with a population of 20 patients who carried the same diagnosis but whose illness did not begin until after age 45, when they presumably had resolved adolescent identity issues. Age at onset was defined as the age at which the patient or a family member first noted affective symptoms of an intensity or duration that represented a change from the patient's usual personality. This may or may not have been the first time these individuals received treatment for these symptoms.

The two groups were examined using both the extensive admission histories obtained on all patients and the semi-structured personal interviews of patients and spouse or family member that were part of the previously reported follow-up study (12). Data included 1) duration of illness, 2) number and type of episodes, 3) occupational and educational achievement, 4) family history of affective disorder, 5) marital status, and 6) functioning at the time of follow-up. Statistical analysis involved Student's t test and chi-square analysis to compare early- and late-onset groups; Pearson product-moment correlations were used to compare the variables with each other.

RESULTS

Age at onset, duration of illness, sex distribution, and present age are presented in table 1. No statistically significant differences were found between the adolescent-onset and late-onset patients in sex distribution, type of onset (i.e., depression or mania), or distribution of episodes. Furthermore, the mean frequency of episodes per year (total number of episodes divided by duration of illness in years) was about the same for both groups (mean = .40).

TABLE 1
Clinical Characteristics of Adolescent- and Late-Onset Patients

Item	Adolescent Onset (N=28)	Late Onset (N=20)
Sex		
Male	12	11
Female	16	9
Age at onset (years)		
Mean	15.8	50.6
Range	8–19	45–63
Duration (years)		
Mean	19.3	10.4
Range	3–46	4–20
Current age (years)		
Mean	35.4	60.3
Range	16–65	47–83
Marital status		
Never married	12	1*
Ever married	16	19
Divorced	5	8
Mean episode frequency per year**	0.38	0.42
Patients with known depression onset	18	10
Patients with known mania onset	10	9
Patients with predominantly depressive episodes	6	3
Patients with predominantly manic episodes	14	7
Patients with equal numbers of manic and depressive episodes	5	4
Rapid cyclers***	2	6

*$p < .001$, chi-square.
**Number of episodes divided by duration of illness in years.
***More than 4 episodes per year.

Family history of psychiatric illness could be assessed reliably only in first-degree relatives. Histories of psychiatric illness other than affective disorder were negligible in both groups. Although there was no statistically significant difference in the percentage of affected first-degree relatives between the adolescent- and late-onset groups (54% and 35%, respectively), the adolescent group reported more siblings with depression and/or mania and the late-onset group had more affected parents.

Almost half of the early-onset group had never been married, compared with 5% of the late-onset group ($p < .001$). The two groups had similarly high divorce rates.

Outcome was assessed by educational level, income level, and a 4-point rating scale that compared the patient's level of functioning in job or school, social interactions, and family at follow-up with premorbid levels. In this rating system, explained in detail elsewhere (12), 4 is optimum functioning and 1 is poorest functioning. In cases in the adolescent-onset group where current job status could only be compared with prior school performance, we rated academic achievement and ultimate employment at follow-up.

The two groups had comparable educational achievement (≥ 14 years of schooling). The difference

in income (mean = $15,000 in the early-onset group and $25,000 in the late-onset group) is probably secondary to the current age disparity between the two groups. The average function score in both groups on the 4-point overall scale (mean = 2.5 in early onset, 2.3 in late onset) suggests some impairment, but there is no statistically significant difference between the two groups. The distribution of scores was also similar in the two—60% are doing quite well (scores of 4 and 3), 20% are significantly impaired but functioning (score of 2), and 20% are chronically incapacitated (score of 1). As might be expected, more of the late-onset group are dead; 4 of the late-onset group have died (2 suicides), compared with 1 death (from suicide) in the adolescent-onset group. Of the 48 patients, 2 have been lost to follow-up (1 from each group).

DISCUSSION

Recent reports (13, 14) have suggested that age of onset of affective disorder might delineate subgroups of unipolar and bipolar illness, but our findings do not support this. Although one might hypothesize that having a first episode of mania or depression in late childhood or adolescence could predispose patients to a poorer outcome by virtue of their having been afflicted at an earlier and more critical time or having had a longer duration and therefore more episodes of illness, most of our data suggest that early age of onset has little measurable impact on outcome. This is not to say that the adolescent-onset group does universally well, but they do no worse than their older counterparts. Winokur (15) drew the same conclusion in a comparison of patients with onset before the age of 30 with those whose illness onset was after 30.

Although specific follow-up studies on adolescent-onset bipolar affective disorder are not common in the literature, there have been a few that have made a distinction between unipolar and bipolar manic-depressive illness, which makes it possible to compare their findings with ours (16–19). These previous studies had a total of 99 patients between the ages of 12 and 22. Although it was noted that the appearance of the earliest episodes was often atypical, the periodicity of symptoms and eventual resemblance to classical mania and depression allowed the diagnosis of bipolar manic-depressive illness to be made. Criteria for good functioning differed, but approximately half of the patients seemed to be functioning well and about a fourth were chronically disabled at follow-up. These figures agree both with those reported here and with follow-up studies of whole populations of bipolar manic-depressive patients (12, 15).

We found no statistically significant difference in numbers of first-degree relatives with affective disorder in our two groups. Since the siblings and parents of the adolescent-onset group are not past the age of risk for manic-depressive illness, it is possible that there will ultimately be a difference in these groups. Proband reporting of illness in family members is unreliable (20), so corroborating data from family members were obtained where possible. It is conceivable that the late-onset group, who were considerably older, recalled those data less well than the early-onset group.

The income difference between the adolescent- and late-onset groups is probably a result of the 25-year difference in the mean ages of the two groups. Furthermore, the majority of adolescent-onset patients who were counted as having no income were, in fact, still in college.

Educational achievement, which is currently about equal for the two groups, may change as some of the adolescent-onset patients who are still in college and graduate school get their degrees. It is still probable that any difference between the groups could be explained by disparities in age and generation.

We noted that a larger proportion of adolescent- than late-onset patients was single. The 12 single adolescent-onset patients had a mean age of 27.8 years and the 16 married or divorced patients of 41.3 years. Furthermore, of the 9 oldest adolescent-onset patients (current mean age of 50 years), only 1 is still single. Thus it is possible that the unmarried early-onset patients will marry at a later age. There were no statistically significant differences in age of onset, frequency of episodes, education, family history, or general functioning at follow-up between the ever-married and never-married adolescent-onset groups.

The high rate of divorce in both groups suggests the traumatic interpersonal impact of bipolar affective disorder. It seems that the adolescent-onset group is no more at risk for marital dissolution. Moreover, recent data (21) on the divorce rate for a non-psychiatrically-ill population also reveal high lifetime divorce rates (26%).

Prospective studies will be necessary to answer more definitively the question of the impact of affective disorder in the young, since it is difficult to determine retrospectively what might have happened to these people had they not had to contend with this disruptive illness. In his 1952 article on manic-depressive psychosis in children, Campbell (22) noted, "Just as in adults, we observe some manic-depressive individuals who never seem to reach normalcy, others who have distinct but repeated episodes, and still others who have distinct but scattered episodes." It would appear from our data that having the onset of a major affective disorder in adolescence determines neither the severity of the illness nor its course.

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Impotence Responsive to Glyceryl Trinitrate

BY JOHN W. MUDD, M.D.

The author presents suggestive evidence that glyceryl trinitrate served to treat and diagnose atherosclerotic impotence in a man with psychogenic impotence. The prevalence of atherosclerotic impotence is not known but may be relatively high in older men. Vasodilators may be effective in diagnosing these patients, who do not have clear signs of arterial insufficiency of the lower extremities (e.g., Leriche's syndrome). The author postulates a pharmacological interaction between organic nitrates and imipramine.

SEXUAL INTEREST and activity during the middle and older years has been shown to decline (1–3) but nevertheless remains in those who enjoy good general health and the availability of a conjugal partner (1, 4, 5). The hormonal changes noted in aging men may contribute to their attenuated sexual physiological response (5, 6), but they do not cause impotence (5).

Few epidemiological studies of impotence appear in the literature. Kinsey and associates (7) reported an increase in the prevalence rate of impotence from 6.7% in 55-year-old white men to 27% in those 70 years old. Pfeiffer and associates (2) showed that 15% of 123 volunteers 60 years or over developed impotence during a 9-year prospective study period.

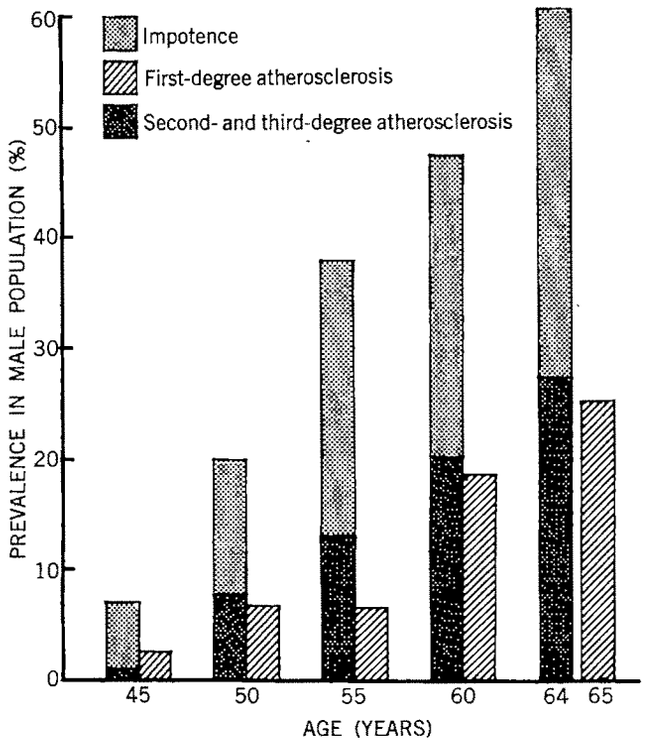
The reason for the apparent decline in potency with advancing age is as yet unknown. Of the many organic diseases associated with impotence or known to cause it (4, 8, 9), atherosclerotic vascular disease in particular has been suspected of a major causal role in aging men (10).

In addition to the syndrome described by Leriche and Morel (11), which includes impotence, claudication, absence of femoral pulses, and other signs of vascular insufficiency, other studies of surgical patients (12, 13) indicate that impotence often accompanies symptoms of pelvic arterial obliterative disease with or without diminished femoral pulsations.

It is well known that the prevalence of atherosclerosis increases with age (14). Schilling and asso-

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FIGURE 1
Prevalence Rates for Iliac Atherosclerosis (15) and Impotence (7) in Older Men



ciates (15) reported the prevalence of first- (0–20 mm of continuous calcification), second- (21–80 mm calcification over one or several sites), and third- (greater than 80 mm calcification) degree iliac atherosclerosis found in radiographic studies of 2,663 white men. Their rates are compared with those for impotence reported by Kinsey and associates (7) (see figure 1). The similar rates of increase of second- and third-degree atherosclerosis and impotence are striking and suggest that atherosclerosis may play an important role in impotence in aging men. The similar prevalence rates of iliac calcifications in excess of 20 mm and impotence further suggests that this degree of atherosclerotic disease may be necessary to cause impotence. Unfortunately, Schilling and associates did not report potency data.

The treatments of impotence are as varied as the historical periods in which they were applied (16). In my search of the literature, however, I failed to discover a report of the use of organic nitrates for erectile failure. Nevertheless, I found unpublished accounts of folk use of amyl nitrite "poppers" by sailors and others "to improve erections," which further encouraged me to report the following case.

CASE REPORT

A 56-year-old Cuban father of two was first hospitalized in 1968 for involutional melancholia. Related psychopathology included impotence, hypesthesias of the arms and legs, dread of cancer and heart attack, episodic alcohol abuse, preoccupation with homosexual fantasies, and a premorbid passive-dependent personality. During the previous 4 years he had experienced frequent partial or complete erectile failure lasting 1–3 months and premature ejaculation associated with marital quarrels and dyspareunia of his wife. At other times he performed "like a 15-year-old."

Although he was generally well and worked as a stock clerk during the next 7 years, he had several brief psychotic depressions precipitated by, for example, the death of his mother or the threatened loss of disability benefits (he later developed a compensation neurosis). He was evaluated four times during this period for intermittent impotence without loss of erection upon waking. Suspected prostatitis with epididymitis and scrotal cellulitis were each diagnosed once and treated with erythromycin. Serum testosterone, spine films, and neurological and peripheral vascular examinations were normal. Psychogenic impotence responsive to continuing psychotherapy was the primary and consistent impression. In psychotherapy the emergence of aggressive fantasies toward his wife commonly alleviated his symptom. He was also evaluated for variable complaints of chest pain, which were not related to sexual activity.

In 1973 he was hospitalized at Columbia Presbyterian Medical Center for heat stroke with impairment of hepatic and renal function and myoglobinemia complicated by pneumonia, *E. coli* septicemia, and an undiagnosed mild left peroneal neuropathy. He received kanamycin, ampicillin, and tetracycline. Of his liver function studies, his alkaline phosphatase, lactate dehydrogenase, and serum glutamic oxaloacetic transaminase have remained borderline or mildly elevated. His ECG showed right bundle branch block at discharge.

During the next 2½ years he was impotent in spite of sexual interest. He failed to note a full erection even on waking, and he was unable to have intercourse. He once complained of diminished sensation, temperature, and size of his penis, and of some urinary dribbling (which he attributed to "poor circulation"). Regular urological examinations were normal or showed recurrent scrotal cellulitis or questionable epididymitis. Follow-up neurological examinations showed no evidence of a progressive neuropathy and a fluctuating sensory examination, which could not be explained neuroanatomically. The additional diagnosis of hysterical conversion neurosis was considered.

He also complained of occasional calf pain or cramps. There was no history of gluteal claudication. Vascular examination of the lower extremities failed to disclose trophic changes, bruits, or diminished pulses or temperature. In addition, he complained of a variety of chest pains in several locations not typical of angina. They were diagnosed as musculoskeletal or pleuritic in origin, with hysterical painful elaboration. At no time in psychotherapy did he fear or give evidence

of unconscious fear that sexual activity could aggravate the heart disease he suspected (17). His fear of heart attack was attributed to his hypochondriacal neurosis.

The patient was again hospitalized in January 1976 for depression precipitated by the threat of divorce by his wife. Although his mood soon normalized in response to his wife's reconsideration, he volunteered for a research trial of 250 mg of imipramine and 15 mg of flurazepam daily. After 8 weeks with serum imipramine levels of 190–200 ng/100 ml, the researchers concluded that these drugs, like other psychotropics in the past, had no important clinical effect on him. At the end of the hospitalization, while the patient was still taking imipramine and flurazepam, one cardiologist initiated a trial on .3 mg of glyceryl trinitrate (GT) sublingually in response to complaints of chest pain.

The patient reported that the nitrate relieved a particular substernal chest pain and any leg pain (but not cramps) within seconds. He has continued to take one to four tablets daily. A presumptive diagnosis of angina pectoris with intermittent claudications was made. Laboratory stress and 24-hour ECG monitoring disclosed no abnormality. His fasting cholesterol was normal; his triglycerides were elevated between 335 and 473 mg/100 ml. With exercise (pulse 166–120) a loud flow murmur was heard over the lower abdomen transmitted to his femoral arteries. His peripheral pulses remained full.

Two days after initiating the nitrate and 6 hours after a .3-mg application, he was surprised to be awakened by his first erection in 2½ years. He subsequently reported that within seconds of taking the nitrate, his penis became partially or fully erect for perhaps 10 minutes (he did not experience an enhanced sense of libido or well-being) and that waking erections, some of which were unusually painful, occurred three to four times a week. He masturbated and achieved intercourse with orgasm.

One week later he began taking 5 mg of pentaerythritol tetranitrate (PT) four times daily. He spontaneously reported that within 30 to 45 minutes of ingestion of this medicine, his penis regained its normal size and sensitivity. He noted that the effect persisted as long as he took the medicine in four doses daily.

His potency response did not change when he reduced his cigarette consumption to one package daily or discontinued the imipramine 3 weeks after beginning PT therapy. Concurrently with discontinuance of imipramine he was given trimethaprim and sulfamethoxazol (Septra); he is now taking erythromycin for recurrent painful prostatitis and seminal vasiculitis, which has inhibited his ejaculations. Hemogram, erythrocyte sedimentation rate, Venereal Disease Research Laboratories test, serum B_{12}, 9-hour postprandial glucose, acid phosphatase, urinalysis, intravenous pyelogram, and cystoscopy were normal.

A delayed diagnosis of atherosclerotic impotence in addition to psychogenic impotence was made.

DISCUSSION

The reported temporal association of nitrate ingestion with changes in penile volume, temperature, and sensation, and relief of chest and leg pain strongly suggests a vasodilatory effect of nitrates on this man's pelvic circulation. The appearance of a lower abdominal bruit during high cardiac output strengthens the suspicion that he has atherosclerotic disease of his coronary and peripheral arterial circulation. The failure to note typical ECG changes during the stress study could indicate, as demonstrated arteriographically in some patients with variant angina, that he has predominately arteriospastic disease of the coronaries that is not typically aggravated by exercise (18). Probable claudications with a normal peripheral examination also suggest primarily spastic, in contrast to obliterative, disease in his distal circulation. Similar pathology is likely to be present in his internal iliac system. It is probable that this man's hypertriglyceridemia heightened his risk for atherosclerosis (19).

GT and PT are known to vasodilate a number of visceral (20) and peripheral (21, 22) arteries by relaxing smooth muscle. It seems likely that the internal iliac and pudendal circulation (the integrity of which with parasympathetic innervation is primarily responsible for potency [23]) is similarly influenced by nitrates.

In this case the time interval of several seconds between the patient's application of GT and the reduction of chest and leg pain and the onset of his erections is commensurate with current pharmacological knowledge of the serum dynamics of GT (21, 24, 25). The patient's interpretation of the effects of oral PT, i.e., that its clinical onset occurs in 30–45 minutes and lasts several hours, may also be accurate in his case. However, this length of action of PT has been questioned (26).

The patient's waking erection 6 hours after applying .3 mg of GT sublingually before starting PT is unexplained. Most likely the patient's suspected arteriosclerotic/spastic condition is such that segmental spasms occur only infrequently but last a long time. This formulation is compatible with his inconsistent symptoms of vascular insufficiency. Once the spasm is relieved by GT, the vessels remain without stenosis for several hours and permit normal nocturnal autonomic stimulation of his erection. Other hypotheses (27, 28) can be argued, but one that may also explain the long-lasting effect of PT is particularly interesting.

Denitration is enzymatically mediated by glutathione reductase, and depletion of glutathione (substrate) may greatly slow the inactivation of the organic nitrates (20). During the first month of nitrate treatment the patient was receiving imipramine. It has been recently shown that during the degradation of imipramine to 2-hydroxyimipramine an epoxide intermediary may be formed which transiently but strongly binds glutathione (29). According to a personal communica-

tion from Dr. J. Perel, New York State Psychiatric Institute, this postulated metabolic step of imipramine may have trapped hepatic glutathione to such a degree as to explain a 6-hour action of GT.

This patient's apparent response to nitrates may be idiosyncratic or peculiar to an altered metabolic capacity. However, further research may reveal nitrates or other vasodilators effective in diagnosing and treating atherosclerotic impotence whether or not clinically evident or shown by arteriography, Doppler flow, or plethysmographic penile studies.

The prevalence and specific causes of impotence also need to be determined in a large representative sample of aging men. This information will help psychiatrists who are continually challenged to differentiate organic from psychogenic impotence.

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This section contains reports of new clinical or laboratory observations, single cases of unusual interest or importance, descriptions of pilot studies, suggestions for future research, etc. Reports should be submitted in duplicate, typed double-spaced throughout, and may contain 1,000 words, no more than 1 table, and a maximum of 8 pertinent references. Figures cannot be used. Reports will be reviewed by at least one referee and the Editor; the author will receive a postcard informing him/her of the disposition of the submission. Reports will be edited for clarity and conformance with Journal *style.*

Lack of Effect of Naloxone on Schizophrenic Symptoms

BY DAVID S. JANOWSKY, M.D., DAVID S. SEGAL, PH.D., FLOYD BLOOM, M.D., ALAN ABRAMS, M.D., AND ROGER GUILLEMIN, M.D., PH.D.

Several lines of evidence suggest that naloxone (*N*-allylnoroxymorphone), commonly used as a narcotic antagonist, may have antipsychotic properties. Like other substances with proven antipsychotic properties, naloxone is reported to inhibit apomorphine-induced stereotyped behavior in the rat (1). Naloxone and chlorpromazine, given together, enhance each other's rate-decreasing effects on schedule-controlled behavior in the pigeon (2), which also suggests that naloxone has neuroleptic-like behavioral effects. In addition, naloxone inhibits *d*-amphetamine-induced increases in locomotor activity in the rat (3), although in recent unpublished studies we have found that naloxone pretreatment does not diminish, and in fact may enhance, *d*-amphetamine- and methylphenidate-induced stereotyped behaviors in the rat.

In regard to schizophrenic disorders, Bloom and associates (4) have reported that β-endorphin induces a "catatonic like state" in rats that they described as "reminiscent of some aspects of schizophrenia." Further, they reported that naloxone immediately antagonized this and other actions of β-endorphin. Bloom and associates proposed that administration of opiate antagonists such as naloxone might be expected to modify the clinical status of some psychiatric patients if altered β-endorphin function were involved in the etiology of their illness. More significantly, Gunne and associates (5), in a single-blind pilot study, reported that I.V. naloxone in doses of .4 mg rapidly and selectively abolished auditory hallucinations in four chronic schizophrenics.

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Method

The subjects were eight male schizophrenic patients (three paranoid, three schizo-affective, and two chronic undifferentiated) ranging in age from 19–48 years (mean=30), all of whom were receiving antipsychotic drugs (fluphenazine in doses of 10, 40, 80, and 100 mg/day, respectively; chlorpromazine, 300 and 600 mg/day, respectively; haloperidol, 50 mg/day; and trifluoperazine, 30 mg/day). All gave fully informed consent to participate in the study of naloxone's effects on hallucinations and other schizophrenic symptoms. The diagnosis of schizophrenia was based on *DSM-II* (6) classifications and the presence of Schneider's first-rank symptoms (7). All patients were grossly psychotic and could be considered process schizophrenics. Six patients were having active auditory hallucinations at the time of the experiment.

In a double-blind crossover design with counterbalanced order of administration, 1.2 mg of I.V. naloxone was rapidly given; saline placebo was given either 24 hours before or 24 hours after naloxone. Each patient was interviewed by a psychiatrist for a period beginning 15 minutes before and ending 60 minutes after each I.V. infusion. The psychiatrist rated the patients at 15-minute intervals using the Brief Psychiatric Rating Scale (BPRS) and asked them to describe any psychological or physical effects.

Results

Analysis of variance of individual BPRS items and total BPRS scores revealed no significant or nearly sig-

nificant differences between placebo and active naloxone. Specifically, no significant changes occurred in the following BPRS items indicative of schizophrenia: hallucinations, conceptual disorganization, unusual thoughts, mannerisms, grandiosity, blunted affect, and depression. None of the patients reported a decrease in auditory hallucinations. Two patients said they felt less nervous after naloxone infusion compared with one after placebo infusion, and two felt drowsy after naloxone infusion compared with one after placebo. No other mood changes were noted.

Discussion

We have not been able to confirm the report of Gunne and associates that naloxone has antihallucinogenic properties (5). It is significant that our patients, like those of Gunne and associates, were receiving neuroleptics and were severely and chronically ill. Thus in our eight subjects naloxone does not seem to have immediate antipsychotic and antihallucinatory effects when administered in I.V. doses of 1.2 mg. However, it should be mentioned that our results do not preclude the possibility that 1) higher and/or more chronic doses of naloxone might have antipsychotic effects, 2) naloxone may ameliorate symptoms in other subgroups of psychotic patients, or 3) naloxone may relieve psychotic symptoms in schizophrenics who are not receiving neuroleptics.

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Low Levels of Dopamine β-Hydroxylase and Psychosis

BY JOHN A. EWING, M.D., ROBERT A. MUELLER, M.D., PH.D., BEATRICE A. ROUSE, M. ED., AND DAVID SILVER, M.D.

Disulfiram (Antabuse) is known to be an inhibitor of dopamine β-hydroxylase (DBH) and has been used for this purpose in animal research (1). It is currently hypothesized that the presence of excessive amounts of dopamine within the mesolimbic system is involved in schizophrenia (2); therefore, it seems possible that disulfiram could provoke a psychosis by blocking the production of norepinephrine from dopamine. This would presumably be more likely to occur in persons whose supply of DBH is already minimal or borderline. Heath and associates (3) showed that larger than normal doses of disulfiram (1–1.5 g/day) led to an increase in psychotic symptoms in schizophrenia.

We have recently seen a case of psychosis in the course of a double-blind study of disulfiram that sheds some light on these issues. The full study will be described elsewhere; its purpose was to compare side effects of disulfiram with placebo in healthy males and to measure levels of plasma DBH for evidence of possible inhibition. All subjects took disulfiram in dosages of 500 mg/day for 1 week and 250 mg/day for the next 2 weeks within the total 9-week observation period.

Case Report

During the study described above, 1 of our 30 subjects had a psychotic reaction. All subjects provided a health history before admission to the study, but this subject had lied to us about his psychiatric history. When he was 25, he had had an illness diagnosed as schizophrenia (slightly more than 1 year before the study). Treatment had been recommended at that time, but he refused. The episode that occurred while he was in our study began after he had been on disulfiram for 3 weeks and then on placebo for 8 days. He

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failed to report for one of his thrice-weekly evaluations and was incoherent when we reached him by telephone. A home visit was made, and he was then seen in the hospital emergency room and admitted to the psychiatric service. His girl friend, who lived with him, told us that she had noticed him acting in a peculiar manner for 3 or 4 days previously.

The notes of the treating psychiatrist state that the patient had been observed speaking to people who were not present, mumbling, withdrawing socially, and posturing in a catatonic manner. He made irrational statements, responded better to female than to male personnel, and showed severe looseness of association and blunting of affect. At times he grimaced and smiled inappropriately and would not explain why. There was a paranoid flavor to some of his ideas; he reported a feeling of vague uneasiness and felt that some undefined force was against him.

There was some improvement after treatment with antipsychotic drugs, but since he had been assaultive on two occasions, the psychiatrist sought to have him committed for a longer stay in the hospital. An examining judge refused to sign commitment papers and the patient was able to leave the hospital against medical advice. Full neurological studies, including EEGs, had revealed no evidence of organicity and the final diagnosis was schizophrenia. We have learned from his girl friend that there is no reason to suspect that his psychosis was induced by hallucinogenic drugs and that he has now returned to his premorbid personality. Both she and the patient's father stated that this episode was exactly like the one that had occurred a year earlier.

All subjects in this study were seen three times each week for evaluation and for venipuncture so that DBH levels could be estimated. When the code was broken and the results were scrutinized, we discovered that this subject had a markedly lower blood DBH level than the other subjects. Results are expressed as nanomoles of octopamine per milligram plasma protein per hour; the methodology has been described previously (4). This subject's initial blood sample showed no detectable DBH activity. The mean DBH value for the other 29 subjects on the first day of the study was 28.2 nmol/mg (SD=18.7). Fourteen blood samples were taken before this subject dropped out of the study—the mean for all 14 was 1.28±0.80 nmol/mg. Thus, this patient's DBH activity was consistently lower than that of all the other subjects, who had values ranging from 2.11 to 75.0. There was no sudden change in DBH values at the time of the onset of psychosis.

Discussion

DBH does not cross the blood-brain barrier, and the levels found in peripheral plasma are believed to come primarily from the sympathetic nervous system (5). If peripheral blood levels of DBH correlate positively with brain levels, it might be that this subject was more easily thrust into a state of psychotic decompensation because of the low reserves of DBH in his central nervous system. Under these circumstances the administration of a DBH inhibitor such as disulfiram would be likely to produce a build-up of dopamine and a relative excess of dopamine to norepinephrine in parts of the central nervous system.

Other workers have found no consistent relationship between peripheral DBH levels and psychiatric syndromes (6). At one time it appeared that DBH levels in human brain were significantly lower in schizophrenics than in normal subjects (7), but this finding has not been corroborated (8). Peripheral blood levels of DBH remained consistent within each individual throughout this 9-week study, and we were not able to demonstrate peripheral DBH inhibition by disulfiram. However, this case is interesting because it correlates with the earlier report of Heath and associates (3) and indicates that this patient, by having less reserves of DBH, may have been more at risk for a psychotic state.

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Secondary Mania? A Case Report

BY KENNETH N. WIESERT, M.D., AND HUGH C. HENDRIE, M.D., CH.B.

The concept of affective disorder occurring as a secondary phenomenon in the course of other illnesses is now well established. With few exceptions, however, the syndrome described is that of depression. Although elevation of mood occurs in association with various drug therapies and manic symptoms have been described in the postencephalitic syndrome (1), case reports of the complete syndrome of secondary mania are rare, and one group of investigators has expressed doubt that it exists (2). However, over the past 4 years there have been three reports of apparent mania occurring in association with other illnesses (3–5). The establishment of the category of secondary mania has considerable diagnostic, etiological, and treatment significance, and it is important to document cases as they occur. The purpose of this paper is to report one such case.

Case Report

In September 1975, a 57-year-old woman was admitted to the medical ward of the Indiana University Hospital with a 5-day history of progressive headaches and drowsiness. At the time of admission she was confused and had a temperature of 104° F, but the rest of the examination was essentially normal. Her history revealed a diagnosis of chronic lymphatic leukemia 4½ years earlier; the disease was subsequently considered to be in remission. An EEG, brain scan, echoencephalogram, and cerebral anteriogram were all normal. Serial lumbar punctures were performed. The modal examination revealed 50–60 leukocytes, no erythrocytes, 100 mg/100 ml of glucose and 30/100 ml of protein. A diagnosis of encephalitis was made and the patient was given a course of cephalothin sodium and tobramycin. She remained lethargic and drowsy for the first 3 weeks of hospitalization and continued to spike high temperatures. She then showed progressive improvement and was discharged on no medication 5 weeks after admission, at which time her mental status was reported to be approaching normality, with the exception of minor memory impairment. Convalescent titres for St. Louis Type arborvirus were positive at 1:32 dilution. The discharge diagnosis was St. Louis Type A encephalitis. At home, she quickly returned to normal physically and mentally and was reported to be healthy on follow-up examination.

She continued to function well until the end of May 1976, when, according to her husband, she underwent a change in behavior. She became increasingly hyperactive, developed pressured speech, and had marked insomnia. She exhibited increasingly poor judgment and became quite grandiose and argumentative with no obvious psychosocial precipitant. There was no history of psychiatric disturbance; she had been a very conscientious, cautious woman. She was admitted to the psychiatric unit in August 1976 on a temporary hold order.

On admission she was quite hyperactive and had pressured speech. Her affect fluctuated from mild euphoria to anger. Her gross intellectual functions appeared normal, as did her orientation and memory. Physical and neurological examinations were normal. Laboratory investigations, including cerebrospinal fluid examinations and EEG, were all essentially within normal limits. A neuropsychological evaluation revealed minor difficulties with complex problem solving. Although a thorough family history revealed no evidence of psychiatric disturbance, a tentative diagnosis of mania was made. She was started on haloperidol, 4 mg b.i.d., which decreased her hyperactivity. However, she remained irritable and continued to have difficulty sleeping. The third week after admission, she was given a weekend pass and was argumentative and impulsive at home. When she returned to the hospital, lithium therapy was started at a dose of 300 mg t.i.d. and haloperidol was discontinued. She quickly attained and maintained a serum lithium level of 0.7–0.8 mEq/liter. Her hyperactivity improved, her sleeping pattern returned to normal, and her mood stabilized. She was discharged 5 weeks after admission with a diagnosis of mania. Since that time she has been seen monthly and has remained on lithium. She continues to perform well and is normoactive and euthymic 3 months after discharge.

Discussion

We believe the diagnosis of mania was justifiable in this patient. She fulfilled the criteria for that diagnosis, with the obvious exception of a previous neurological illness, and responded well to lithium. The relationship of the manic symptoms to her prior encephalitis is less certain, but we believe it is a reasonable assumption given that the patient had no history of major psychiatric disturbance, an onset at the age of 58, and no family history of affective disorder. In addition, although her intellectual functions were intact on mental status examination, a minor deficit in cognitive function was found during neuropsychological evaluation, which is consistent with a mild static cerebral disorder.

There is a marked similarity between the clinical picture of this patient and that of the patient previously

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described by Rosenbaum (5). Both patients had histories of cerebral insult followed by a period of several months during which they performed well; then both developed manic symptoms. Clinical mental status examination in both patients revealed little evidence of organicity, and both patients responded to lithium.

It is difficult to establish the number of cases of mania occurring in association with other illnesses that would be necessary to justify a category of secondary disorder. However, given this case and the previously reported cases, it seems that this category is at least a possibility. Thus, the syndrome of mania, like that of depression and schizophrenia, could represent a final common pathway with multiple etiologies and could be subclassified into primary and secondary components.

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Anger Arousal by a Motion Picture: A Methodological Note

BY WALTER ARMIN BROWN, M.D., DONALD P. CORRIVEAU, M.A., AND PETER M. MONTI, PH.D.

Although anger and its regulation are thought to play a crucial role in healthy and pathological personality development, interpersonal relations, "psychosomatic" illness, and criminal violence, there has been little systematic research on the physiology of anger or the psychological variables involved in its regulation and display (1). This dearth of systematic inquiry results in part from the lack of a laboratory model for eliciting and observing anger in a controlled, systematic, and ethical fashion.

Previous laboratory studies have used harassment of subjects or frustrating tasks to provoke anger (2). These procedures involve deception and have serious methodological as well as ethical flaws (3). Films with threatening and erotic content have been shown to reliably evoke anxiety and sexual arousal in laboratory settings (4) but have not, to our knowledge, been used to evoke and study anger in a laboratory setting. We now report the results of a preliminary study in which we showed that a film evoked specific and significant anger. These findings suggest a potential research tool for the study of anger and may provide information regarding stimuli for anger provocation.

Method

The subjects were 36 undergraduate men (N=14) and women (N=22) aged 18–31. They were randomly assigned to view one of two 20-minute made-for-television Alfred Hitchcock films or two 10-minute documentary films. The first Hitchcock film, "Man from the South," involves a wager in which the protagonist could have a finger chopped off and has been shown in previous psychophysiological research to regularly evoke subjective anxiety and its physiological concomitants (4, 5). The second Hitchcock film, "The Crooked Road," is the story of a couple taking an automobile trip who are unfairly accused of breaking the speed limit, mistreated by the police and judge, and coerced and cheated by the local service station. We hypothesized that this film would evoke anger on the basis of its content, our own reactions to it, and the response of laboratory personnel who saw it. The documentary films we used have been shown to evoke no changes in reported psychological state, autonomic nervous system, or endocrine variables (4).

There were no differences in age or sex distribution among the subjects in the three film groups. All films were shown at midmorning on the same day. Subjects were told that we were interested in the effect of films on mood and were asked to complete the Profile of Mood States (POMS) immediately before and after the films (6). The POMS, a widely used mood assessment instrument, is composed of 65 mood adjectives rated on a 5-point scale and scored for 6 mood factors. This test has been shown to be sensitive to transient changes in mood including mood changes evoked by films with threatening content (6).

Results

A 3 × 2 repeated-measures analysis of variance was carried out on the mean pre- and postfilm POMS scores (see table 1). Significant interactions between

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TABLE 1
POMS Scores Before and After Viewing Three Films

POMS Factor	"Crooked Road" Before	"Crooked Road" After	"Man from South" Before	"Man from South" After	Documentary Before	Documentary After
Tension*	7.4	7.8	6.8	13.5	7.3	5.0
Depression	5.3	6.1	7.0	6.6	3.9	3.3
Anger**	2.8	9.7	4.1	6.6	3.3	2.6
Vigor***	15.5	11.3	16.4	14.6	17.0	14.8
Fatigue‡	7.7	6.1	5.3	5.2	6.9	5.8
Confusion	6.2	6.3	7.2	7.3	5.7	5.0

*$p<.001$, analyses of variance, Trials×Conditions interaction.
**$p<.01$, analyses of variance, Trials×Conditions interaction.
***$p<.001$, analyses of variance, trials effect only.
‡$p<.05$, analyses of variance, trials effect only.

changes in mood and type of film were found only for anger ($F=5.40$, $p<.01$) and tension ($F=8.78$, $p<.001$). Further analyses for simple effects reveal anger to be increased significantly only after "The Crooked Road" ($p<.001$), with no significant change in anger after the other films. Tension was increased significantly ($p<.001$) only after "Man from the South." All films were associated with decreases in fatigue ($F=4.62$, $p<.05$) and vigor ($F=13.2$, $p<.001$), with no differences among films on these dimensions (trials effect only). Depression and confusion scores did not change following any film condition. Baseline POMS scores did not vary among the film groups and were consistent with reported normative POMS data from college populations (6).

Discussion

Our study revealed an unexpectedly significant and specific increase in reported anger following a film. These findings, if replicated with larger and more diverse groups, would suggest a role for selected films as research instruments in multifaceted studies of anger and aggressive behavior. In attempting to understand the relative potency of the experimental film in evoking anger, the possibility that the demand characteristics of the situation influence reported mood must be taken into account. Further research with these and other films using varied populations, changes in instructional set, and physiological measures will be necessary to clarify the influence of the demand variable on reported mood change. Feshbach (3) pointed out that "violations to self-esteem through insult, humiliation, or coercion are powerful elicitors of hostility, probably the most important source of anger and aggressive drive in humans." "The Crooked Road" depicts a series of insulting, humiliating, and coercive experiences in a way that seems to facilitate identification with the victim. This type of vicarious experience may be sufficiently powerful to elicit a degree of anger measurable on both subjective and physiological dimensions. Differences in response to such a film may be related to a variety of historical, personality, and physiological variables. Identification of the correlates and predictors of differential response to a potentially anger-evoking film may provide a step toward understanding the role of anger and its regulation in personality development, illness, and violence.

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Methadone Maintenance in a Patient on Chronic Hemodialysis

BY WILLIAM M. GLAZER, M.D., AND GEORGE L. COHN, M.D.

In this paper, we will report the case of a patient who is on chronic dialysis and who has been seen regularly by the consultation-liaison psychiatrist on the dialysis unit (W.M.G.). This patient illustrates interesting points about patients on chronic hemodialysis who are also addicted to narcotics.

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Case Report

The patient is a 56-year-old man who has been on chronic hemodialysis for the past year because of renal failure secondary to recurrent renal calculi of 25 years' duration. Because of his illness, he was unemployed

for 25 years and lived at home with his wife and four children, supported by Social Security benefits. The deterioration of his renal function resulted in hemodialysis beginning in February 1975. In addition to his renal failure, the patient has been addicted to morphine for the past 20 years; he feels that this addiction is iatrogenic secondary to narcotics given to him for renal colic. Over the past 5 years he has been a methadone maintenance patient in a nearby drug dependence unit.

For much of his 6 months in the hemodialysis unit the patient was a difficult management problem—he would become agitated and make derogatory and embarrassing remarks to the nursing staff and on occasion pulled out his needles and walked off the unit. This disruptive behavior occurred at least once a week during the course of his thrice-weekly 5-hour dialysis treatments. The disruptive state was not improved by administration of diazepam, haloperidol, or nonnarcotic analgesics.

The other medications the patient received were routine. Laboratory values were in the range to be expected for patients with chronic renal failure. Mental status examination revealed no psychosis or evidence of an organic brain syndrome.

The liaison psychiatrist was requested to see the patient in September 1975 because of a recent suicide attempt. The patient talked about not wanting to live and admitted to two suicide attempts in the preceding month. He said that he was upset because his wife and family had been ignoring him recently and were threatening to send him to a nursing home. The hemodialysis unit staff offered the information that one of his sons had recently married and left home. The patient felt that his disruptive behavior on the hemodialysis unit resulted from pain at the site of his fistula. He was hospitalized on a brief-treatment psychiatry unit because of his suicidal ideation, and his thoughts of suicide ceased during the hospitalization. The staff explored his family situation and noted severe communication problems.

One week after discharge, the disruptive behavior recurred. The psychiatrist believed that most therapeutic approaches had been exhausted and arranged a meeting between the staffs of the hemodialysis and drug dependence units in the hope that they could share information that would lead to a solution of the problem. It was learned that the drug dependence unit had been delivering methadone to the patient's home 3 days a week on nondialysis days; these deliveries always contained the next day's methadone supply. The staff speculated that the patient's difficult behavior during dialysis might be understood as a psychological withdrawal—the patient was taking the methadone meant for dialysis days on the preceding day, and therefore had no methadone on the days when he was dialyzed.

The patient was instructed to bring his methadone to the dialysis unit; a member of the dialysis staff administered the drug. Once this plan was initiated, the patient's difficult behavior ceased and he developed a good relationship with the staff that continues at this writing.

In patients with complicated medical problems who require chronic care from several disciplines, it is essential to have good communication between the systems involved. In the case we have described sharing of information resulted in a solution to a problem that had not been solved by the usual approach (1), in which attention is focused on the patient, the staff, or both. Several authors (2, 3) have spoken of the importance of paying attention to systems beyond the boundaries of the one in which the patient is being treated.

Since the passage of new legislation that entitles anyone, including those with severe psychiatric disorders and drug or alcohol addiction, to receive hemodialysis maintenance, psychiatrists working with hemodialysis units have been faced with difficult diagnostic and management problems. It is hoped that the presentation of this case will remind the psychiatrist of the importance of considering forces affecting the patient that arise outside the system in which he is being treated.

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Lithium-Induced Aggravation of Tardive Dyskinesia

BY EUGENE L. CREWS, M.D., AND ARTHUR E. CARPENTER, M.D.

There is a strong association between tardive dyskinesia and the use of neuroleptic drugs (1), and signs of pseudo-parkinsonism may persist long after discontinuation of neuroleptics (2). Observers have also been finding some association between lithium treatment and pseudo-parkinsonism symptoms (3, 4). In nontoxic patients lithium is known to produce a fine tremor that may become coarse with development of muscle twitching in intoxication (5). There has been little written about lithium and tardive dyskinesia, although one paper suggested that lithium might be of benefit in the treatment of this syndrome (6). In this report we will describe a patient in whom lithium carbonate seemed to aggravate substantially a preexisting tardive dyskinesia.

Case Report

The patient, a 46-year-old woman, was admitted to the hospital in a state of irritable excitement. This was her fourth psychiatric admission since the age of 27. She had been taking trifluoperazine and benztropine mesylate, which were stopped 1 week before her admission. Her mood was expansive and her affect was labile. She had pressured speech and flight of ideas and was diagnosed as having bipolar affective disorder, manic phase. Neurological exam revealed an oral dyskinesia, repetitive purposeless turning of the head (to the left more than the right), and continuous athetoid movements of the fingers of both hands (left more than right). These movements, which the patient said began about 5 months previously, were diagnosed as a tardive dyskinesia. Family history, clinical picture, and computerized axial tomography did not support a diagnosis of Huntington's chorea.

Lithium carbonate, 300 mg q.i.d. per os, was started on the 2nd hospital day. There were no other medications. On the 9th day the hyperactivity, irritability, and rapid speech had cleared partially. The serum lithium level was 2.02 mEq/liter. On the 12th day the patient developed massive, continuous choreoathetosis of fingers, hands, arms, head, and jaw, and "fly-catcher" tongue. There was no vomiting, weakness, muscle twitching, or impairment of consciousness, but she had difficulty speaking and swallowing. The lithium level was 2.18 mEq/liter. No lithium carbonate was given for the next 48 hours. The choreoathetoid movements returned almost to the admission level by the 14th day.

Lithium carbonate was resumed at 300 mg t.i.d., and deanol, 100 mg t.i.d., was started. After 1 day the movements began to increase in severity. The serum lithium level was 1.72 mEq/liter. The lithium carbonate dosage was decreased to 300 mg b.i.d. By the 20th hospital day, the movements were less severe than they had been at admission and at times were almost absent; the serum lithium level was 1.11 mEq/liter. The patient's mood was euthymic.

Discussion

The abrupt worsening of this woman's tardive dyskinesia may have been a progressive and continuing unblocking phenomenon that began after the phenothiazine was discontinued a week before this admission. The lithium treatment then would have been a coincidental and noncausal factor. However, the abruptness of the worsening over the course of 4 hours on the 12th hospital day in association with a lithium level of 2.18 mEq/liter is suggestive of a relationship. This possibility is strengthened by the marked diminution of the movements after lithium was discontinued. When lithium was resumed, albeit in a decreased dosage and concurrently with administration of deanol, the movements began to worsen again. The movement disorder again improved substantially with a decrease in the lithium dosage and continued administration of deanol.

The course of events in this case indicates that lithium toxicity may be expressed as an aggravation of a tardive dyskinesia. This patient tolerated lithium without exaggeration of her movement disorder before a high serum level was reached. Eventually, the severity of her abnormal movements was less at a therapeutic lithium level than it was when she was admitted. It is difficult to estimate the contribution of deanol to the final clinical state; lithium and deanol may be synergistic in the treatment of tardive dyskinesia or deanol may have a beneficial effect on dyskinetic movements that is not inhibited by therapeutic levels of lithium.

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IN MEMORIAM

David M. Levy
1892–1977

DAVID M. LEVY, M.D., who died on March 1, 1977, lived and worked during the first 50 years of modern child psychiatry. He offered leadership to the child guidance movement when it was characteristically new, provocative, eclectic, interdisciplinary, invested in therapy, yet mindful of the scientific, the evaluative, and the experimental. He became a model for several generations of child psychiatrists.

He was director of the Institute of Juvenile Research (the first of the modern child guidance clinics), chief of staff of the Institute of Child Guidance in New York, clinical professor of psychiatry at Columbia University, and president of the American Psychoanalytic Clinic for Training and Research. He received many honors, including membership in the Institute for Advanced Study, an honorary lectureship and citation by the Salmon Committee, and, most recently, a citation by the Society for Research and Child Development for his leadership and research in child development for 50 years.

Apart from his leadership in developing treatment programs for children, he was keenly aware of the opportunities for investigation in psychiatric treatment. As an investigator, he achieved fame for his straightforward simplicity, his originality, his range of creativity, and his unyielding honesty. Beginning his career as a neurologist and later trained in psychoanalysis, he exemplified the eclectic point of view. He retained his interest in the biological as well as the psychosocial roots of behavior. He employed a variety of therapeutic modalities. For example, he used psychoanalysis, play therapy (which was painstakingly accommodated to the needs of the individual child), and behavioral modification for the alteration of specific symptoms. His observations were uncomplicated and leaned primarily on the simple, direct authority of what he saw. In so doing, he introduced many new constructs into psychiatry, including maternal overprotection and rejection, affect hunger, sibling rivalry, and oppositional behavior.

Free play was the primary instrument for exploring the child's emotional life. However, play was shaped into situations that yielded to the requirements of scientific procedure, exemplified most brilliantly in his analysis of the hostile act in his sibling rivalry experiment.

His concept of the act as a functional unit in search of a goal was illustrated in his studies of sucking by babies and puppies, in which interference with satiation resulted in unusual oral habits. He showed how to use case records to test hypotheses regarding the origin of childhood behavior disorders, and he reported the impact on chidren of maternal overprotection, rejection, and deprivation. He emphasized the merits of "experiments of nature," and in his classical observations of oppositional behavior, he demonstrated that the normal developmental peak of such behavior occurs between 18 and 24 months.

Similarly, in the study of the baby's memory of inoculation, he established that memory cries did not occur before 6 months of age, so that these memories were not likely to be a source of anxiety for later events.

David was modest in his aspirations, entirely uncontrived and devoid of affectation, objective, straightforward, and free of bias. Always using ordinary language, he was exquisitely austere and clear in his expression. He enjoyed the act of contemplation and he was the most curious of men. His favorite game was the prediction of human events. Disarmingly simple, he was enveloped by a luminous aura of originality, inventiveness, and independence of spirit. He was remarkably self-possessed and his outward composure was, at times, mistaken for absent-mindedness. He was actually intensely aware and impelled by a straightforward interest in the comings and goings of all living things without prejudgment. His fundamental attitude was best described as curious and tender. He understood man's loving nature and accepted his banality, his dissimulation, and, at times, his wickedness. In calmly reporting their interplay, he communicated his overpowering consciousness of the beauty of living things.

WILLIAM GOLDFARB, M.D., PH.D.

LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Opiate Receptor – Natural Ligand System: Causal Factor or Defense System?

SIR: Enkephalin administration generates emotional detachment from pain experience, dissociated states of consciousness, and schizophrenia-like motor symptoms such as waxy flexibility and catatonic rigidity (1). On the basis of these observations, a causal relationship (genetic importance) between hyperactivity of the CNS opiate receptor system and severe emotional illnesses has been suggested (2–4). If this relationship does exist, emotional illnesses would be significantly improved by administration of inhibitors of opiate receptor activity. If, however, the organism increases its opiate receptor – natural ligand system to generate an effective defense against the pathogenic agent that is causally related to the emotional illness, such inhibitors would severely impair the patient's condition.

Animal experiments have been performed to obtain conclusive evidence differentiating these two mechanisms (causal factor versus generation of a defense system against it); results were evaluated in view of the available clinical literature. The effects of recurrent pain-related stressful experiences during early postnatal life on the development of the CNS opiate receptor – natural ligand system were studied in the rat (an animal born with 50% of its opiate receptor system, which functions qualitatively on an adult level) (5, 6). Recurrent daily stimulation of the periaqueductal gray matter by means of implanted microelectrodes or daily hot-plate treatment resulted in statistically significant and somewhat permanent increases from 30% to 50% in both CNS opiate receptor activity and natural ligand (enkephalin) content (7). These results suggest that the newborn (and probably the adult) organism is able to mobilize its defenses against pain perception by inhibition of the protopathic system.

The severe withdrawal symptoms observed in infants born to mothers who consume opiates suggest that man is born with a well-functioning opiate receptor system. These symptoms result from the sudden withdrawal of the ligand by cessation of transplacental supply at birth and by generation of the same (or similar) metabolic changes observed in the adult during acute abstinence.

According to current knowledge (8, 9), the future schizophrenic perceives maternal emotions empathetically and experiences hostility, rejection, double-bind communications, etc., in addition to the discomfort generated by the as yet unidentified etiological factors. It seems that such newborns attempt to decrease their discomfort by inhibiting protopathic pain perception through increased activity of their opiate receptor – natural ligand system. This overfunctioning of the opiate system interferes with intracerebral control (1) of the voluntary musculature, a system with an onset during the fourth postnatal month. The resulting tendency toward waxy flexibility and catatonic rigidity reinforces the newborn defense against pain: decreased motility makes it probable that the individual will have fewer new encounters with exogenous sources of pain. Development of this early defense mechanism promotes its future preferential use (9) during exposure to stressful events.

The observations described herein suggest that increased opiate-system functions are developed as attempts to defend against a causally related pathogenic factor; they also caution against attenuation of these defense systems before the causative factors of emotional illnesses are resolved.

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On Placebo Effects

SIR: The readers owe thanks to Michael H. Sheard, M.D., and associates for being so explicit regarding their subjects' ability to guess whether they were on lithium ("The Effect of Lithium on Impulsive Aggressive Behavior in Man," December 1976 issue). They reported a "double-blind, placebo-controlled" experiment on the effect of lithium in diminishing chronic aggressiveness. Unfortunately, those subjects whose behavior improved knew they were on lithium (80%,

$p<.01$); moreover, a possibly "surlier" group of subjects who might not have improved so much dropped out of the study as soon as they felt side effects. The authors dismiss this with the assertion that their questionnaire showed these subjects did not feel the drug was improving their behavior, as if conscious awareness were necessary for placebo effect to occur.

What this study does show is that inmates who know they are on lithium and do not drop out because of side effects have fewer "major" and a tendency toward more "minor" aggressive episodes. The explanation for this might as well be placebo effect (known to be enhanced when the placebo is physiologically active like lithium) as any specific action. This would certainly be an interesting and potentially useful finding and might be verified by a replication study with three groups: one on lithium, one on inert placebo, and one on an active placebo that mimicked some of lithium's side effects.

BEN EZRA GREEN, M.D.
Portland, Ore.

Drs. Sheard and Marini Reply

SIR: Dr. Green makes two criticisms of our study: that dropouts from the lithium group might not have improved as much as the subjects we were able to study, and that the lithium subjects' correct medication guesses compromised our conclusions.

With regard to dropouts, since we did not evaluate "surliness," we can neither confirm nor deny Dr. Green's proposal. Not all of the dropouts quit "as soon as they felt . . . side effects": of the 14 lithium subjects who quit, 1 rejoined and completed, 7 quit within 2 weeks of starting medication, and 6 quit after 3–5 weeks. As we pointed out in our report, we found no evidence that subjects who dropped out had different infraction histories than those who completed, and other results (see below) suggest that many dropouts may have been due to an antiaggressive effect.

With regard to the double-blind, we are aware that "conscious awareness" is not a necessary condition for a placebo effect. However, it obviously is a sufficient condition: the behavior of subjects who are aware they are receiving a drug with putative antiaggressive properties may well be influenced by their knowledge. We therefore attempted to evaluate their subjective responses rather than "dismissing" this problem; our evaluation provided no evidence for a frank influence on behavior.

There are three observations that support a specific, rather than a placebo, effect of lithium (1). First, a comparison of placebo subjects who guessed they got placebo with those who guessed lithium reveals no difference in major infractions per man-week while on medication, a slight increase from the premedication to the medication period for those who guessed lithium, and no change for those who guessed placebo. Second, in studies referenced in our report Sheard (2) and Tupin and associates (3) have shown that lithium has an antiaggressive effect in subjects to whom aggression is ego-syntonic: reduced aggressivity causes uncharacteristic anxiety and these men may drop out of the study. Third, the subjects in those studies and in our own had histories of repeated incarceration and a variety of therapeutic and psychopharmacological treatments with no evident improvement in their behavior: they are self-selected to be refractory to amelioration of their violence. For these reasons a placebo effect is a much less satisfactory explanation than a specific antiaggressive effect of lithium.

We discussed the problems of double-blind methodology with lithium at length in a paper (4) referenced in our report and concluded that use of an "active placebo" would be an appropriate strategy to test in lithium psychopharmacology. We have also pointed out that there are several nontrivial conditions which must be met for such a strategy to be valid (4).

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MICHAEL H. SHEARD, M.D.
JAMES L. MARINI, PH.D.
New Haven, Conn.

Politics or Science?

SIR: "The Why of Fragging," by Thomas C. Bond, M.D. (November 1976 issue), is a disturbing article from a number of standpoints. At the most superficial level it concerns me that the Editorial Board did not show greater discrimination in blocking this unscientific and political document from publication. I cannot fault the author, who obviously represents a defined political position and chooses to support it through the use of "science."

For those readers who may have missed the political position, I would like to raise the following issues not answered by the article.

The author describes a study group, offering no comparative sample and no indication of whether this type of person is usual or unusual among enlistees. Having done psychiatric screening for the military myself, I am aware that he is describing a large number of individuals in the military, not only the ones who "fragged."

A second question is why violence against superior officers experienced a phenomenal increase in one war (the Viet Nam war) compared with all other major wars (World Wars I and II and the Korean war). The author's answer is ostensibly apolitical. He cites the lack of activism, both political and racial, in the persons studied and conjectures that "drugs" led to the difference in this war.

If the same level of analysis were applied to the riots of the 1960s in the inner city, one might have found that individuals who threw Molotov cocktails possessed the same traits or lack of traits as "fraggers." Does that mean we give them individual therapy or blame the difference on "drugs" and the problem is done with? Obviously, we are dealing with larger social issues that catch people up as their victims.

In summary, I find the publication of this kind of article particularly dangerous because it misuses "science" to perpetuate a belief system that blames the victim on the bottom of the social structure. Perhaps I would not feel as disturbed about this article if I could read psychiatric studies of the

military and political leaders who generated and perpetuated a war that killed hundreds of thousands. Surely they warrant more psychiatric inquiry than their relatively ineffective counterparts among the enlistees.

STUART L. OKEN, M.D.
Baltimore, Md.

Dr. Bond Replies

SIR: Dr. Oken finds my article "disturbing"; I find his letter quite annoying and one-sided. His criticisms are largely simplistic, anecdotal, and unsubstantiated, more reflective of predetermined ideas than serious scrutiny of the article itself.

Dr. Oken raises the spectre of political motivations in the article and then elucidates his own political views quite clearly. He appears to want to explain everything on the basis of malevolent social forces, ignoring any interaction of personal forces that might explain why these specific men acted as they did. I would suggest he reread the article, but I doubt he would feel differently.

I would welcome any questions in the spirit of scientific inquiry, but I see no purpose in addressing vague, global criticisms. Deciding who were the offenders and victims in Viet Nam is something to be left to others.

THOMAS C. BOND, M.D.
Belmont, Mass.

Editor's Note. This paper was sent to a highly qualified reviewer who recommended its acceptance and felt that the paper would be of value to psychiatrists in the military and in the Veterans Administration. Dr. Oken faults me for publishing this paper, so in correspondence with him I asked whether he would want me to censor papers. He answered, "The position you occupy as Editor must of necessity warrant your role as censor or filter of information to the APA membership." I disagree; my position is that of a judge of the caliber of manuscripts, and I considered this paper worthy of publication.

Innovation or Fad?

SIR: I am writing about a Clinical and Research Report by David Rosen, M.D., entitled "A Primal Primer for Psychiatrists" (April 1977 issue). Many scientific papers are refused publication every month because the number of sound contributions exceeds your limited space. It is surprising you considered it justified to allot two pages to a testimonial for "primal therapy," recounting a personal experience of the author and an anecdote. The author claimed that he treated 800 patients by this method, but he does not report the results.

The paper proves what every experienced clinician knows—that "almost anything" helps some people. Since the time of Mesmer and long before, we have known of the strong impact of suggestion on susceptible persons, especially if administered in isolation from one's usual environment and supported by a group of believers.

You must be aware that publication in the *Journal* confers an aura of scientific authenticity on the author and his contribution. I hope that the Editor and Editorial Board will show better judgment in distinguishing between unorthodox innovative experimentation and the many fads currently exploiting the public.

ALEXANDER S. ROGAWSKI, M.D.
Los Angeles, Calif.

The Editor Replies

The paper in question was subjected to our usual review procedure and was sent to a person who is highly respected in the field. The reviewer's comments indicated that the paper should be published because it would add to the general level of knowledge about Janov's primal therapy and because there has been little information on the subject in the psychiatric literature. It should also be noted that one of the reasons we developed the Clinical and Research Reports section was to provide a vehicle for publication of papers that presented interesting ideas or observations in a brief form without the more stringent requirements related to methodology, sample size, analysis of data, etc., that apply elsewhere in the *Journal*. This does not mean that we accept mere "testimonials." I feel this paper has something to offer the readership. I regret that Dr. Rogawski does not agree with my judgment, but this is invariably the lot of the Editor.

F.J.B.

Dehumanization in Medical Education

SIR: I heartily applaud the proposals set forth by Paul Jay Fink, M.D., regarding psychiatry's involvement in the training of primary care residents (February 1977 issue). However, I fear that his focus is insufficient.

The main emphasis in the curricula of most medical schools is facts, not feelings. Students learn to repress emotions and to distance themselves from empathic interactions as a way of enduring daily confrontation with illness and death. From the first day, when they are faced with a cadaver, students are given little aid in dealing with their inevitable feelings. Later they learn to minimize the human part of suffering. For example, hospital ward rounds often are conducted with the brusqueness of a department store inventory. A six-week clinical rotation in psychiatry may come too late to begin to counteract these situations. Furthermore, the exposure of an unprepared medical student to the extremes of emotional illness could increase anxiety levels and even lead to a rejection of psychiatry.

The medical school curriculum should include forces to counterbalance this loss of empathy. Students need assistance in dealing with patient distress, anxiety, confusion in a strange environment, and the terror of a malfunctioning body. The medical student can be made aware of the benefits to be gained by enlisting patients as active rather than passive participants in their treatment and recovery.

Psychiatry needs to participate more vigorously in medical education. It is the only "primary care" specialty that must teach its own basic science, and this is frequently postponed until the third-year rotation. Foundations could be laid in the first two years for usable basic concepts and skills: recognition of emotional responses like depression and depressive equivalents, the stages of death and dying, the effects of separation from family on a hospitalized child, patterns of mental functioning and how to examine them, recognition of basic character types and how they might respond to illness, how learning takes place, etc. Clinical time might include

demonstration not only of severe psychiatric illness but also of the subtle variants that are much more common in medical situations. The training during medical school may be the only clinical contact with psychiatry that many students will have for the rest of their lives.

There are many potential benefits from this type of program. We might expect an improvement in patient care and an increase in psychiatric referrals, as well as an enhancement of the relationship between psychiatry and the rest of medicine. We might also find more students desiring to become psychiatrists; there would be more choice in the selection of residents for training programs, leading to an even higher quality of psychiatric practice. Furthermore, a long-term benefit in the form of decreased incidence of malpractice difficulties due to patient-physician misunderstanding and conflict might result.

Physicians have been getting a great deal of bad publicity lately. The traditional image of the kindly doctor has vanished. Some may say that it is not up to psychiatry alone to restore that image, but I believe that we are in the best position to take the initiative.

ELLIOTT M. STEIN, M.D.
Miami Beach, Fla.

Dr. Fink Replies

SIR: Dr. Stein is absolutely correct in his statements regarding the dehumanizing process of medical education and the need for medical school curricula to provide opportunities for dealing with affect-laden material. There has been a nationwide effort to introduce a variety of such learning opportunities into the first two years of medical school so that students' basic science experiences will not rigidify them early in their training. In many schools the development of behavioral science programs has done just what Dr. Stein prescribes with regard to affect, ethics, human development and behavior, and a host of important topic areas. In 1973 the National Board of Medical Examiners introduced a Behavioral Science Examination into the Part I exam. This has required deans and curriculum committees throughout the nation to pay attention to and introduce such topics and courses into the preclinical years. In addition, the nationwide trend to introduce clinical activities in these years also provides an enhancement of this humanizing process in medical education. Unfortunately, many schools have dealt with this issue by either insisting that these experiences be "scientific" or undermining them by disparagement or tokenism.

Over the past two years, a series of articles in the *Journal of Medical Education* has described such courses.[1] Almost every school in the country has introduced some variation on the same theme. Some important ingredients must be included if they are to serve the purposes outlined by Dr. Stein. First, the courses should be organized so that the smallest student-teacher ratio is used. Small-group teaching in groups of 8–10 students to 1 or 2 faculty members is optimum. These groups are improved when there is more than 1 faculty leader so that issues of controversy, uncertainty, and debate can be introduced. At Jefferson Medical College, each group has 3 instructors, a psychiatrist, a behavioral scientist, and a nonpsychiatric physician, preferably from one of the primary care professions. Such groups must be run with the correct combination of structure (for the obsessive-compulsive, content-oriented students) and nonstructure (for the introduction of affect and attitudinal issues). In addition, organized course material dealing with interviewing, communication theory, growth and development, ethics in medicine, legal medicine, and the sociology and economics of medicine, to name a few, should be scheduled as serious, required, and respected parts of the preclinical curriculum.

The goal of a long-term improvement in the doctor-patient relationship for all physicians might be achieved through this and other methods. There is still a great deal of reluctance on the part of medical educators to give psychiatry and behavioral science a proper place in the curriculum, but gradual progress is being made. All of these comments do not negate the position I have taken concerning adequate education of nonpsychiatric physicians at the graduate level as outlined in my article.

PAUL JAY FINK, M.D.
Philadelphia, Pa.

[1]A list of these references is available on request from Dr. Fink, Department of Psychiatry and Human Behavior, Jefferson Medical College, 1020 Locust St., Philadelphia, Pa. 19107.

Placement and Evaluation of Southeast Asian Children

SIR: The article by LCDR Richard K. Harding and John G. Looney, M.D., on "Problems of Southeast Asian Children in a Refugee Camp" (April 1977 issue) was interesting as a perspective for our experience working with a family that was caring for a 15-year-old Vietnamese girl whom they were considering adopting. Our contact with this case was through the county mental health center to which the girl was referred by an organization called Foster Care Relocation of Vietnamese Children. The girl had had two brief placements (one with a Vietnamese family) that were not successful because of behavioral problems. She also had had a brief psychiatric hospitalization.

The foster family consisted of the parents, two sons aged 10 and 9, and two daughters aged 4 and 2 (the younger girl is an adopted Korean child). The family brought the Vietnamese girl to the center for evaluation of aggressive behavior, which included calling the police and stating the family was hurting her (when she did not get her way), being mean to their oldest son, and being very demanding. This well-functioning, white middle-class family felt embarrassed and somewhat devastated that their good-will mission was not producing the desired results. The family was aware of the great need expressed by others involved for this placement to be successful.

A lengthy family session plus a number of subsequent conferences with the parents demonstrated clearly that the girl could not tolerate others receiving attention. When asked to share the limelight with her foster siblings, she responded by screaming, being aggressive toward the older son, and/or attempting to elicit intervention from outside persons or agencies.

A psychiatric evaluation performed with the aid of an interpreter showed a pleasant, friendly, well-dressed, illiterate Asian girl with minimal command of English, limited ability to communicate, and short attention span. Her functioning at school, in the family, in the office, and on the "draw a person" test was similar to children we have seen with IQs of approximately 70. On the Vineland Scale she had a socialized age equivalent of 9. The limited information given on the Wechsler Intelligence Scale for Children indicated severe retardation. A Bender Motor Gestalt test confirmed

moderate organicity. The neurological exam and sleep EEG were negative. The girl appeared to be of low intelligence, with previous psychosocial and environmental deprivation. She stated that she had been a servant girl since age 9 and that the woman she worked for in Viet Nam hit her with a stick on the head. The patient was started on 25 mg of thioridazine h.s. The dose was reduced to 10 mg because of morning drowsiness and the patient tolerated this dosage well.

The patient lived with this family for 9 months, but she was not adopted because of the problems described above and has been placed with another family. This case demonstrates the evaluation and placement difficulties that arise when transcultural problems are further complicated by developmental, organic, and emotional disturbances.

BARBARA L. KENNEDY, M.D., PH.D.
BETSY L. ERSLEV, M.S.S., A.C.S.W.
West Chester, Pa.

Confidentiality: A Solution to the Problem?

SIR: I would like to suggest a solution to problems related to confidentiality. Let all physicians provide all patients they see with a complete statement of their account, including diagnosis, method of treatment, and outcome, plus all charges and substantiation thereof. Each patient, then, will have all the information that is needed for legal disclosures, e.g., insurance, employment, school, disability, Social Security, etc. Information-seekers would then go directly to the patient; the patient would decide who will be given this information and who will not. With the patient's permission, physicians might communicate with other physicians for purposes of medical treatment only.

FRANCIS J. BURGIN, M.D.
Syracuse, N.Y.

Methodology in REM Sleep Studies

SIR: I would like to comment on "REM Sleep and Adaptation of Psychiatric Patients: An Application of Sleep Studies" by Ramon Greenberg, M.D., and Chester A. Pearlman, Jr., M.D. (October 1976 issue). The authors' attempt to apply available physiological data (REM latency) to explain psychological phenomena (dream content and patients' current conflicts) seems stimulating and intriguing, but unfortunately the method they used for the study is subject to some criticisms.

The authors used an extremely heterogeneous population. Their criteria for mental illness are vague and ambiguous, and they had no criteria for the selection of the patients (44 male inpatients were selected for study). No baseline information was obtained to enable the authors to compare their data. Physiological variables were correlated with psychological phenomena in a heterogeneous population without any data to justify this. The experimental data are so contaminated that any conclusion drawn from them is open to serious question. For example, shortened REM latency may be due to many factors, such as rebound phenomena following withdrawal from any REM-suppressing agents. It is also not clear whether patients were drug-free during the study.

The authors were blind in the areas that necessitate careful screening (they note that they "never saw the patients or heard anything about their histories") and were not blind in areas where they should have been. Finally, there was no mention of who the therapists were or in what capacity they served. The dynamic orientation of the authors could probably explain the above mentioned flaws in their methodology. If Drs. Greenberg and Pearlman used more stringent criteria for patient selection and dealt with a more homogeneous group, they would certainly be more able to make a reasonable hypothetical bridge between REM sleep characteristics and related dream content and prediction of response to different treatment modalities.

IRADJ MAANY, M.D.
Philadelphia, Pa.

Drs. Greenberg and Pearlman Reply

SIR: It is difficult not to respond by simply asking Dr. Maany to reread our paper. We did not, as Dr. Maany suggests, "attempt to apply available physiologic data (REM latency) to explain psychological phenomena (dream content and patients' current conflicts)." Instead, we used data collected in the sleep laboratory, both physiological and psychological (dreams), to predict the eventual outcome of the patients' treatment programs.

With this in mind, it becomes clear that Dr. Maany's points, methodological criticisms, and suggestions could only make this predictive task more difficult. The fact that we were able to make significant predictions in the face of such problems seems to us to indicate the power and meaningfulness of the sleep laboratory data. Keeping our aim in mind should also make it clear to the reader why we had to be blind to clinical information when we made the predictions. Otherwise, the predictions would have been contaminated by our use of clinical experience. Finally, we specifically mentioned the problem of drugs and drug withdrawal and how such a situation could only work against accurate predictions.

In conclusion, in his attempt to dismiss the implications of the study, Dr. Maany appears to have ignored both the data and the means by which they were collected.

RAMON GREENBERG, M.D.
CHESTER PEARLMAN, M.D.
Boston, Mass.

The Function and Organization of NIMH

SIR: Bertram S. Brown, M.D., clearly summarized the precarious, underfunded state of psychiatric research in his article "The Crisis in Mental Health Research" (February 1977 issue). I disagree with his conclusions that the National Institute of Mental Health (NIMH) should continue as an independent bureau with responsibility for both research and nationwide community mental health center operation. As I understand the largely oral history of the development and eventual separation of NIMH from the National Institutes of Health, this move was given impetus by psychiatrists' views that the future of psychiatry lay not with medicine, but conceptually and politically with a coalition of nonphysician workers in the mental health field. That rationale has been weakened by the failure of social psychiatric research to yield causal information about the major psychiatric syndromes (1) and the expansion of information in psycho-

pharmacology and the neurosciences in the past 20 years. The expansion of the service delivery function has created many problems. The community mental health center development, for all its benefits, has resulted in an illogical balkanization of treatment services in many communities (2). The segregation of psychiatry from the rest of general medical services has been detrimental to patients and psychiatrists, especially in consultation-liaison work and emergency care.

Rather than expanding the role of the psychiatrist, mental health centers have tended to constrict job functions to those of prescription writer and legitimizer of clinical functions performed by other staff members. This narrowing of role has been furthered by the elimination of psychiatrists from most program directorships and a reduction in the percentage of mental health center employees who are psychiatrists.

I believe that these problems dissipate the administrative energies of NIMH personnel and infiltrate the intramural activities of NIMH, bringing about the same conflicts one sees in the field; this seriously hurts the image of the psychiatric profession in the eyes of the public and politicians, as expressed in funding. When total budgetary dollars increase, it is difficult for many politicians to realize that research dollars may be suffering, especially if the service efforts are themselves confusingly referred to as "broad scale experiments," by attaching evaluation components to them. In most cases, the evaluation has been no more than the counting of services and tabulation of data that flow in a one-way fashion to Washington. Outcome research on the cost-effectiveness of psychiatric care in mental health centers remains inadequate.

Suggestive evidence that the more time-intensive group and individual psychotherapies can be provided efficaciously by persons without the expensive training of psychiatrists is leading to redefinitions of what psychiatrists commonly do and what third-party payers will pay them highly to do. The biobehavioral advances of recent years and the stronger role of psychiatry in the education of psychosocially skilled physicians equipped to provide primary care to patients with chronic, multifactorial disorders enable one to make a strong case for psychiatry to work more closely with the rest of medicine. I favor the return of research and training programs to NIH, with limitation of direct service endeavors to pilot projects that are carefully designed and systematically evaluated. Policy decisions for implementation on a larger scale could be made more wisely in this framework, avoiding the costly failures and oversold, unmet expectations that accompanied the "Great Society" programs of the 1960s.

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CHARLES L. BOWDEN, M.D.
San Antonio, Tex.

Challenging the Psychiatric Education Establishment

SIR: The publication of "Challenging the Medical Student in a Psychiatry Clerkship" by Leslie S. Linet, M.D. (January 1977 issue) leads me to write this letter to set forth a challenge to the psychiatric education establishment. Isn't it about time that experimental training programs attempted to collect some valid information to substantiate the rhetoric used?

Some measures of success for evaluating training programs are feasible and should be used. The first step would be to specify the changes in knowledge, skills, or attitudes expected as a result of a new program. For example, the author might have considered reporting National Board scores in psychiatry for this group of students. Faculty not involved in this particular clerkship might have reviewed and judged the adequacy of the students' written case reports; it would be even more convincing if evaluation included faculty ratings of videotaped interviews by the students compared with others in different clerkships. These methods could assess the degree to which the students in this clerkship develop skills and knowledge in treating acute psychiatric illness.

To assess selection bias, it would be useful to have information on how students are selected or select themselves. In a more complete evaluation of such a clerkship, it would also be interesting to have follow-up at six months to a year to see how much was retained and to follow the students on other clerkships to see to what extent the skills and knowledge are used on other services and what relevance the clerkship had for their other clinical work. One could also see if the clerkship changed students' attitudes toward psychiatry in general, toward their own interest in entering psychiatry as a specialty, or toward referring future patients to psychiatrists.

Although there has been precious little evaluation of training programs, there may be some rays of hope. Eaton and associates have published criteria for evaluating psychiatric education programs (1). In addition, after 40 years of evaluating the results of training programs by certifying psychiatrists, the American Board of Psychiatry and Neurology has authorized the development of a design for a validation study to determine the relationship between performance on the examination and clinical competence in the field (2).

With increasing pressure for public accountability, psychiatric training programs must demonstrate what effects they are having on students and trainees in order to maintain credibility. I hope that psychiatric educators will step up their interest in this area.

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BENJAMIN LIPTZIN, M.D.
Rockville, Md.

Dr. Linet Replies

SIR: Dr. Liptzin is right in suggesting several measures for evaluating teaching techniques. However, there should be a place in a noted publication such as the *Journal* for reports on methods, whether they are education or therapy techniques, without the requirement of a research design. I entirely agree that the pressure for accountability in psychiatry must be addressed, but it is not necessary that every psychiatrist do research to validate a particular hypothesis or to validate psychiatry in general. We have among our ranks psychiatrists who are qualified and have chosen to do this kind of research. Dr. Liptzin is Acting Chief of the Program

Analysis and Evaluation Branch of NIMH and is, I trust, qualified to lead us in this direction.

LESLIE S. LINET
Brooklyn, N.Y.

Treating Blurred Vision Associated with Antipsychotic Medication

SIR: While working with patients whose psychiatric condition necessitates maintenance on long-term antipsychotic medication, we have noted that blurred vision is a frequent untoward side effect. This can be particularly incapacitating to patients whose normal functioning requires extensive use of near vision and can lead to problems with drug compliance.

Hyperopia occurs in patients on antipsychotics because of the anticholingeric properties of these agents; this iatrogenic hyperopia causes the blurring of vision. Although this is a well-recognized phenomenon, it is usually either ignored by the treating psychiatrist or accepted as an unfortunate but untreatable side effect. A review of the literature revealed that no significant work has been done in this area.

We have found that the topical use of pilocarpine hydrochloride solution temporarily reverses the hyperopia and allows a more normal visual functioning. Pilocarpine has proved to be a relatively safe drug, suitable for long-term use in patients with glaucoma without the high risk of cataract development that is associated with certain anticholinesterase agents.

In a trial with 12 consenting patients who had blurred vision as a consequence of antipsychotic medication, treatment with pilocarpine hydrochloride, 2% solution, produced improvement in vision in all cases. Our initial treatment regimen was one drop applied to each eye three times a day. It should be kept in mind that individual dosage requirements vary, and titration of dosage is necessary. In these patients we have not observed the development of any significant untoward effects associated with the cholinomimetic actions of the alkaloid. In 3 patients, however, initial administration of the topical solution resulted in myopia. Adjustment of the medication dosage resulted in improved vision in these patients, and the initial myopia resolved with a decrease in dosage.

We are presently planning a carefully designed study to substantiate these observations.

J. GARY CARTER, M.D.
R. SCHMIDT
PAMELA ANN CARTER, M.D.
Charlottesville, Va.

Correction

There was an error in "Criteria for the Diagnosis of Alcoholism," by the Criteria Committee, National Council on Alcoholism (129:127–135, August 1972). The first entry in the right-hand column of table 2 (p. 130) should read: "Serum osmolality (reflects blood alcohol levels): every 22.4 increase over *290* mOsm/liter reflects 50 mg./100 ml. alcohol."

BOOK REVIEWS

On Psychosis, *by Paul Schilder; edited by Lauretta Bender. New York, N.Y., International Universities Press, 1976, 576 pp., $22.50.*

This collection is a tribute to the vision of Dr. Schilder. Although the papers are based on his experience from 1930 through 1940 with psychotic patients, they are replete with cogent observations that resonate with the interests of contemporary psychiatrists. Dr. Schilder's holistic approach to the psychoses is indicated by the subjects of his papers, which range from anatomy to psychoanalysis.

The lead article, which has not been previously published, summarizes most of the pertinent issues. Dr. Schilder viewed the psychoses as a regressive response to an overwhelming situation. He suggests that although the cognitive difficulty in psychosis is different from that in organic disorders, the disturbances are sufficiently similar to postulate an organic defect in psychosis. The regression that occurs in psychosis affects not only the libido but also the functions of the ego.

Dr. Schilder accepts the idea that narcissistic regression occurs in the psychoses, but he points out the confusion that has resulted from the failure to differentiate between narcissism as a developmental phase and narcissism as a libidinal investment of the self. He disagrees with those who posit an objectless psychic state in the infant, citing evidence that from the first the infant responds to stimuli from the outer world. However, the infant barely distinguishes what occurs inside his/her body from that which happens outside. Psychic development depends on the differentiation of inside from outside. Projection, introjection, and identification play an important role in this differentiation and in the development of psychic structure. Interference with development results in ego and superego deficits that render an individual vulnerable to psychosis. Psychosis is a response not only to the anxiety and guilt engendered by a conflictual situation but also to the helplessness and perplexity associated with the inability to cope with a situation.

Many of the subjects to which the author alludes in his comprehensive first paper are elaborated in subsequent papers. Dr. Schilder repeatedly tries to delineate the nature of the organic basis of the psychoses. The exact nature eludes him, but on the basis of careful clinical observation and psychological testing he indicates that the disturbances in the perceptual and motor faculties of the psychotic patient are not the same as those in which anatomical or toxic lesions can be demonstrated. In purely organic syndromes there is little connection between the patient's history and the illness, but in the psychoses symptoms are related to past experiences and have symbolic meaning. The perceptual disorders, including absence of pain in catatonia and psychotic distortions of body image as well as the thought disorder characterized by lack of differentiation of signal from referent, although suggestive of organic pathology, can be attributed to no known organic disorder.

Because Dr. Schilder did not believe in a sharp distinction between the somatic and the psychic but thought that psychological disorders may be associated with somatic changes and vice versa, he stressed the importance of the psychotherapeutic approach to the psychoses. In these papers he discusses the psychodynamic issues in both schizophrenia and manic-depressive phychosis that may have etiological significance as well as those which are important in therapy. He hopefully anticipated the day when we would be better informed about somatic factors.

Dr. Schilder recounts his experiences with insulin coma, pentylenetetrazol (Metrazol), and nitrogen inhalation, which were the somatic therapies in his time. Although these treatments have not proved useful and Dr. Schilder was sometimes too enthusiastic about them, his conviction that the psychoses are psychosomatic illnesses has been substantiated by continued investigation.

REBECCA Z. SOLOMON, M.D.
Hartford, Conn.

Adolf Hitler: A Family Perspective, *by Helm Stierlin. New York, N.Y., Psychohistory Press (Atcom), 1976, 153 pp., $9.95.*

This book is a gallant and ambitious attempt to explore the mystery of Adolf Hitler by considering him, like any other child, the product of the family that reared him. Helm Stierlin uses as his major conceptual tool his own well-articulated theory of transactional modes that operate as the covert organizing background to the more overt, specific child-parent interactions. In the case of Hitler Dr. Stierlin focuses chiefly on the delegating mode, in which the child's ties to his/her parents impel the child to fulfill certain missions, sometimes acknowledged but often quite out of awareness. This delegated role can be healthy but is often derailed, leading to various pathological consequences, which Stierlin describes in detail.

In brief summary, Stierlin's theory is that Hitler was bound (another transactional mode) and delegated by his anxious, pathetic mother, who, brutalized and exploited by her husband, Hitler's father, poured into Adolf all of her frustrated yearnings, which were augmented by the early deaths of her first three children. The missions that Hitler was to fulfill were to both redeem and avenge her. His mother's desperate grip, however, was stifling and elicited in Hitler a profound ambivalence that was turned against the projected figures who became for Adolf the enemies of Germany when the country itself assumed the role of his mother after her death.

Hitler's mythic enemies, of course, were the Jews, but how they came to occupy this position seems to me the weakest part of Stierlin's formulation. He relies on acceptance of the seminal role in the development of Hitler's anti-Semitism attributed by Rudolph Binion to the Jewish doctor Eduard Bloch, who cared for Hitler's mother during her final

illness. This, in turn, depends on the assumption of a massive unconscious reaction formation because the small amount of evidence available indicates that Hitler was consciously friendly in his feelings and actions toward Bloch.

Stierlin's portrait of Hitler does not depend on family theory alone, although this is central to it. There are also contributions from psychoanalysis and group theory, from current and past history, and from the sociology of the German character. The book is marked by a broad erudition that makes it consistently interesting to read.

What can be said about the success of Stierlin's attempt to make Adolf Hitler explicable in psychohistorical terms? It is clear that the author, like other Hitler theorists, is hampered by the lack of reliable and detailed information about Hitler's early life and family relationships. This paucity of facts unfortunately opens the way to unsupported speculation that, to some extent, cannot be avoided, although Stierlin attempts to stick to what is known. The picture of Hitler that emerges, as the product of a close-binding mother and a distant, rejecting father, has become a psychiatric cliché, a family constellation that has been used to explain the development of a bewilderingly wide variety of pathological character structures and clinical conditions.

It is clear that Hitler's family background and experiences were not at all unusual for German and Austrian children of his generation. It is difficult to conceive of this rather banal breeding ground as the prime factor responsible for the gestation of a monster like der Fuehrer. One is left with the feeling that although much of what Stierlin says about Hitler seems plausible, it constitutes a thin and unsatisfactory explanation of his boundless malignity. We are left with the Iago problem, and to this psychology can make only modest contributions.

PAUL CHODOFF, M.D.
Washington, D.C.

Depression: Clinical, Biological and Psychological Perspectives, *edited by Gene Usdin, M.D. New York, N.Y., Brunner/Mazel, 1977, 338 pp., $15.00.*

This is the eighth publication in the American College of Psychiatrists' series of annual surveys. It reviews the more prominent of the many perplexing features of depressive states. Almost 60% of the text—7 of the 13 chapters—is devoted to an exposition of the biological substrate of depression. The authors of these chapters detail the salient evidence concerning the role of genetics, psychoneuroendocrinological features, biochemical research findings, somatic treatment methods, toxicological manifestations of psychotropic medications, and certain aspects of the phenomenology of sleep and sleep disturbance as they relate to the depressive states.

Although the psychogenic aspects of depression are not neglected in this book, Dr. Usdin frankly provides the rationale that led to the preferential focus on biological aspects in his introduction: "There is . . . a voluminous body of psychoanalytic and psychotherapeutic literature regarding depression, while the field of pharmacotherapy is a relatively new one in which dramatic strides . . . are being made."

Each of the 13 chapters is appended by a series of reference citations that amplify for the interested reader the necessarily compacted texts of the individual essays. The nature of the material treated does not lend itself to easy reading. In effect, the compleat clinical psychiatrist these days has to become a polyglot. He/she has to be conversant with the languages of neurobiological sciences in order to translate their research into the idiom of patients. The gap between the enzymology of neurotransmitters and psychodynamics has not yet been bridged by even a unifying conceptual grammar. This deficiency, among other things, leads to an unavoidable difference in the styles of the individual essays in their attempts to encompass a comprehensive perspective of the clinical, biological, and psychological aspects of the depressive states. Nevertheless, this compilation of studies achieves the objective of the American College of Psychiatrists in that it provides a vade mecum for an eclectic approach to a better understanding of depression.

HOWARD P. ROME, M.D.
Rochester, Minn.

Harry Stack Sullivan: His Life and His Work, *by A.H. Chapman, M.D. New York, N.Y., G.P. Putnam's Sons, 1976, 270 pp., $8.95.*

Patrick Mullahy, the foremost interpreter of Sullivanian views, lamented that no clinical psychiatrist—preferably a former student of Sullivan—had brought forth a comprehensive exposition of Sullivan's ideas. Thereupon, Mullahy proceeded to publish *Psychoanalysis and Interpersonal Psychiatry* (1), a monumental book presenting Sullivan's views. *Harry Stack Sullivan* by Arthur H. Chapman adds some interesting biographical details about Sullivan to Mullahy's scant six pages.

Although it is not so comprehensive a book as Mullahy's, Chapman's book makes for quicker, easier, and more interesting reading. Easy readability is sometimes thought to be a vulgar attribute, but the fact that the writing itself is interesting, taken together with the intrinsic interest of Sullivan and his works, makes for a book that will not only attract and inform psychiatrists, most of whom are probably already Sullivanian enough, but also inform and interest the general reader. Chapman is a psychiatrist with a knack for writing lucidly and entertainingly.

The book begins with a chapter on "The Life and Emotional Problems of Harry Stack Sullivan," in which Sullivan's loneliness, homosexual preferences, private life, and tendencies to mystify his own childhood and adolescence are brought out of the closet. Following the biographical material, which is discreet and respectfully incomplete, Chapman brings Sullivan's principal views out into the open in chapters concerned with Sullivan's basic concepts of interpersonal relationships; participant observation, cognitive distortions, consensus, anxiety, and dynamisms; child and adolescent development; psychotherapy; and Sullivan's relevance to such current problems as racial discrimination and intergenerational difficulties.

Chapman steps carefully in and around some of the stark inconsistency, murkiness, and unsound formulations to be found in the writings and lectures of Harry Stack Sullivan. Chapman is not idolatrous, but he is definitely favorable to Sullivan's more consistent, more sensible, better reasoned, clearer, and sounder conceptualizations. All in all, this is a book that will edify the professional as well as the patient, the mildly curious as well as the devoted follower of Sullivan, and many of the friends and supporters of psychotherapy of all persuasions. One hopes that Sullivan and books about Sullivan are not blacklisted by analytic institutes because this study by Chapman could aid many trainees in

analysis and psychiatry to gain a useful working knowledge of the unhappy genius Harry Stack Sullivan. Like Freud, Sullivan was exceedingly close to his mother; Sullivan did not have a happy life, but he did not believe in Thanatos. Chapman's book is a most welcome contribution.

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PAUL L. ADAMS, M.D.
Louisville, Ky.

From Shaman to Psychotherapist: A History of the Treatment of Mental Illness, *by Walter Bromberg, M.D. Chicago, Ill., Henry Regnery Co., 1975, 350 pp., $4.95 (paper).*

Walter Bromberg has been writing about the history of psychiatry for 40 years, which makes him one of a very select and durable group of workers who have continued their researches for an extended period of time. I believe that Dr. Bromberg has not received the credit and recognition he deserves for his historical contributions. His *The Mind of Man* (1), which appeared in 1937, is a nicely written book covering the total sweep of past events in psychiatry and is still very useful, especially for laypeople and young psychiatrists who wish to understand how modern psychiatry has evolved. Bromberg's second book, written with John K. Winkler (2), used a biographical style to study the field. *Man Above Humanity* (3), published in 1954, is a more explicit study of the history of psychotherapy, a theme running through all of Bromberg's historical writings.

Man Above Humanity is my favorite of Bromberg's works. Less concerned with historical and social background, he concentrated in this book on the details of therapy—its philosophies and its methods. By so doing, he made a rich research contribution to our understanding of how psychotherapy has changed over the years.

From Shaman to Psychotherapist is a completely rewritten work, although it appears in a softbound format and is described by the author as the fourth edition of *The Mind of Man*. The revisions are impressive. Dr. Bromberg has retained the chronological approach of *The Mind of Man*, which naturally dictates a similarity of total structure, but the development of the story and the illustrations used are new. Among the topics discussed are early therapy, showing the role of the shaman and the extensive use of magic; the growth of rationalism during Greek and Roman times; the tensions that developed between Christianity and the emerging naturalism of the Renaissance, manifested in the witchcraft epidemics; and the impact of the increased use of the scientific method in the seventeenth and eighteenth centuries, which led to the "coming of age" of the asylum.

The last half of the book is devoted to more modern subjects: the derivation of hypnotism from mesmerism, conflicts in the hospital setting, the growth of faith healing and psychotherapy, the emergence of psychoanalysis, the recent burgeoning of group and encounter therapies, and, finally, organic treatments in psychiatry.

Bromberg's book is notable in that it is based on a wide reading of original literature and many years of contemplating the issues involved. It may be safely recommended to residents and others interested in learning about psychiatry and its origins. It is well written and has many useful references, although more extensive coverage of the growing secondary literature of the past 25 years (e.g., on the concept of the Victorian woman and her symptoms as exemplified in hysteria) would have made this textbook of greater value. The book ends with a serviceable index. Dr. Bromberg has every reason to be proud of this completely revised fourth edition of *The Mind of Man*.

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ERIC T. CARLSON, M.D.
New York, N.Y.

Genetic Engineering: Threat or Promise? *by Laurence E. Karp, M.D. Chicago, Ill., Nelson-Hall, 1976, 230 pp., $15.00.*

This is a book about the role of genetics in the diagnosis and therapy of disease; it is also a book about reproductive engineering. The first part includes such current topics in genetics as counseling, eugenics, genetic screening, genetic therapy, and prenatal diagnosis of genetic disease. Except for artificial insemination, the subjects discussed in the second part of the book are for the distant or not-so-distant future: sex determination, ectogenesis (fertilization and maturation of embryos in vitro), parthenogenesis (development of ova to maturity without fertilization), cloning, and the synthesis of life from nonliving matter.

Although the book is written for the layperson, all of the topics have psychological implications with which every psychiatrist should be familiar. In genetic counseling, for example, the emotional significance of the information given is considerable; Karp discusses the role of the counselor in helping the family make decisions, the issue of invasion of privacy, and the assessment of the intellectual and emotional status of the counselee. He also refers to the psychological difficulties in genetic screening caused by the knowledge that one has a genetic disease and to the psychodynamic implications of artificial donor insemination. Typical of the many believe-it-or-not facts in this informative book is Karp's estimate that between 5,000 and 10,000 babies are born in the United States each year through artificial donor insemination.

As the title of this book implies, the author is concerned mainly with the biological and ethical consequences of the new genetics. As a professional and as a citizen the psychiatrist cannot avoid involvement in the larger practical and moral issues posed by genetic knowledge. This is a good book with which to start learning.

The author sides with the more optimistic contingent in the field of genetics, reiterating that time, informed consent, and democratic institutions will prevent misuse and pointing out with clarity and imagination the potential benefits of genetic engineering. The polemical tone of his sallies at those who are more pessimistic often seems to express more sarcasm than scientific dispassion. However, regardless of his point of view, Karp is well equipped as a prominent obstetrician and geneticist to present the many facts on medical ge-

netics and human reproduction with which no psychiatrist can remain unfamiliar. The bibliography is up-to-date, and the index and glossary make the book useful for reference.

JOHN D. RAINER, M.D.
New York, N.Y.

Stress Response Syndromes, *by Mardi Jon Horowitz, M.D. New York, N.Y., Jason Aronson, 1976, 362 pp., $15.00.*

This volume is a scholarly investigation of stress response syndromes. Dr. Horowitz brilliantly provides a natural history and clinical depiction of a psychopathological state as it is manifest in the context of various personality styles. He also presents results of psychotherapy research that are applicable in the clinical setting. Probably the most unique accomplishment of this volume is the author's success in treating his subject comprehensively and organizing it in such a way that the reader is likely to experience a sense of rational order. *Stress Response Syndromes* should have a broad general appeal to psychiatrists because it addresses syndromes that are often disguised but are always in evidence among some of our patients.

Dr. Horowitz details his strategy and preliminary tactics early in the book. First he describes the psychopathological or maladaptive states under consideration. Then he considers the patient in the context of his/her current phase of response to stress and the patient's disposition in terms of personality for organizing and processing of defenses, thoughts, emotions, and coping patterns during such states. Finally, Dr. Horowitz examines therapeutic relationships and techniques currently in use in terms of how or why they may or may not be useful. He reviews psychoanalytic and contemporary psychological models and makes an extensive effort toward their integration. He briefly considers psychobiological models but does not analyze them.

Three basic styles of countering painful and intrusive emotional responses to stress are given major consideration in separate chapters of the book. These are titled "Inhibiting Operations: Reactions to Stress and Psychotherapy with Hysterical Personalities," "Switching Maneuvers: Reaction to Stress and Psychotherapy with Obsessional Personalities," and "Sliding Meanings: Reaction to Stress and Psychotherapy with Narcissistic Personalities." These exemplary models provide a vivid insight into the significance of personality trait variation in the manifestations of stress response as well as in the effects of various psychotherapeutic modalities. Specifically, Dr. Horowitz provides a knowledge of which therapeutic interventions may be effective and why or why not. In a series of discussions he uses clinical examples that consider how single external stress events trigger divergent reactions. Included are chapters on the loss of a limb, visual shock, automobile accidents, death of a parent, suicide of a friend, and multiple stress events.

Dr. Horowitz' efforts in this volume provide a well-organized and flexible conceptual model for understanding the stress syndromes, permitting improved clinical management and future systematic assembly and critique of clinical knowledge. We are indebted to Dr. Horowitz because he has rendered a rational scientific description and study of a psychopathological state in such a way that we may sequentially conceptualize, challenge, and improve on it.

WILLIAM E. THORNTON, M.D.
Chicago, Ill.

The Behavioral Management of Anxiety, Depression and Pain, *edited by Park O. Davidson, Ph.D. New York, N.Y., Brunner/Mazel, 1976, 188 pp., $13.50.*

One great value of behavior therapy has been its focus on observables and its subsequent refusal to consider phenomena not subject to objective verification. Thus the title of this book presents a contradiction of terms. Is it valid to apply behavioral methods to affects and sensations—subjective states that do not seem to be reliably verifiable by an observer?

The authors are aware of this issue. For example, Davidson states that there is

> increasing evidence that all three of these conditions can be conceptualized as operant responses or, at least, that large components of the anxiety, depression, or pain experience consist of operant responses, and that these are subject to modification using behavioral technology. (p. xi)

Meichenbaum and Turk state,

> Something is happening to behavior therapy! Its "learning theory" basis is being challenged and is being replaced, in part, by a cognitive orientation. . . . Illustrative of this shift in emphasis is our treatment program . . . of modifying the client's internal dialogues [i.e., self-statements and images]. . . . A more appropriate title than behavior therapy would be cognitive-behavior therapy. (pp. 1–2)

Keeping in mind the question of the validity of employing behavior therapy for nonobservable events, I was reassured by other aspects of the authors' approaches. Both in the definition and initial evaluation of the patient's clinical state and in the evaluation of the effectiveness of treatment methods they rely heavily on objective measures. Laboratory simulations of relevant stressors, home visits for naturalistic assessment, and tape-recordings of patient-spouse interactions are some of these procedures.

The five chapters of the book are derived from papers presented at a conference. Fordyce's chapter on behavioral management of chronic pain and illness gives a lucid, convincing discussion of the relationship between two types of pain behavior: respondent pain behavior involves the patient's response to pathophysiological processes, including the sensation of pain; operant pain behavior involves the patient's communication of his/her distress to others. It is toward the latter that behavioral management is primarily, but not exclusively, directed. Fordyce also describes the medical treatment system in which such a patient finds himself/herself, effectively delineating the forces at work on the two main characters in this social system—patient and physician. These analyses lead Fordyce to the rational treatment plan detailed in the chapter.

The other four chapters include a discussion of stress-inoculation training for anxiety, anger, and pain by Meichenbaum and Turk and a discussion of preventive behavioral approaches to individuals at risk for maladaptive responses by Poser, who emphasizes the lack of demonstrable effects of preventive interventions. There are two chapters on the behavioral treatment of depression, one by McLean and one by Lewinshon and associates; these discuss several behavioral models of depression, emphasizing cognitive and behavioral approaches to the problem areas delineated.

In summary, this volume focuses on important clinical areas for psychiatry, employing a largely cognitive emphasis with behavioral methodology for evaluation. It will be especially interesting to psychology graduate students, psychiatric residents, researchers in these areas, and the industrious practicing psychiatrist—who probably can find some of these areas presented more readably elsewhere. The format of the book is heavily textual, with few figures. The price is laudably low.

THEODORE WEISS, M.D.
Philadelphia, Pa.

Culture-Bound Syndromes, Ethnopsychiatry, and Alternate Therapies: Vol. IV of Mental Health Research in Asia and the Pacific, *edited by William P. Lebra. Honolulu, Hawaii, University Press of Hawaii, 1976, 299 pp., $16.00.*

This book, based on one of the East-West Center conferences, offers an excellent selection of papers that bring a great deal of order to the phenomena of mental health research in Southeast Asia and the Pacific. Anyone visiting this part of the world cannot help but be impressed by the lack of modern psychotherapeutic programs and the abundance of indigenous health methods on which the majority of people rely. The persistence of the population explosion and the rising costs of modernization suggest that modern programs will probably not be developed in the foreseeable future and that the indigenous programs will continue to provide care for the mentally ill. These techniques provide many of the universal elements of therapeutics as well as many culture-specific features geared to special problems in specific cultural settings.

The conference organizers sought to comprehend some of the therapeutic factors inherent in indigenous programs. Their goal was to find ways of collaborating with the native folk healers, who are certainly in closer contact with the population than the medical communities.

In Taiwan, where adequate facilities exist, many patients choose the more familiar and more intimate folk methods. The Japanese, who rank high in industrialization, literacy, and life expectancy, pursue both modern and indigenous therapies simultaneously. The majority of patients attending Yaji Sasaki's clinic in Tokyo also sought assistance in the increasingly popular new religions or in magicoreligious cults, which proved especially effective for neurotic patients. One of the more sophisticated techniques is Morita therapy, which is used in the treatment of *shinkeshitu*, a culture-bound obsessional-phobic state. Morita therapy combines a week's rest period with behavior modification techniques involving meditation and performing routine chores.

Many Asian participants in the conference reported difficulty in applying Western psychotherapeutic approaches in their own cultures. The emphasis on familial obligations and ancestral ties among the Japanese, as exemplified in the guided meditation of Naikan therapy, could not be reconciled with the emphasis on self-reliance and individualism found in the analytically oriented therapies. This raises important questions about the culture-specific nature of our own psychiatric approaches, which may be more value laden and less universally applicable than we thought. Recognizing this, we may be able to understand the current enthusiasm for a variety of quasi-therapeutic cults in the United States; these might be more responsive to the current needs of troubled people than conventional therapeutic methods.

The book covers a range of important substantive and methodological themes in transcultural psychiatry. A series of articles examines *Latah*, epidemic hysteria, possession, and *malgri*, a previously unreported culture-bound syndrome found in the Mornington Islands in New South Wales. This condition, characterized by fatigue, pain, and vomiting, develops after failure to eliminate traces of land food before entering the sea. Other chapters focus on indigenous healing practices in the Philippines, Polynesia, Malaysia, Taiwan, Korea, Japan, China, and among the Maori of New Zealand. This volume will be a useful addition to the growing field of transcultural psychiatry.

ARI KIEV, M.D.
New York, N.Y.

Death, Dying, and the Biological Revolution: Our Last Quest for Responsibility, *by Robert M. Veatch. New Haven, Conn., Yale University Press, 1976, 318 pp., $12.95.*

Society is failing to meet its obligation to its dying members. Too frequently people with terminal illnesses suffer isolation and neglect in hospitals, receive overzealous treatment by physicians, and are kept in ignorance of their situation by families and medical personnel. This state of affairs, as reported by observers of the medical care system and by dying patients themselves, has generated widespread concern.

According to Robert Veatch, the problem is one of rights. He believes that people facing death are being stripped of human freedom and responsible choice. Dying patients, he claims, have a moral right to know the truth about their condition, no matter how painful, and a legal right to refuse treatment, regardless of the reason. By means of closely reasoned and persuasive arguments Dr. Veatch arrives at a series of policy positions that he feels would ensure these rights and restore human dignity. He urges public debate on the issue, and this clearly written volume should provide a stimulus.

Dr. Veatch writes in a refreshing style and takes a clear stand on a host of philosophical, social, medical, ethical, and legal issues. He brings new and practical thinking to bear on such matters as the definition of death and the donation of organs for transplantation, subjects of continuing discussion and controversy.

Approaching his subject from a number of perspectives, he enriches his treatment of it. However, his changes in perspective may confuse the unwary reader at times. For example, the right to refuse treatment, which the author emphasizes, is a legal but not necessarily a moral right. Consequently, a patient may have a legal but not necessarily an ethical right to refuse treatment whenever he/she wants to and a physician may have a legal but not necessarily a moral obligation to discontinue treatment whenever a patient asks him/her to. The ethical positions of physician and patient are not satisfactorily defined.

For the physician, Dr. Veatch's book makes painful reading. He blames the medical profession, which he calls a "technocratic elite," for depriving dying people of their rights. He views physicians as motivated by self-interest and a desire for power and perceives them as using deceitful and high-handed tactics to maintain their authority. Thus he joins a growing attack on the professionalism of physicians so evident in the courts and the public press. His call for a physician-patient relationship with dying patients characterized

by mutual cooperation rather than the more traditional parent-child mode of interaction is sensible, but his attack on the medical profession is, for the most part, uncritical and one-sided.

However, this is an important book. It promises to become a force for needed change and deserves the attention of those who are concerned about the welfare of dying patients, their families, and their communities.

RUSSELL NOYES, M.D.
Iowa City, Iowa

Crusaders Criminals Crazies: Terror and Terrorism in Our Time, *by Frederick J. Hacker, M.D. New York, N.Y., W.W. Norton & Co., 1976, 346 pp., $9.95.*

Terrorism is a social phenomenon that is of interest to clinical psychiatrists. In this somewhat popularized volume, Dr. Hacker applies his personal experience with terrorists and his theoretical constructs to the attempt to understand phenomenological differences between various kinds of terrorists—those who are psychotic, those who have criminal and mercenary intent, and those who represent a social cause. Each type uses a different method of negotiating with the public and the media, each handles victims in a different manner, and each requires a different response on the part of officials.

Dr. Hacker makes a number of statements regarding the appropriate role of the media. In a postscript on the Israeli rescue at Entebbe he laments the fact that it received such favorable publicity; he suggests that the event should be viewed somberly by authorities in spite of its successful outcome. Generally, Dr. Hacker stresses the seriousness of terrorists and the causes they represent, reflecting on the fact that these causes are often valid. He counsels patience and describes the role of verbal dialogue with terrorists.

Some parts of this book are rhetorical; one section presents an engrossing account of the author's involvement in the Hearst case and his experiences with hijackers in Vienna in 1973. Dr. Hacker is a professional who has struggled to understand a widespread and alarming behavior; as such he enlightens us with regard to episodes we are often called to comment on as psychiatrists.

JOHN R. LION, M.D.
Baltimore, Md.

A Complete Guide to Therapy: From Psychoanalysis to Behavior Modification, *by Joel Kovel, M.D. New York, N.Y., Pantheon Books, 1976, 273 pp., $10.00.*

Today, people seeking relief from crippling neurotic symptoms, freedom from limiting neurotic character traits, or improvement of their lives through discovering and freeing latent resources within themselves are confronted with a variety of therapies. Dr. Kovel discusses the principles and techniques of these therapies so that each person can make what he/she perceives to be the most appropriate choice.

Following a general discussion of his concepts of adult neurosis and treatment, Dr. Kovel describes 14 types of therapies, beginning with Freudian psychoanalysis and its derivatives and a variety of individual and group approaches classified under the human potential movement. In this latter group are included such divergent approaches as Rogerian therapy, est, various group therapies, and behavior therapies. Following a summary of the concepts, goals, and techniques characterizing each therapy, Dr. Kovel ends each chapter with a synopsis in which he anticipates the reader's questions regarding indications and contraindications of each therapy in comparison with the others.

Dr. Kovel points out that all neurotic conflict is part of the totality of the individual's unique life experience. He believes that a common denominator lies in our advanced capitalistic social organization, which he feels imposes contradictions on all aspects of personal life. He acknowledges, however, that his belief that a free socialistic state would reduce or eliminate neurotic conflicts is an "experiment which has to wait its turn." Once a neurotic state develops, it takes on a life of its own, basic aspects of which are repressed.

Dr. Kovel considers Freudian psychoanalytic theory to be "indispensable for an authentically human psychology poised dialectically between biology and society." He says that psychoanalysis helps to conceptualize the irrational in rational terms and points out that most of the current therapeutic schools owe their impetus to Freud.

In spite of substantial differences, all therapies offer some model of neurotic disturbance and call on the patient to relive his/her conflicts in terms of their model. Beyond this similarity there is a great deal of dispute and confusion, with each therapy claiming superiority over all others.

Dr. Kovel sees the human potential movement as a distinctly contemporary American trend arising out of our affluent but emotionally unsatisfactory culture. Belief in the perfectibility of man provides some people with a "scientifically based" faith in a godless world. Dr. Kovel wisely cautions that one should bring the quality of emotional stability with himself/herself when joining human potential groups rather than enter them in search of it.

This book both satisfies and disappoints. As a guide for a troubled person seeking the best route to health it fails because of the inherent nature of neuroses, with their repressions, defenses, distortions, and rationalizations. Unfortunately, the perplexed cannot be their own diagnosticians. The effort to fairly present the pros and cons of each therapeutic approach while only subtly indicating disagreements among them is not likely to make the choice easier. However, to an interested layman the book is quite valuable for the information it presents in a clear, eloquent, and literary fashion. It is delightful reading for those who wish to learn about foreign lands, but, unhappily, it is not a guide for the traveler himself/herself.

LEWIS L. ROBBINS, M.D.
Glen Oaks, N.Y.

Prescriptive Psychotherapies, *by Arnold P. Goldstein and Norman Stein. New York, N.Y., Pergamon Press, 1976, 334 pp., $16.50; $10.00 (paper).*

In formulating the goals and purposes of this book, the authors propose that psychotherapy be much more specific. *Prescriptive Psychotherapy* addresses itself to the interaction of patient, therapist, and treatment and deals with "which type of patient meeting with which type of therapist for which type of treatment will yield which outcomes." Conversely, the authors state that a nonprescriptive point of view holds that the therapist's preferred treatment approach is equally and widely applicable to most patients.

With this purpose in mind, the authors have collected 11 papers written by various workers in the area of psychotherapy. These papers have been published in various journals and are included because the authors of the book view them as contributions to prescription development. At this point we come to one of a number of curious contradictions in the book. There is a section on prescription building that describes various steps at least preconsciously familiar to many of us. Certainly the imagery of a prescription is to most of us a medical one. The contradiction posited is that the authors themselves and a number of the other contributors criticize what they term the medical model, meaning in this context a developmental point of view, a focus on the significance of antecedent events and relationships, diagnosis, and a plan of treatment based on this comprehensive view of the patient. Although the authors favor prescriptive psychotherapy they oppose what they call the medical model because diagnosis means labeling and/or because knowing something of how the patient came to his/her current difficulty in some way interferes either with the set of the therapist or the course of the treatment.

Schlesinger's paper is a notable exception to this point of view. He points out that labeling is not diagnosis and that he does not share the view that diagnosis is an unnecessary precursor to treatment. Schlesinger also makes some effective comments about the categorizations of expressive and supportive psychotherapy. His discussion of supportive psychotherapy is worthy of attention in that he considers it more specific than commonly perceived. He suggests that we consider in what sense a patient needs support and that both expressive and supportive psychotherapy have their place and time in the treatment of any given patient.

Throughout the book considerable emphasis is placed on rather specific patient-therapist matching. One paper seemed to suggest that these matches might be developed to such an extent that there would be therapists who would treat only obsessional, hysterical, or schizophrenic patients. I find this hard to imagine from a practical point of view and troubling from the point of view of education and training. The psychiatric residency training programs with which I am familiar anticipate that residents will acquire the capacity to diagnose and treat a wide array of patients.

The final section of the book proposes a series of specific treatment modalities for a list of disorders, including psychoneuroses, sexual deviations, smoking, bronchial asthma, obesity, and schizophrenia. The recommendations are based on reviews by the authors of treatment results reported in the literature for each condition. The prescriptions, their expected effectiveness, and the methods by which the authors arrive at their recommendations can best be characterized as naïve.

JACOB SWARTZ, M.D.
Boston, Mass.

Participant Observation, *by Leston Havens, M.D. New York, N.Y., Jason Aronson, 1976, 174 pp., $10.00.*

This book promises to develop "a systematic psychotherapeutic technique that can be called, in Sullivan's words, participant observation." The author also compares participant observation with existential and psychoanalytic therapeutic strategies. As he develops this idea he differentially links the various clinical situations in which participant observation may best be used. Throughout the book Dr. Havens asserts that psychotherapy is a rational process in which the therapist makes conscious decisions about which techniques should be used to achieve specific goals. He includes clinical examples from his own practice, from his students' case reports, and from Sullivan's writings.

An active, interpersonally focused therapy that emphasizes the real aspects of the patient's history is the core of participant observation. Counterprojection, a technique that is typical of this approach, assumes that the patient projects onto the therapist distortions from the patient's past. The goal of the counterprojective technique is to remove the patient's projections by thwarting their development (transference) and deflecting them onto a "screen" that the therapist and patient can look at together. This serves to isolate the behavior of important real people of the past and allows objective review, thwarting the development of any intense feelings or fantasies about the therapist while promoting an increasingly accurate appraisal of the past. Subsequent intervention continues along the same lines, using a process Dr. Havens identifies as widening and balancing. Each successive clarification brings the patient and therapist closer to the real past.

Dr. Havens points out that psychoanalytic and interpersonal theory are equally concerned with the past; however, the interpersonal approach focuses on reality while psychoanalysis focuses on fantasy. Dr. Havens also states that objective-descriptive psychiatry seeks symptoms and signs while psychoanalysis seeks fantasies, existentialism seeks feelings, and interpersonal psychiatry seeks "the facts."

Through clinical examples Dr. Havens brings the reader a rich fund of information that begins to approach a point of deliberate use of specific techniques for specific situations. He focuses particularly on the usefulness of Sullivanian interpersonal psychiatry for borderline and paranoid patients (chapters 6 and 7 are especially useful in this regard).

I liked the general thrust of this book—the interpersonal/reality focus, the matching of techniques to situations, and the clarification of process. However, the book suffers badly from a lack of organization and clarity. As an example, counterprojective interpretation, a central focus of the book, is never carefully defined. Furthermore, there are sections in which the author starts to delineate major concepts and never seems to quite achieve that end. The breezy style only adds to this sense of imprecision. The book reads more like the free exchange of a seminar than an orderly, systematic, and scholarly exposition of theoretical and clinical material, which the field so badly needs. The book is full of great promise but falls short of its mark. Perhaps even worse, it may reinforce the myth that psychotherapy is unintelligible, unteachable, and incapable of being described accurately and precisely.

If read as a compilation of extensive case notes, *Participant Observation* is a rewarding and useful book for the senior clinician or advanced student. However, should the reader expect more, disappointment seems likely.

JOE PAUL TUPIN, M.D.
Davis, Calif.

Studies in Violence and Television, *by Melvin S. Heller, M.S., M.D., and Samuel Polsky, J.D., Ph.D. New York, N.Y., American Broadcasting Co., 1976, 503 pp., no price listed (spiral bound).*

Ten studies conducted over a five-year period are presented in this book. Five of the studies were conducted with two groups of institutionalized children who were hypothesized to be both vulnerable to television stimuli and predisposed to violent behavior. One of the groups consisted of emotionally disturbed children with secondary learning disabilities; the second group contained children from broken homes who had minimal or no emotional or learning disabilities. Beyond age range, sex, and race, few specifics are given about these children, although six clinical histories of emotionally disturbed children are given at the end of the book as typical examples.

Using clinical observations and a variety of psychological tests, the authors studied the influence on the children of violent television shows, violent cartoons, and action-adventure dramas with socially useful ("pro-social") themes. In general, the authors found no significant increase or decrease in aggressive or violent behavior in the children after they had seen any of these types of programs.

Another study reported in this volume hypothesized a relationship between cognitive styles (Kagan's reflection-impulsivity) and cognitive controls (Santostefano's leveling-sharpening) and the response of four groups of children to a television show with violent and prosocial content. A questionnaire regarding the children's viewing habits was also given to the children's parents. Few correlations were noted in the first part of this study; however, the answers to the questionnaires showed that children with special education needs or learning disabilities spent more time watching television and less time in other activities than children in regular school classes.

Three of the remaining studies were surveys. Two involved poor, black, young adult violent offenders, and the other involved white, middle-class, young adult college students. Among other data, the information gleaned dealt with drug use, violent behavior, and television viewing habits. About 25% of both groups reported that television was unrealistic and useless for learning anything. At least 20% of the offenders indicated that they had learned various criminal techniques from television programs.

The final study looked at the effects on violent offenders of television programs with various levels of violent content. The authors concluded that the only significant finding was that the more violent shows produced more recall of violent material.

Although these studies are interesting, I found this book rather difficult to read. The information it reports tends to be incomplete, typographical errors are numerous, and the data in the tables are occasionally inconsistent with the conclusions drawn or with data noted in the narrative. The results of the studies of children would imply a negation of the hypothesis that these children were predisposed to violence rather than of the hypothesis that violent television shows lead to violence. This point is especially important in light of the fact that none of the violent offenders came from environments like those of the study children, i.e., residential treatment and, apparently, a Catholic boys' home.

Despite these and other methodological difficulties and the typographical errors already noted, I feel that this book is a useful addition to the literature on television and violent behavior. It is likely to be most useful to the interested researcher (it has more than 100 pages of details on the tests and questionnaires used in the studies) than to a general reader seeking up-to-date reviews.

DANIEL A. DANSAK, M.D.
Washington, D.C.

This Month's Reviewers

REBECCA Z. SOLOMON, M.D., is Supervisor of Psychotherapy, Institute of Living, Hartford, Conn., and Training and Supervising Analyst, Western New England Institute for Psychoanalysis, New Haven, Conn.

PAUL CHODOFF, M.D., is Clinical Professor of Psychiatry, George Washington University School of Medicine, Washington, D.C.

HOWARD P. ROME, M.D., has retired as Senior Consultant and Professor of Psychiatry, Mayo Clinic and Mayo Graduate School of Medicine, Rochester, Minn.

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Psycheye: Self-Analytic Consciousness—A Basic Introduction to the Natural Self-Analytic Images of Consciousness Eidetics, *by Akhter Ahsen, Ph.D. New York, N.Y., Brandon House, 1977, 274 pp., $7.50.*

The Hyperactive Child in the Classroom, *by Frank P. Alabiso, Ph.D., and James C. Hansen, Ph.D. Springfield, Ill., Charles C Thomas, 1977, 311 pp., $15.75.*

AMA Drug Evaluations, 3rd ed., *by the American Medical Association Department of Drugs in cooperation with the American Society for Clinical Pharmacology and Therapeutics. Littleton, Mass., Publishing Sciences Group, 1977, 1,246 pp., $29.50.*

Adult Assessment: A Source Book of Tests and Measures of Human Behavior, *by Richard S. Andrulis, Ph.D. Springfield, Ill., Charles C Thomas, 1977, 308 pp., $12.50 (spiral bound).*

Word, Object, and Conceptual Development, *by Jeremy M. Anglin. New York, N.Y., W.W. Norton & Co., 1977, 289 pp., $14.95.*

New Dimensions in Psychiatry: A World View, Vol. 2, *edited by Silvano Arieti, M.D., and Gerard Chrzanowski, M.D. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1977, 489 pp., $26.95.*

An Assessment of the Community Mental Health Movement, *edited by Walter E. Barton and Charlotte J. Sanborn. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1977, 191 pp., $18.50.*

The Practitioner's Guide to Psychoactive Drugs, *by Ellen L. Bassuk, M.D., and Stephen C. Schoonover, M.D. New York, N.Y., Plenum Medical Book Co., 1977, 311 pp., $19.95 (spiral bound).*

Bircher-Benner Nutrition Plan for Headache and Migraine Patients: A Comprehensive Guide with Suggestions for Diet Menus and Recipes, *by the staff of the Bircher-Benner Clinic; translated by Timothy McManus. New York, N.Y., Pyramid Books, 1977, 158 pp., $1.50 (paper).*

Personality Description in Ordinary Language, *by D.B. Bromley. New York, N.Y., John Wiley & Sons, 1977, 263 pp., $19.95.*

The Art of Adoption: The "Hows" and "Whys" by an Adoption Worker Responsible for Over 900 Adoptions, *by Linda Cannon Burgess. Washington, D.C., Acropolis Books, 1976, 154 pp., $8.95.*

Women in Industry, *edited by Pasquale A. Carone, Sherman N. Kieffer, Leonard W. Krinsky, and Stanley F. Yolles. Albany, N.Y., State University of New York Press, 1977, 225 pp., $10.00.*

Behavior Analysis Forms for Clinical Intervention, *by Joseph R. Cautela. Champaign, Ill., Research Press Co., 1977, 223 pp., $16.95 (spiral bound).*

Great Ideas in Psychotherapy, *by Richard D. Chessick, M.D. New York, N.Y., Jason Aronson, 1977, 456 pp., $18.50.*

Intensive Psychotherapy of the Borderline Patient, *by Richard D. Chessick, M.D. New York, N.Y., Jason Aronson, 1977, 296 pp., $15.00.*

Interpersonal Approach to Psychoanalysis: Contemporary View of Harry Stack Sullivan, *by Gerard Chrzanowski. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1977, 240 pp., $16.95.*

Handbook of Behavioral Assessment, *edited by Anthony R. Ciminero, Karen S. Calhoun, and Henry E. Adams. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1977, 721 pp., $27.00.*

Psychologists on Psychology, *by David Cohen. New York, N.Y., Taplinger Publishing Co., 1977, 360 pp., $12.95; $6.50 (paper).*

Working with Police Agencies: The Inter-Relations Between Law Enforcers and the Behavioral Scientist, *edited by Robert Cohen, Robert P. Sprafkin, Sidney Oglesby, and William L. Claiborn. New York, N.Y., Human Sciences Press (Behavioral Publications), 1976, 200 pp., $9.95.*

Introductory Psychology: A Text Book for Health Students, *edited by John C. Coleman. Boston, Mass., Routledge & Kegan Paul, 1977, 343 pp., $15.00; $7.50 (paper).*

R.D. Laing: The Philosophy and Politics of Psychotherapy, *by Andrew Collier. New York, N.Y., Pantheon Books (Random House), 1977, 211 pp., $10.00.*

Working with Patients: Introductory Guidelines for Psychotherapists, *by Helen A. De Rosis, M.D. New York, N.Y., Agathon Press, 1977, 190 pp., $10.50.*

Women: A Psychological Perspective, *by Elaine Donelson and Jeanne E. Gullahorn. New York, N.Y., John Wiley & Sons, 1977, 313 pp., $13.95; $9.95 (paper).*

Psychiatry Patient Management Review, *by William M. Easson, M.D. New York, N.Y., Arco Publishing Co. 1977, 159 pp., $8.00 (paper).*

Basic Processes in Adult Developmental Psychology, *by Merrill F. Elias, Ph.D., Penelope Kelly Elias, Ph.D., and Jeffrey W. Elias, Ph.D. St. Louis, Mo., C.V. Mosby Co., 1977, 327 pp., $13.95.*

An Anthology of the Psychoanalytic Study of the Child: Physical Illness and Handicap in Childhood, *edited by Ruth S. Eissler, Anna Freud, Marianne Kris, and Albert J. Solnit. New Haven, Conn., Yale University Press, 1977, 310 pp., $17.50; $5.95 (paper).*

An Anthology of the Psychoanalytic Study of the Child: Psychoanalytic Assessment: The Diagnostic Profile, *edited by Ruth S. Eissler, Anna Freud, Marianne Kris, and Albert J. Solnit. New Haven, Conn., Yale University Press, 1977, 359 pp., $17.50; $5.95 (paper).*

Elements of Psychotherapy, *by Allen J. Enelow, M.D. New York, N.Y., Oxford University Press, 1977, 144 pp., $8.95; $3.95 (paper).*

Cocaine and Other Stimulants. Advances in Behavioral Biology, Vol. 21, *edited by Everett H. Ellinwood, Jr., and M. Marlyne Kilbey. New York, N.Y., Plenum Press, 1977, 715 pp., $49.50.*

Transvestites and Transsexuals: Mixed Views, *by Deborah Heller Feinbloom. New York, N.Y., Delta (Dell Publishing Co.), 1976, 296 pp., $3.95 (paper).*

Modern Synopsis of Comprehensive Textbook of Psychiatry/II, *by Alfred M. Freedman, M.D., Harold I. Kaplan, M.D., and Benjamin J. Sadock, M.D. Baltimore, Md., Williams & Wilkins Co., 1976, 1,336 pp., $35.00.*

Help for Your Grief, *by Dr. Arthur Freese. New York, N.Y., Schocken Books, 1977, 200 pp., $9.95.*

Science Studies Yoga: A Review of Physiological Data, *by James Funderburk, Ph.D. Glenview, Ill., Himalayan International Institute of Yoga Science and Philosophy, 1977, 248 pp., $5.95.*

Geropsychology: A Model of Training and Clinical Service, *by W. Doyle Gentry, Ph.D. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1977, 137 pp., $15.00.*

Effective Child Rearing: The Behaviorally Aware Parent, *by F. William Gosciewski, Ph.D. New York, N.Y., Human Sciences Press, 1976, 153 pp., $9.95.*

The Meaning of Illness: Selected Psychoanalytic Writings, *by Georg Groddeck; selected by Lore Schacht; translated by Gertrud Mander. New York, N.Y., International Universities Press, 1977, 266 pp., $12.50.*

Let's Play and Learn, *by Harriet Hartman. New York, N.Y., Human Sciences Press, 1976, 136 pp., $5.95 (spiral bound).*

Rx: Spiritist As Needed. A Study of a Puerto Rican Community Mental Health Resource, *by Alan Harwood. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1976, 241 pp., $18.95.*

Behavioral Architecture: Toward an Accountable Design Process, *by Clovis Heimsath, AIA. New York, N.Y., McGraw-Hill Book Co., 1977, 192 pp., $15.50.*

Starting Over: The Autobiographical Account of a Psychologist's Experience with Multiple Sclerosis, *by Ernest A. Hirsch. North Quincy, Mass., Christopher Publishing House, 1977, 169 pp., $6.95.*

Decision Making: A Psychological Analysis of Conflict, Choice, and Commitment, *by Irving L. Janis and Leon Mann. New York, N.Y., Free Press (Macmillan Publishing Co.), 1977, 473 pp., $15.95.*

Sensory Isolation and Personality Change, *compiled and edited by Mark Kammerman, Ph.D. Springfield, Ill., Charles C Thomas, 1977, 304 pp., $12.75.*

Your Second Life: Prevent, Treat, and Recover from a Heart Attack, *by Harold L. Karp, M.D., and Sam Locke. New York, N.Y., Pyramid Books (Harcourt Brace Jovanovich), 1975, 254 pp., $1.75 (paper).*

Death, Society, and Human Experience, *by Robert J. Kastenbaum. St. Louis, Mo., C.V. Mosby Co., 1977, 321 pp., $7.95 (paper).*

The Suicidal Patient: Recognition and Management, *by Ari Kiev, M.D. Chicago, Ill., Nelson-Hall, 1977, 153 pp., $12.00.*

Treatment and Rehabilitation of the Chronic Alcoholic. The Biology of Alcoholism, Vol. 5, *edited by Benjamin Kissin and Henri Begleiter. New York, N.Y., Plenum Press, 1977, 614 pp., $39.50.*

The Unconscious: Invention or Discovery? A Historico-Critical Inquiry, *by D.B. Klein. Santa Monica, Calif., Goodyear Publishing Co., 1977, 231 pp., no price listed (paper).*

Escaping the Hostility Trap, *by Milton Layden, M.D. Englewood Cliffs, N.J., Prentice-Hall, 1977, 243 pp., $7.95.*

Social Work Ethics, *by Charles S. Levy, D.S.W. New York, N.Y., Human Sciences Press (Behavioral Publications), 1976, 258 pp., $14.95.*

Abrégé de Neuro-Psycho-Physiologie, Tome 1: Fonctions Sensori-Motrices, *by M. Meulders and N. Boisacq-Schepens. Paris, France, Masson, 1977, 164 pp., no price listed (paper).*

If the Patient Is You (or Someone You Love): Psychiatry Inside-Out, *by Milton H. Miller, M.D. New York, N.Y., Charles Scribner's Sons, 1977, 261 pp., $9.95.*

Rape Intervention Resource Manual, *compiled and edited by Patrick Mills, Ed.D. Springfield, Ill., Charles C Thomas, 1977, 281 pp., $14.75.*

Guidelines for Research Involving Recombinant DNA Molecules, *by the National Institutes of Health. Bethesda, Md., U.S. Department of Health, Education and Welfare, 1976, unnumbered pp., no price listed (looseleaf).*

One, *by Orest. San Francisco, Calif., Strawberry Hill Press, 1977, 125 pp., $4.95 (paper).*

Nobody Home, *by Jennifer Lloyd Paul. New York, N.Y., W.W. Norton & Co., 1977, 247 pp., $7.95.*

Psychiatric Problems in Ophthalmology, *edited by Jerome T. Pearlman, M.D., George L. Adams, M.D., and Sherwin H. Sloan, M.D. Springfield, Ill., Charles C. Thomas, 1977, 159 pp., $12.50.*

How to Begin a Psychotherapy Group: Six Approaches, *edited by Herbert M. Rabin and Max Rosenbaum. New York, N.Y., Gordon and Breach, 1976, 137 pp., $12.50.*

B.T. Behavior Therapy: Strategies for Solving Problems in Living, *by Spencer A. Rathus, Ph.D., and Jeffrey S. Nevid, Ph.D. New York, N.Y., Doubleday & Co., 1977, 314 pp., $8.95.*

Psychotherapy for Women: Treatment Toward Equality, *edited by Edna I. Rawlings, Ph.D., and Dianne K. Carter, Ph.D. Springfield, Ill., Charles C Thomas, 1977, 463 pp., $17.50.*

Freud and His Followers, *by Paul Roazen. New York, N.Y., Alfred A. Knopf, 1975, 602 pp., $15.00.*

Grief and Mourning in Cross-Cultural Perspective, *by Paul C. Rosenblatt, R. Patricia Walsh, and Douglas A. Jackson. New Haven, Conn., HRAF Press, 1976, 219 pp., $9.00; $4.50 (paper).*

Child Psychiatry: Modern Approaches, *edited by Michael Rutter, M.D., D.P.M., and Lionel Hersov, M.D., D.P.M. Oxford, England, Blackwell Scientific Publications (Philadelphia, Pa., J.B. Lippincott Co., distributor), 1977, 991 pp., $62.50.*

The Psychology of Rigorous Humanism, *by Joseph F. Rychlak. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1977, 525 pp., $19.95.*

Mental Health in Children, Vol. I: Genetics, Family and Community Studies, *edited by D.V. Siva Sankar. Westbury, N.Y., PJD Publications, 1975, 602 pp., $37.50 (4-volume set, $115.00).*

Mental Health in Children, Vol. II: Neurophysiological, EEG, MBD, Sleep, Developmental, and Psychological Studies, Autism, etc., *edited by D.V. Siva Sankar. Westbury, N.Y., PJD Publications, 1976, 698 pp., $37.50 (4-volume set, $115.00).*

Mental Health in Children, Vol. III: Diagnostic, Psychiatric, Biologic, Therapeutic and Language Studies, Longitudinal, and Program Studies, etc., *edited by D.V. Siva Sankar. Westbury, N.Y., PJD Publications, 1976, 757 pp., $37.50 (4-volume set, $115.00).*

Psychopharmacology of Childhood (companion volume to Mental Health in Children, Vols. I–III), *edited by D.V. Siva Sankar. Westbury, N.Y., PJD Publications, 1976, 376 pp., $37.50 (4-volume set, $115.00).*

The Childhood Emotional Pattern: The Key to Personality, Its Disorders and Therapy, *by Leon J. Saul, M.D. New York, N.Y., Van Nostrand Reinhold (Litton Educational Publishing), 1977, 319 pp., $15.95.*

Group Counseling and Group Psychotherapy with Rehabilitation Clients, *edited by Milton Seligman, Ph.D. Springfield, Ill., Charles C Thomas, 1977, 320 pp., $16.50; $12.75 (paper).*

Psychopathology of Human Adaptation, *edited by George Serban. New York, N.Y., Plenum Press, 1976, 374 pp., $24.50.*

Changing Sexual Values and the Family, *edited by G. Pirooz Scholevar, M.D. Springfield, Ill., Charles C Thomas, 1977, 173 pp., $11.75.*

Mental Disorder: An Introductory Textbook for Nurses, *by H. Snell, R.M.N. London, England, George Allen & Unwin (Reading, Mass., Allen & Unwin, distributor), 1977, 197 pp., $6.50 (paper).*

Psychological Problems of the Child and His Family, *edited by Paul D. Steinhauer and Quentin Rae-Grant. Toronto, Ont., Canada, Macmillan of Canada, 1977, 449 pp., $12.95.*

Neurosurgical Treatment in Psychiatry, Pain, and Epilepsy, *edited by William H. Sweet, Sixto Obrador, and Jose G. Martin-Rodriguez. Baltimore, Md., University Park Press, 1977, 756 pp., $42.50.*

Men in Crisis: Human Breakdowns in Prison, *by Hans Toch. Chicago, Ill., Aldine Publishing Co., 1975, 327 pp., $14.75.*

Perspectives in Behavior Therapy, *edited by Dennis Upper. Kalamazoo, Mich., Behavioral Psychology Series (Behaviordelia), 1977, 282 pp., $6.50 (paper).*

Neuroregulators and Psychiatric Disorders, *edited by Earl Usdin, Ph.D., David A. Hamburg, M.D., and Jack D. Barchas, M.D. New York, N.Y., Oxford University Press, 1977, 598 pp., $29.50.*

Vocabulaire des Psychothérapies, *by André Virel. Paris, France, Fayard, 1977, 373 pp., no price listed (paper).*

The Road to Total Freedom: A Sociological Analysis of Scientology, *by Roy Wallis. New York, N.Y., Columbia University Press, 1977, 279 pp., $12.00.*

Sex Roles: Biological, Psychological, and Social Foundations, *by Shirley Weitz. New York, N.Y., Oxford University Press, 1977, 274 pp., $10.00; $5.00 (paper).*

Criminal Personality, Vol. II: The Change Process, *by Samuel Yochelson, Ph.D., M.D., and Stanton E. Samenow, Ph.D. New York, N.Y., Jason Aronson, 1977, 566 pp., $25.00.*

Creative Process in Gestalt Therapy, *by Joseph Zinker, Ph.D. New York, N.Y., Brunner/Mazel, 1977, 274 pp., $13.50.*

The American Board of Psychiatry and Neurology

The following successfully completed the Board Examination given in Washington, D.C., April 18–19, 1977.

PSYCHIATRY

Abraham, Gerald M., M.D., New York, N.Y.
Alder, William C., M.D., Hartford, Conn.
Almeleh, Jack, M.D., Penn Valley, Pa.
Amanat, Ebrahim, M.D., St. Louis, Mo.
Andreasen, Nancy C., M.D., Iowa City, Iowa
Ariola, Richard, M.D., New York, N.Y.
Aronoff, Michael S., M.D., New York, N.Y.

Ballenger, James C., M.D., Bethesda, Md.
Bartle, Stuart H., M.D., New York, N.Y.
Bassiri, Mohamad R.G., M.D., Sykesville, Md.
Beal, Edward W., M.D., Washington, D.C.
Bell, Anne M., M.D., Milton, Mass.
Berberich, Leo R., M.D., Bethesda, Md.
Berger, Doris E., M.D., Barrington, R.I.
Beute, William H., M.D., Fredericksburg, Va.
Bieganowski, Arthur, M.D., Bronx, N.Y.
Binkowski, Norbert J., M.D., New York, N.Y.
Blaine, Jack D., M.D., Potomac, Md.
Block, David A., M.D., Washington, D.C.
Bozzuto, James C., M.D., West Hartford, Conn.
Brandoff, Neal I., D.O., Philadelphia, Pa.
Brauer, Lee D., M.D., Glastenbury, Conn.
Brown, Herbert N., M.D., West Newton, Mass.
Bugaoan, Josefina G., M.D., Hammonton, N.J.
Burkes, Lynn J., M.D., New York, N.Y.
Buxton, Martin, M.D., Branford, Conn.

Camp, Edmond W. III, M.D., Columbia, S.C.
Cann, Ronald E., M.D., Baltimore, Md.
Carman, John S., M.D., Lorton, Va.
Carmona, Alicia T., M.D., New York, N.Y.
Cassimatis, Emmanuel G., M.D., Silver Spring, Md.
Chand, Suresh, M.D., St. Louis, Mo.
Chaudhri, Nonihal S., M.D., Louisville, Ky.
Chrobok, Jan M., D.O., Gillette, N.J.
Cohen, Daniel E., M.D., New York, N.Y.
Cole, Stephen A., M.D., New York, N.Y.
Collins, James L., M.D., Silver Spring, Md.
Connell, Janet T., M.D., Philadelphia, Pa.
Conrad, Margaret V., M.D., King of Prussia, Pa.
Cowan, Richard, M.D., New York, N.Y.
Cusano, Paul P., M.D., Hasbrouck Heights, N.J.

Daggett, Lorin R., M.D., Brookline, Mass.
DeBetz, Barbara H., M.D., New York, N.Y.
Delmas, Francis C., M.D., Brooklyn, N.Y.
Deverajan, O., M.D., Newtown, Conn.
Docherty, John P., M.D., Woodbridge, Conn.
Dodes, Lance M., M.D., Newton, Mass.
Doyle, Harry A., M.D., Philadelphia, Pa.
Drabkin, Naomi W., M.D., Westport, Conn.

Edelman, Stuart E., M.D., Wayland, Mass.
Edwards, Neil B., M.D., Houston, Tex.
Einbinder, Eli, M.D., New York, N.Y.
Eiseman, Frederick A., M.D., New York, N.Y.
Eisenbud, Monika M., M.D., Newton, Mass.
Evans, Michael A., M.D., Littleton, N.H.
Ezquerra, Jose R., M.D., New York, N.Y.

Faber, Raymond A., M.D., Brooklyn, N.Y.
Favelukes, Hanne E., M.D., New York, N.Y.
Ferrell, Robert W. Jr., M.D., Winchester, Mass.
Ferrero, Vittorio, M.D., Manchester, Conn.
Fields, George L., M.D., Watertown, Mass.
Fishler, Lois A., M.D., Valhalla, N.Y.
Fishman, Herman C., M.D., Philadelphia, Pa.
Fleisher, Michael L., M.D., New York, N.Y.
Florenzano, Ramon U., M.D., Chapel Hill, N.C.
Foodman, Allan, M.D., Topeka, Kan.
Franklin, Jean, M.D., Amherst, Mass.
Freedenburg, Daniel J., M.D., Catonsville, Md.
Freedman, George S., M.D., Salem, Mass.

Gagon, Terry E., M.D., McLean, Va.
Galler, Janina R., M.D., Brookline, Mass.
Gallo, Leslie S., M.D., New York, N.Y.
Gambill, John D., M.D., Belmont, Mass.
Gaston, Edward H., M.D., Arlington, Mass.
Geraty, Ronald, M.D., Stoneham, Mass.
Gerstenzang, Martin L., M.D., Buffalo, N.Y.
Gilbert, Jeffrey M., M.D., Winchester, Mass.
Ginzburg, Harold M., M.D., Silver Spring, Md.
Gold, Warren S., M.D., Pomona, N.Y.
Goldberg, Richard L., M.D., Washington, D.C.
Goldstein, Leonard, M.D., Bethesda, Md.
Golub, Sheldon, M.D., Fresh Meadows, N.Y.
Goodman, Sumner H., M.D., Takoma Park, Md.
Gorzynski, Janusz G., M.D., New York, N.Y.
Gotkind, Lawrence, M.D., Toronto, Ont., Canada
Greco, Philip S., M.D., Annandale, Va.
Green, M. Joan, M.D., Cromwell, Conn.
Green, Nicholas A., M.D., Weston, Mass.
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CHILD NEUROLOGY

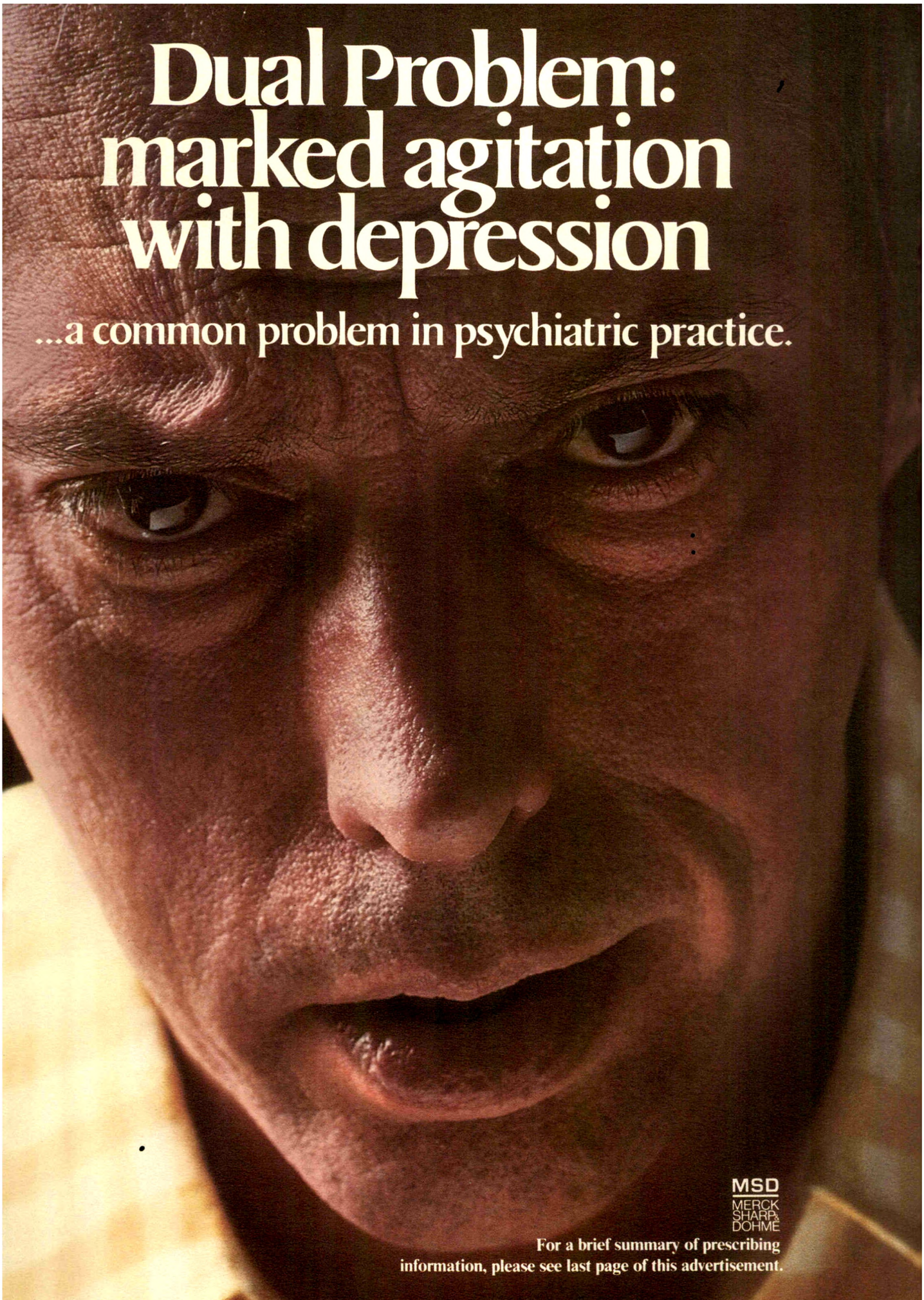
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with depression
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containing perphenazine and amitriptyline HCl

Marked agitation accompanying depression is a common problem in psychiatric practice, a problem often requiring dual-action therapy to effectively cope with both components of the disorder. TRIAVIL 4-25 helps treat both. A formulation particularly suited to psychiatric practice when higher doses are required, TRIAVIL 4-25 affords the psychiatrist the opportunity to treat marked agitation—each tablet containing 4 mg of the tranquilizer, perphenazine—while helping to relieve the coexisting depression with 25 mg of the tricyclic antidepressant, amitriptyline HCl.



helps treat both the marked agitation and the depression

An antidepressant alone may be adequate when the accompanying anxiety is mild, but dual-action TRIAVIL should prove more appropriate when the anxiety or agitation (accompanying depression) is moderate or severe.

The four formulations of TRIAVIL permit the psychiatrist to treat a relatively broad spectrum of depressed patients who manifest symptoms ranging from moderate anxiety to severe agitation, and allow for individualized dosage adjustment.

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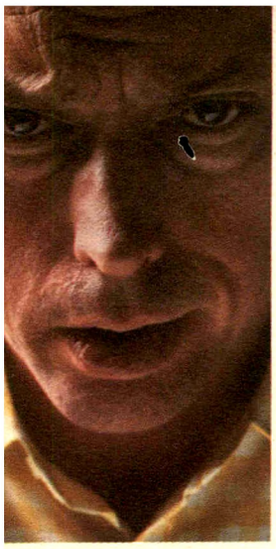
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TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. It is not recommended in children or during pregnancy. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

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for moderate to severe anxiety or agitation and depression

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CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisis hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism) have been reported and can usually be controlled by the concomitant use of effective antiparkinsonian drugs and/or by reduction in dosage, but some times persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy or may occur after drug therapy with phenothiazines and related agents has been discontinued. The risk appears to be greater in elderly patients on high dose therapy, especially females. Symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw. Involuntary movements of the extremities sometimes occur. There is no known treatment for tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage of the particular drug increased, or another drug substituted, the syndrome may be masked. Fine vermicular movements of the tongue may be an early sign of the syndrome. The full-blown syndrome may not develop if medication is stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythema urticaria, eczema, up to exfoliative dermatitis); other allergic reactions (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions); peripheral edema; reversed epinephrine effect; hyperglycemia; endocrine disturbances (lactation, galactorrhea, gynecomastia, disturbances of menstrual cycle); altered cerebrospinal fluid proteins; paradoxical excitement hypertension, hypotension, tachycardia, and ECG abnormalities (quinidine like effect); reactivation of psychotic processes; catatonic-like states; autonomic reactions, such as dry mouth or salivation, headache, anorexia nausea, vomiting, constipation, obstipation, urinary frequency or incontinence, blurred vision, nasal congestion, and a change in pulse rate; other adverse reactions reported with various phenothiazine compounds, but not with perphenazine, include grand mal convulsions, cerebral edema, polyphagia, pigmentary retinopathy, photophobia, skin pigmentation, and failure of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (pancytopenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinophilia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after long term administration of some phenothiazines. Although it has not been reported in patients receiving TRIAVIL, the possibility that it might occur should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia have also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for this drug, but which have occurred with other pharmacologically similar tricyclic antidepressant drugs and must be considered when amitriptyline is administered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation; myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred vision; disturbance of accommodation; increased intraocular pressure; constipation; paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Skin rash; urticaria; photosensitization; edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis; leukopenia; eosinophilia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric distress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; parotid swelling; black tongue. Rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness; fatigue; headache; weight gain or loss; increased perspiration; urinary frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrupt cessation after prolonged administration may produce nausea, headache, and malaise. These are not indicative of addiction.

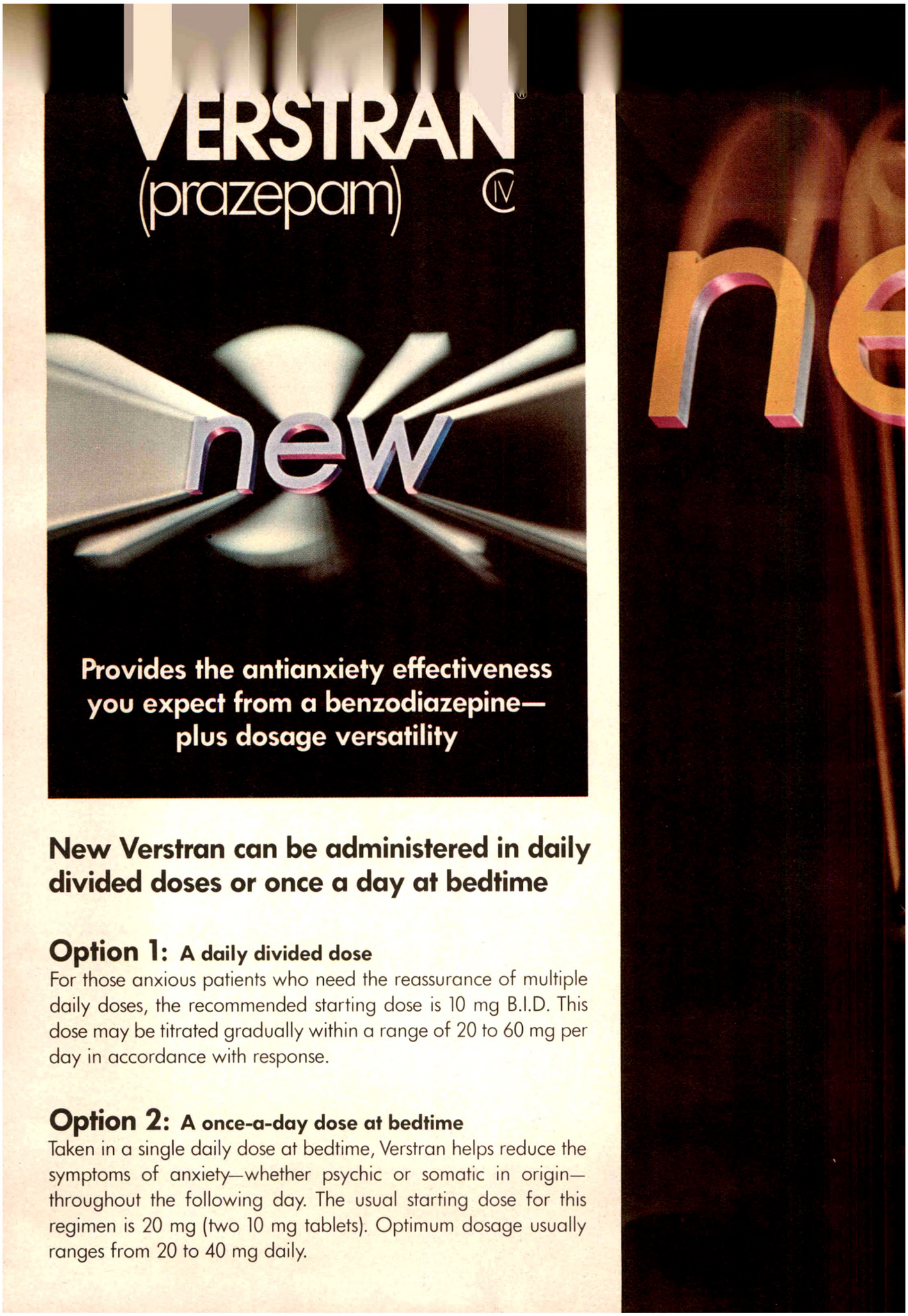
OVERDOSAGE: All patients suspected of having taken an overdosage should be admitted to a hospital as soon as possible. Treatment is symptomatic and supportive. However, the intravenous administration of 1–3 mg of physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage of physostigmine should be repeated as required particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine. On this basis, in severe overdosage with perphenazine-amitriptyline combinations, symptomatic treatment of central anticholinergic effects with physostigmine salicylate should be considered.

J 6TR06 R2 (DC 6613212)

For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486.

MSD
MERCK SHARP & DOHME


Warner/Chilcott Introduces
new
VERSTRAN®
(prazepam) CIV
A new addition for the treatment
of anxiety



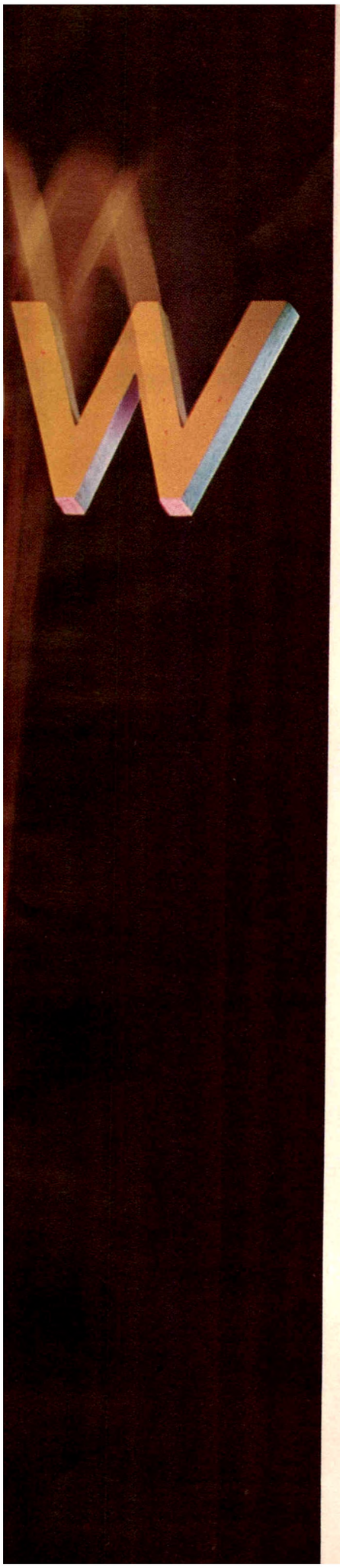
New Verstran can be administered in daily divided doses or once a day at bedtime

Option 1: A daily divided dose

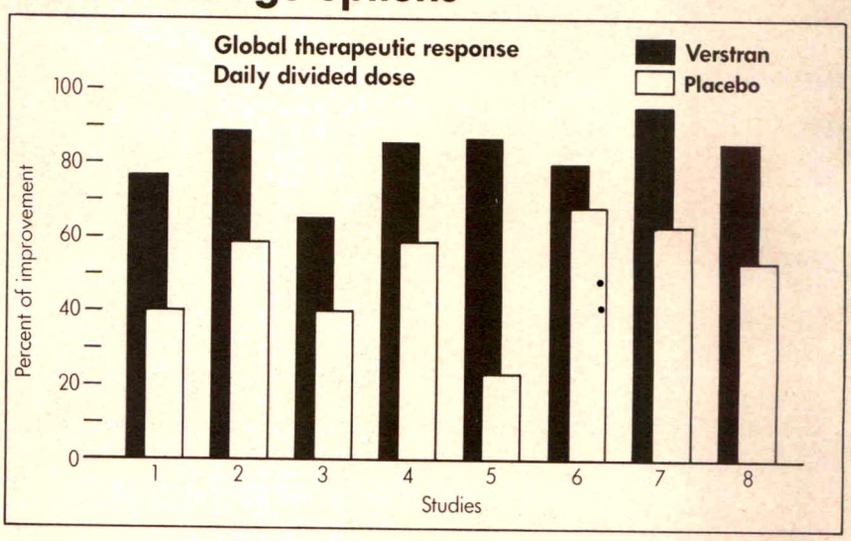
For those anxious patients who need the reassurance of multiple daily doses, the recommended starting dose is 10 mg B.I.D. This dose may be titrated gradually within a range of 20 to 60 mg per day in accordance with response.

Option 2: A once-a-day dose at bedtime

Taken in a single daily dose at bedtime, Verstran helps reduce the symptoms of anxiety—whether psychic or somatic in origin—throughout the following day. The usual starting dose for this regimen is 20 mg (two 10 mg tablets). Optimum dosage usually ranges from 20 to 40 mg daily.

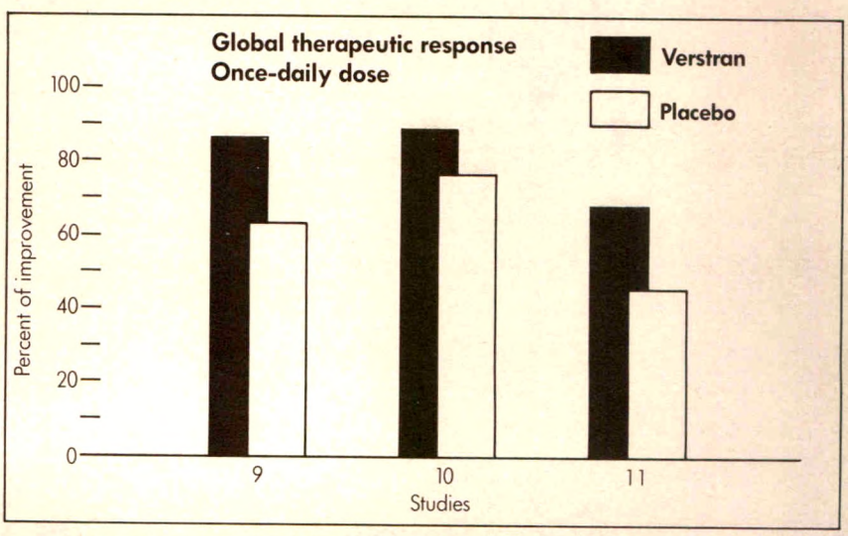


Eleven double-blind studies totaling 560 patients confirm the clinical efficacy of both dosage options*†



Four hundred and four patients suffering nonpsychotic clinical anxiety were given Verstran (228 patients) or placebo (176 patients) in a daily divided dose according to a randomized, double-blind design. Dosage of Verstran was within the recommended limits.

Target symptoms rated include: anxiety, depressive mood, irritability/hostility, tension and insomnia. Global Improvement Ratings were made at week 2, 3, or 4.



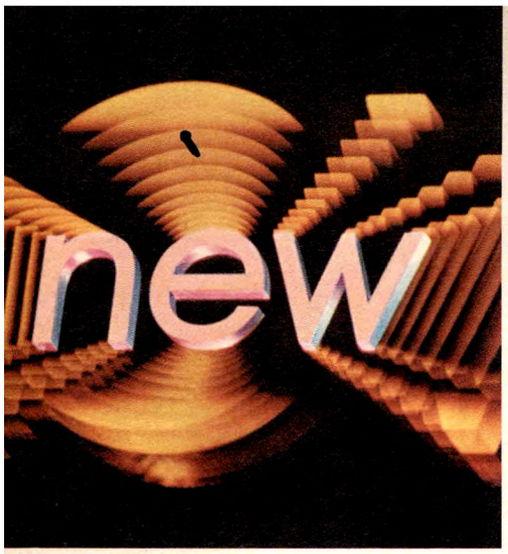
One hundred and fifty-six patients suffering nonpsychotic clinical anxiety were given Verstran (70 patients) or placebo (86 patients) once daily at bedtime according to a randomized, double-blind design. Dosage of Verstran was within the recommended limits.

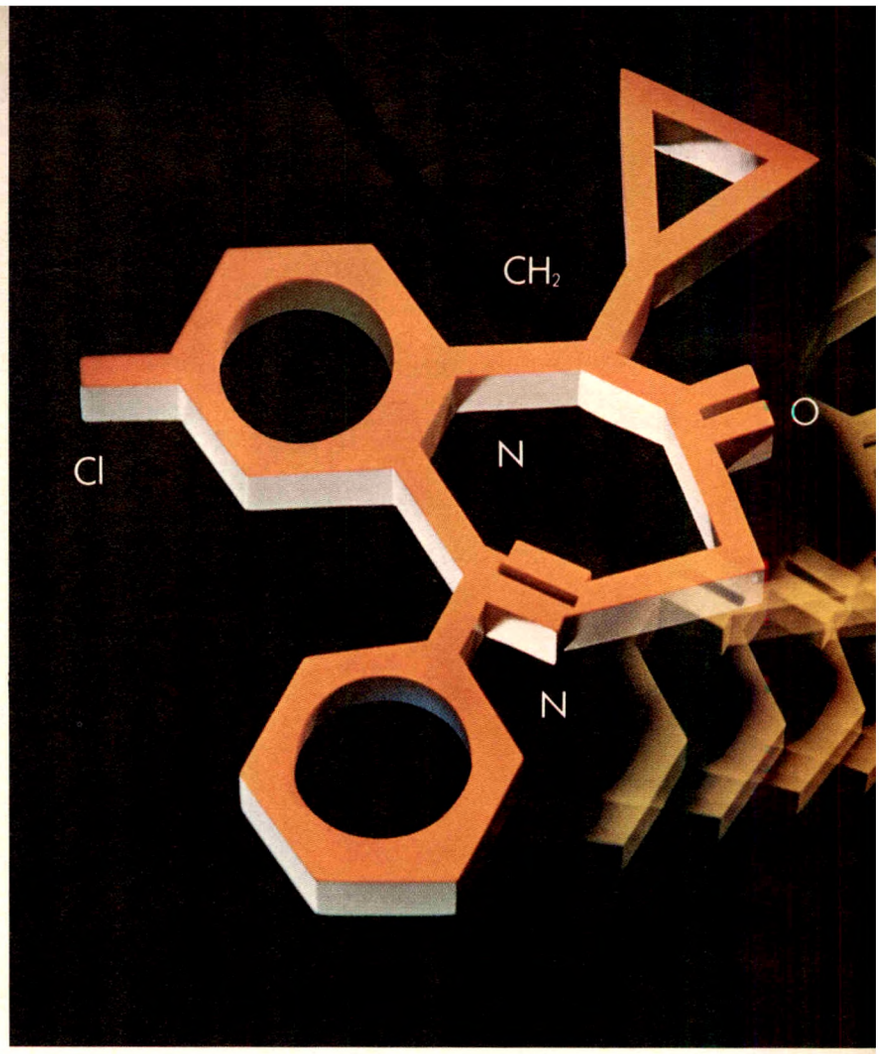
Target symptoms rated include: anxiety, depressive mood, irritability/hostility, tension and insomnia. Global Improvement Ratings were made at week 3 or 4.

*Data on file, Warner/Chilcott Medical Department.

†The number of patients cited represents those evaluated at final observation.

Please see last page of this advertisement for full product information.





New Verstran provides the simplicity of a single 10 mg tablet strength

Verstran is available in one 10 mg tablet strength only. Titration is simplified, and dosage is easy for the patient to remember. Scored tablets facilitate precise titration.



■ **New Verstran.**
Effective for the symptomatic relief of a wide range of anxiety conditions — whether psychic or somatic in origin.

■ **New Verstran.**
The option of fully effective divided daily or single nighttime dose.

■ **New Verstran.**
The simplicity of a single tablet strength.

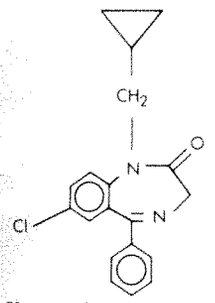
■ **New Verstran.**
Side effects are characteristic of benzodiazepine drugs.*

*Please see last page of this advertisement for warnings, precautions and adverse reactions.

Verstran® (prazepam) ℞

Caution: Federal law prohibits dispensing without prescription.

Description: Verstran (prazepam), a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1,4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows:



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (± 15 s.d.) hours before and 70 (± 10 s.d.) hours after multiple dosing—a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:

Use in Pregnancy and Lactation:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible aberrations of liver function tests have been reported, as have been slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though unlikely, may be controlled with Levophed® (levarterenol bitartrate) or Aramine® (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection, USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

How Supplied: Verstran (prazepam) 10 mg light blue, scored tablets in bottles of 100 (N 0047-0276-51).

STORE BETWEEN 59°-86° F (15°-30° C).

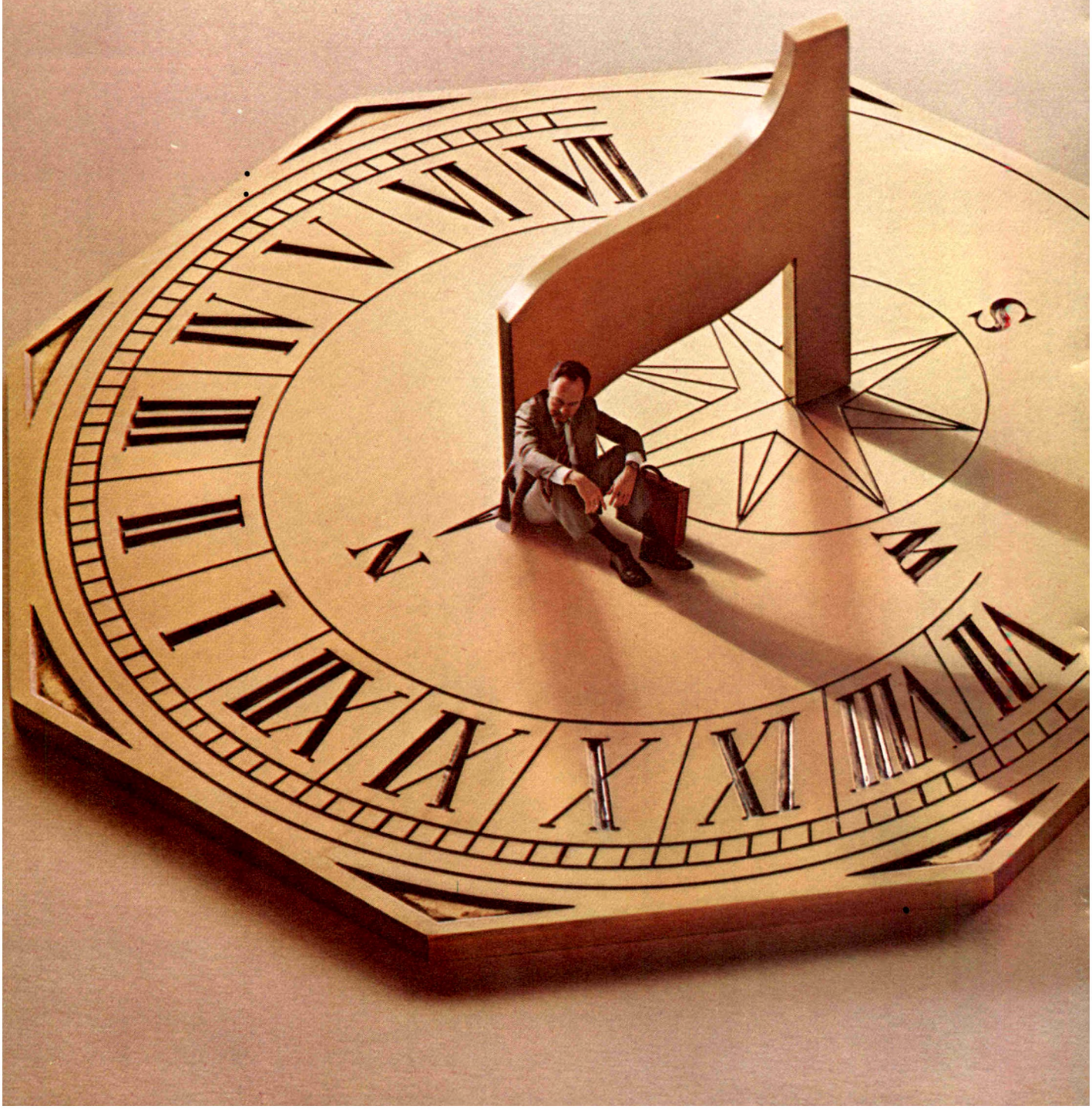
U.S. Patents 3192199, 3192200

Warner/Chilcott
Division,
Warner-Lambert Company
Morris Plains, N.J. 07950
VE-GP.71

VERSTRAN®
(prazepam) ℞

A new addition for the treatment of anxiety – whether psychic or somatic in origin

When depression is worse in the morning...



consider the antidepressant with the a.m./h.s. option

Early morning can be the depressed patient's "darkest hour"—the time when he most feels the need for help. Taken at the low point of the classic depressive diurnal rhythm, Norpramin (desipramine hydrochloride) in the a.m. may help patients get started with daily responsibilities, and encourage participation in various forms of psychotherapy.

The a.m. option
Clinical studies show that a single daily administration of Norpramin is as effective and well tolerated as the same quantity given as a divided dosage. This means that Norpramin may be given once daily, morning or bedtime, whichever is most appropriate to the patient's therapeutic need, drug response and activity patterns. A.M. dosage of Norpramin can be especially useful when your depressed patient feels more depressed in the early morning. Morning administration may also help reduce the potential for confusion when other agents are prescribed concomitantly at bedtime.

The h.s. option
For those patients who experience a sedative response to Norpramin, a bedtime dose is most appropriate. Evening administration of Norpramin rarely produces morning hangover.

Minimal daytime drowsiness —permits an active schedule
With Norpramin, problems of daytime drowsiness and morning hangover are largely avoided. Minimal daytime drowsiness can be especially important for patients who must perform daytime activities at home or at work.

Early therapeutic response— builds hope and provides encouragement
While full therapeutic effect may require two to three weeks, onset of action has often been observed in two to five days. Although results have been variable, the weight of scientific reports suggests a faster onset of action with desipramine than with either imipramine or amitriptyline.

Norpramin®
(desipramine hydrochloride tablets NF)

25 mg., 50 mg., 75 mg., 100 mg., 150 mg.

for the patient who must function during the day

Merrell

See following page for prescribing information.

Norpramin®
(desipramine hydrochloride tablets NF)

Brief Summary

Indications: Norpramin (desipramine hydrochloride tablets NF) is indicated for the relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Desipramine hydrochloride should not be given within two weeks of treatment with a monoamine oxidase inhibitor. Contraindications include the acute recovery period following myocardial infarction and hypersensitivity to the drug. Cross sensitivity with other dibenzazepines is a possibility.

Warnings: 1. Extreme caution should be used in patients: (a) with cardiovascular disease. (b) with a history of urinary retention or glaucoma. (c) with thyroid disease or those on thyroid medication. (d) with a history of seizure disorder. 2. This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. 3. *Use in Pregnancy:* Safe use during pregnancy and lactation has not been established. 4. *Use in Children:* Norpramin is not recommended for use in children. 5. This drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. Therefore, the patient should be cautioned accordingly. 6. In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions: This drug should be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with drugs of this class. If possible, dispense in child-resistant containers. It should be kept out of reach of children. Reduce dosage, or alter treatment, if serious adverse effects occur. Norpramin therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates and may cause exacerbation of psychosis in schizophrenic patients. Use cautiously with anticholinergic or sympathomimetic drugs. Response to alcoholic beverages may be exaggerated. In the concurrent administration of ECT and antidepressant drugs one should consider the possibility of increased risk relative to benefits. The sedative effects of Norpramin and benzodiazepines (e.g., chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. Discontinue as soon as possible prior to elective surgery because of possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients on desipramine hydrochloride. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is neutropenia.

Adverse Reactions: *Cardiovascular:* hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke. *Psychiatric:* confusional states (especially in the elderly), hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis. *Neurological:* numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus. *Anticholinergic:* dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, hypotonic bladder. *Allergic:* skin rash, petechiae, urticaria, itching, photosensitization, edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs. *Hematologic:* bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue. *Endocrine:* gynecomastia, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels. *Other:* jaundice (simulating obstructive), altered liver function; weight gain or loss, perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia. *Withdrawal symptoms:* Though not indicative of addiction, abrupt cessation after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: *Usual adult dose:* 100 mg. to 200 mg. per day. Dosages above 200 mg. per day are not recommended. *Initial therapy:* Should be administered at a low level and increased according to tolerance and response. It may be administered in divided doses or a single daily dose. *Maintenance:* Lower adequate dose once daily to maintain remission. *Adolescent and geriatric patient dose:* 25 mg. to 100 mg. per day, in single or divided doses. Dosages above 100 mg. are not recommended.

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Coverage and Utilization of Care for Mental Conditions Under Health Insurance —Various Studies, 1973–74

By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

This book reports six studies that add to the growing body of data demonstrating the feasibility of covering mental illness under health insurance. Although in some ways it may be considered a supplement to APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (Reed, Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

- Utilization of mental benefits under the Blue Cross and Blue Shield plan for federal employees, documenting that a "plateau" in the use of these benefits was reached in 1973–74.
- Comparison of benefits for mental and other illnesses under selected employee health benefit plans.
- Coverage of mental illness under collective bargaining agreements of selected unions.
- Utilization of care for mental conditions under the Canadian health insurance program, which gives the same coverage for mental as for other conditions.
- Updating of information on Blue Cross benefits for hospital care of mental illness.
- Data from selected Blue Cross and Blue Shield plans on coverage and utilization of mental conditions, with emphasis on major medical coverage.

80pp. Paperbound

Single copy, $4.00; 10–49 copies; $3.80 each; 50 or more copies, $3.20 each.

SPECIAL OFFER: One copy of this book (regular price, $4.00) and one copy of **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (regular price, $6.50) for $8.50.

Publications Services Division
American Psychiatric Association
1700 18th St., N.W., Washington, D.C. 20009

Please send me _____ copies of **COVERAGE AND UTILIZATION OF CARE FOR MENTAL CONDITIONS UNDER HEALTH INSURANCE—VARIOUS STUDIES, 1973–74**. Single copy, $4.00; 10–49 copies, $3.80 each; 50 or more copies, $3.20 each.

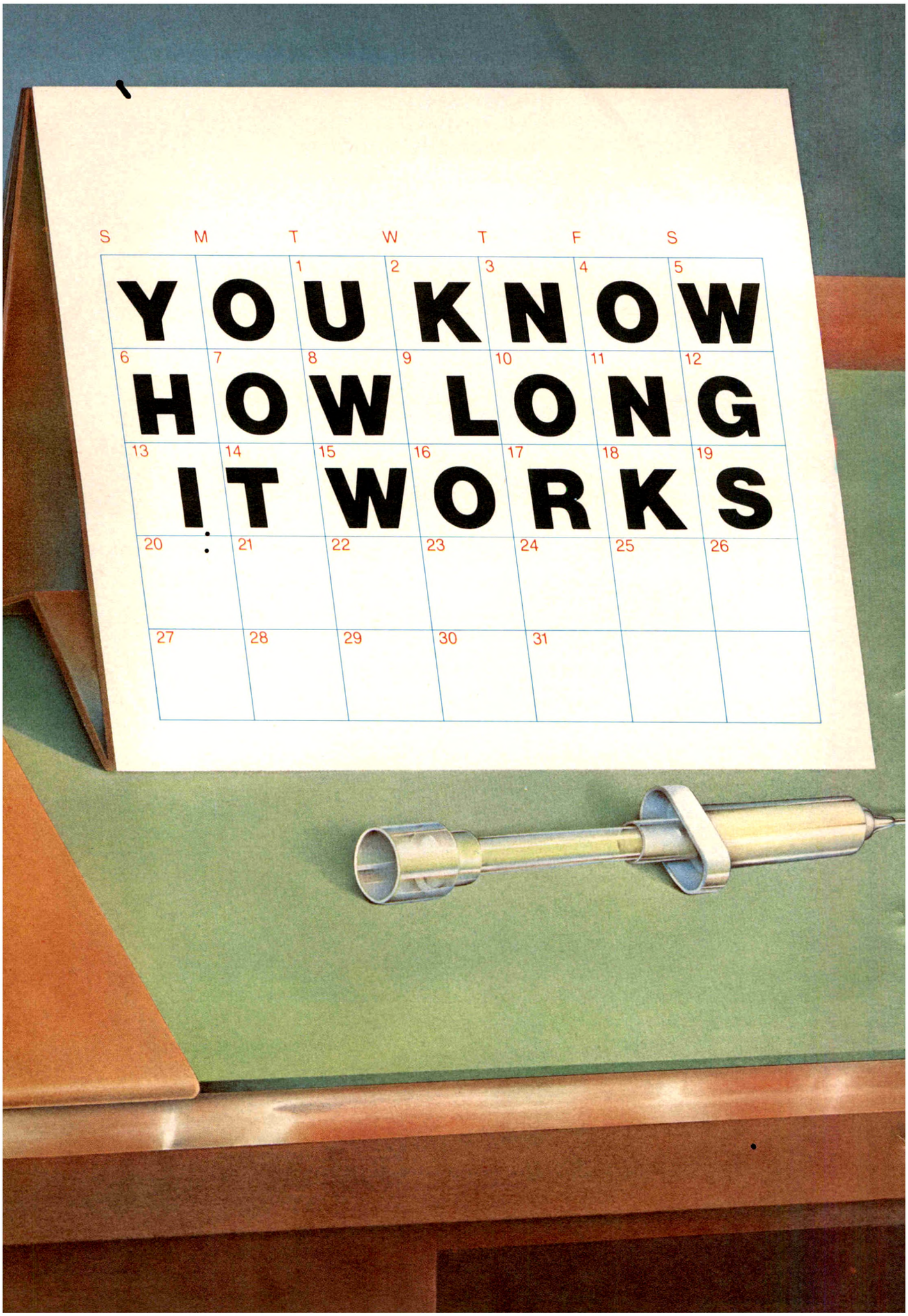
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S M T W T F S
YOU KNOW
HOW LONG
IT WORKS!

HERE'S HOW WELL IT WORKS

Rapidly effective in acute schizophrenia[1]

Average length of hospitalization was 6.1 days (range 4 to 8) for 13 acutely psychotic schizophrenic patients treated with Prolixin Decanoate using the rapid neuroleptization technique. Dosage the first 72 hours averaged 73.2 mg.

Psychotic symptoms diminished rapidly during the first week and continued to abate throughout the 2-month study.

Statistically significant improvement in 13 target symptoms of the Brief Psychiatric Rating Scale, including disorientation, hostility and uncooperativeness, occurred between pretreatment and final evaluation.

Often works where others fail...[2]

Of 52 chronic schizophrenic patients, hospitalized an average 17.5 years, 71% showed clinical improvement after Prolixin Decanoate therapy (25 mg every 7 to 14 days). 32 patients were refractory to prior therapy, including haloperidol combined with chlorpromazine or thioridazine.

General mobilization of the patients was the most significant effect. 7 patients could be discharged from the hospital. In 30 patients refractory to other medication, Prolixin Decanoate tended to inhibit withdrawal symptoms.

...at a fraction of the cost

Prolixin Decanoate therapy at a maintenance dose of 25 mg. every 28 days can cost from 53% to 69% less than the lowest maintenance dosages of several oral medications (see table below).

Comparison of estimated annual hospital costs for oral and injectable therapy, given the following examples:

Drug	Assumed adult dosage*	Form and potency	Cost per unit	Annual cost
Prolixin Decanoate®	12.5-100 mg every 28 days	5 ml vials 25 mg/ml	8¢† per mg	$ 13.00 – 104.00
Thorazine®(SKF brand of chlorpromazine)	300-600 mg per day	100 mg tablets bottles of 1000	5.1¢ per tab**	55.84 – 111.69
Mellaril®(Sandoz brand of thioridazine)	200-800 mg per day	100 mg tablets bottles of 1000	11.6¢ per tab**	84.68 – 338.72
Haldol®(McNeil brand of haloperidol)	5-50 mg per day	5 mg tablets bottles of 1000	20.1¢ per tab**	73.37 – 733.65
Navane®(Roerig brand of thiothixene)	20-60 mg per day	20 mg tablets bottles of 500	21.5¢ per tab**	78.48 – 235.43

*Dosage level and interval should be determined on an individual basis, in accordance with patient's response to the particular drug as well as the manufacturer's specific recommendations for the use of the product. **Based on prices listed in 1976 Red Book. †Based on Squibb list price of $9.95 per 5 ml vial.

1. Donlon PT, Axelrad AD, Tupin JP and Chien C-p: Comp Psychiat 17:369-376, 1976.
2. Christodoulidis H and Frangos H: Curr Ther Res 18:193-198, 1975.

See following page for brief summary.

PROLIXIN DECANOATE®
Fluphenazine Decanoate Injection

PROLIXIN DECANOATE®

Fluphenazine Decanoate Injection

Often works where others fail... at a fraction of the cost

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg. fluphenazine decanoate per ml. in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. These include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, and hyperreflexia; most often these are reversible, but they may be persistent. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of antiparkinsonian drugs such as benztropine mesylate or intravenous Caffeine and Sodium Benzoate Injection U.S.P., and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may have to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection U.S.P. is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

HOW SUPPLIED: 1 ml. Unimatic® single dose preassembled syringes and cartridge-needle units, and 5 ml. vials.

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Just Published . . .

The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

Paperback edition—$5.00 each, may be ordered from the AMERICAN PSYCHIATRIC ASSOCIATION, Publication Sales, 1700 18th St., N.W., Washington, D.C. 20009.

Hardback edition—$7.95 may be ordered from Basic Books, Inc., 10 East 53rd Street, New York, New York 10022.

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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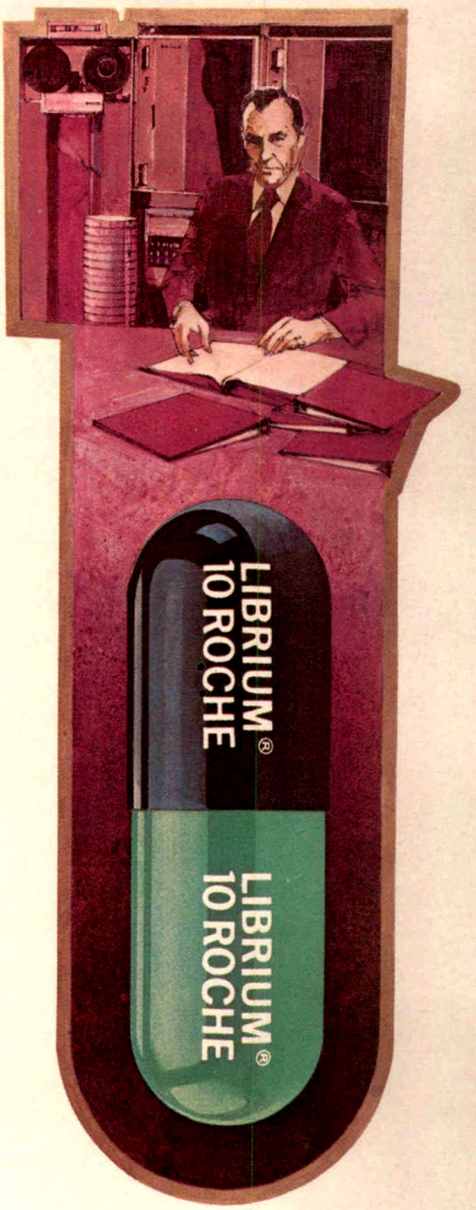
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THE ANXIETY-SPECIFIC

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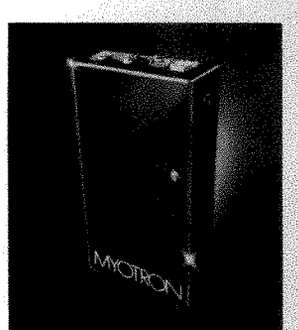
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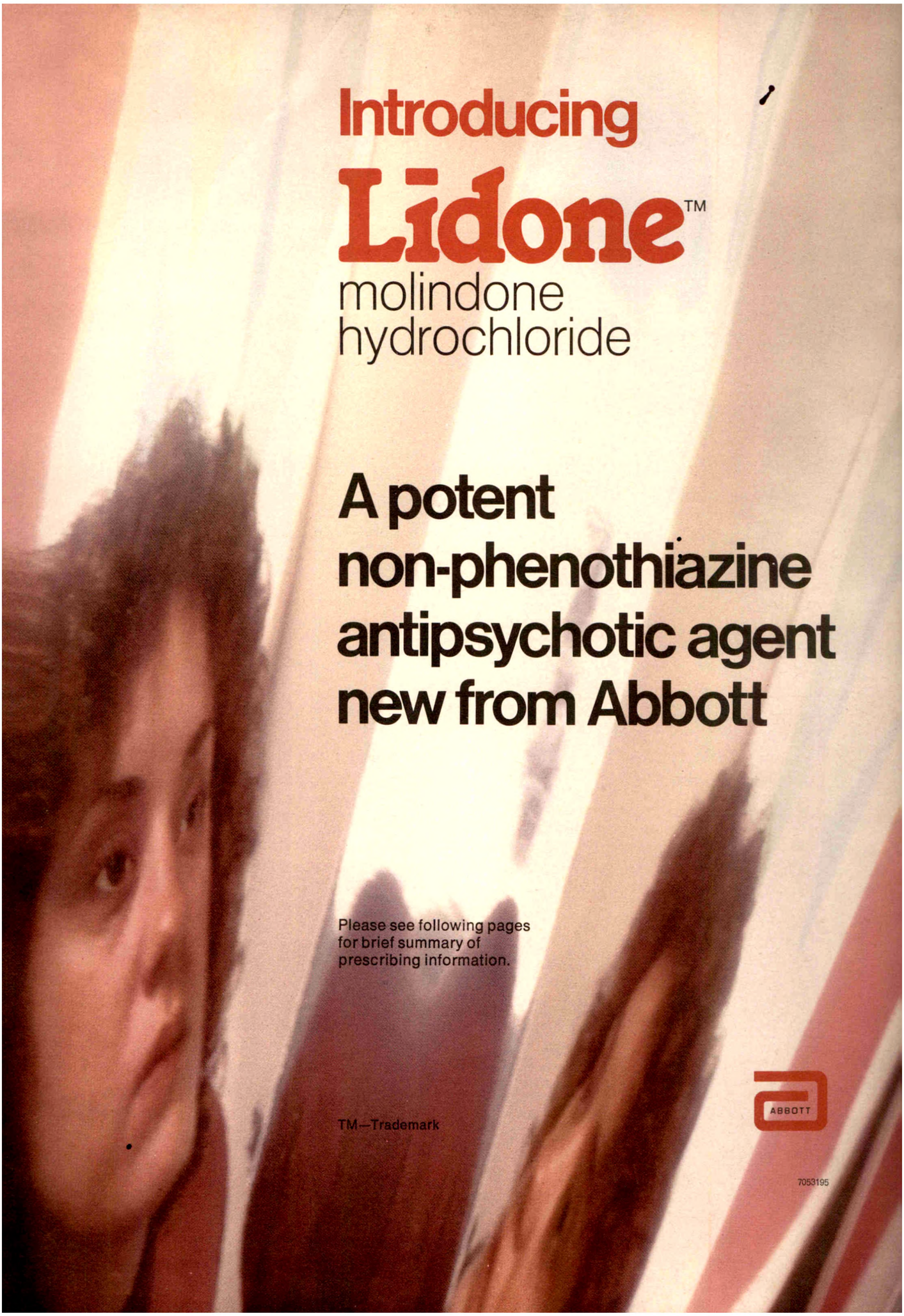
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for brief summary of
prescribing information.
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molindone
hydrochloride

Why Lidone™ belongs among your first-line antipsychotic agents

Because of its effectiveness

Demonstrated control of target symptoms. Lidone is a non-phenothiazine antipsychotic agent, indicated for management of the manifestations of schizophrenia. As such it offers a clinically proven oral treatment effective over a broad range of target symptoms. Improvement has been reported in:

- Conceptual disorganization[1,2,6,9]
- Unusual thought content[3,4,11,12]
- Hallucinations[3,6,9,11]
- Hostility[1,10,11]
- Bizarre motor behavior[3,11,12]
- Retardation[2,8,12]

Improvement in social outlook. Lidone has enabled patients to display progress in social behavior, and improved social competence and interest.[2,7,9,12,13] Interpersonal relationships have accordingly benefited.



Because of its degree of safety

Seldom causes problem sedation. Lidone is classed among the less-sedating group of major tranquilizers. Initial drowsiness usually subsides with continued use.

Extrapyramidal effects usually controlled. These usually respond to appropriate management without cessation of drug therapy.

Significant hypotension rare; hematologic effects limited. Less than 1% incidence of significant hypotension has been reported in clinical studies, and only rare reports of leucopenia and leucocytosis.

No serious ocular effects, impotence, or excessive weight gain. Unlike some antipsychotic agents, Lidone has not been associated with these conditions.

Persistent tardive dyskinesia not reported. Only a few cases of late-occurring dyskinesia have been noted (reversible upon discontinuation of therapy), and there have been no reports of persistent tardive dyskinesia.

Other side effects comparable to other agents. See Brief Summary for a complete statement of adverse reactions.



Lidone™

molindone hydrochloride

Because of its benefits

Patients accessible to psychotherapy. Patients are seldom over-sedated with Lidone. They tend to remain alert and therefore responsive to psychotherapy and rehabilitation.

Social relationships not disrupted. Improved social behavior has been noted, and side effects which are potentially disruptive to social or family relationships (e.g., weight gain, sexual dysfunction) have not been a problem.

Permits continuity of drug therapy. Few of the side effects require discontinuation of drug therapy.

Most economical non-phenothiazine. The relatively low milligram cost and high milligram potency of molindone make it least expensive of the potent new non-phenothiazine antipsychotics, and also less costly than many leading phenothiazines.[14]

Unique alternative for refractory patients. Molindone is unrelated to other antipsychotic drug families. Hence Lidone may benefit patients who are unresponsive to, or who cannot tolerate, other agents.

Please see following page for brief summary of prescribing information.

Because of its ease of administration

Simple Oral Regimen according to severity of presenting symptoms:

Dose strength	Schizophrenic manifestations	Dosage regimen
5 mg	mild	5 mg three or four times daily. (An increase to 15 mg three or four times daily may be required).
10 mg	moderate	10 mg three or four times daily. (An increase to 25 mg three or four times daily may be required.)
25 mg	severe	Daily dosage as high as 225 mg may be required.

Dosage Comparison at approximately equipotent dose levels:

Agent	Comparable doses[14]
Chlorpromazine	100 mg
Thioridazine	100 mg
Lidone	10 mg
Trifluoperazine	5 mg
Thiothixene	3 mg*
Haloperidol	2 mg
Fluphenazine	2 mg*

*Product not supplied in this exact strength.

Supplied in bottles of 100 capsules

Dose strength	Drug code	Color
5 mg	NDC 0074-5542-13	blue and cream
10 mg	NDC 0074-5543-13	red and cream
25 mg	NDC 0074-5544-13	brown and cream

7053195

Brief summary of prescribing information for

Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose —
20 mg/kg/day — 2 weeks, no adverse effect
40 mg/kg/day — 2 weeks, no adverse effect

Pregnant Mice oral dose —
20 mg/kg/day — 9 days, slight increase resorptions
40 mg/kg/day — 9 days, slight increase resorptions

Pregnant Rabbits oral dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial and maintenance doses of LIDONE (molindone hydrochloride) should be individualized, and the minimal effective dose should be employed. Elderly and debilitated patients should be started on lower dosage.

DOSAGE SCHEDULE, BASED ON SEVERITY OF SYMPTOMATOLOGY

1. Mild — 5 mg three or four times a day; an increase to 15 mg three or four times a day may be required.
2. Moderate — 10 mg three or four times a day; an increase to 25 mg three or four times a day may be required.
3. Severe — daily dosage as high as 225 mg may be required.

DRUG INTERACTIONS — Potentiation of drugs administered concurrently with LIDONE (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when LIDONE is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE — Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of LIDONE (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of LIDONE (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by LIDONE in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized LIDONE from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of LIDONE is excreted unmetabolized in the urine.) However, poor response of the patient may justify use of these procedures.

While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized LIDONE in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

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TM — Trademark

*Benadryl — Trademark, Parke Davis and Co.
*Artane — Trademark, Lederle Laboratories
*Cogentin — Trademark, Merck Sharp & Dohme
*Akineton — Trademark, Knoll Pharmaceutical Co.

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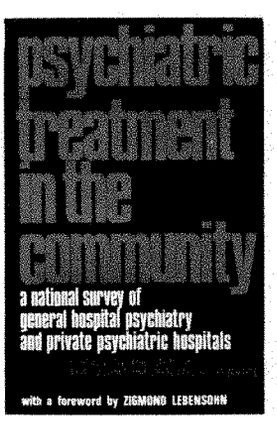
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ACTIONS—Pharmacologically, TRANXENE (clorazepate dipotassium) has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine.

INDICATIONS—TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE for long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS—TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS—TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS—In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS—The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION—

For symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg TRANXENE initially, followed by 30 to 60 mg in divided doses; 2nd 24 hours, 45 to 90 mg in divided doses; 3rd 24 hours, 22.5 to 45 mg in divided doses; 4th day, 15 to 30 mg in divided doses. Thereafter gradually reduce to 7.5 to 15 mg daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS—If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE (clorazepate dipotassium) is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE—Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed, since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY—Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption–Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED—TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

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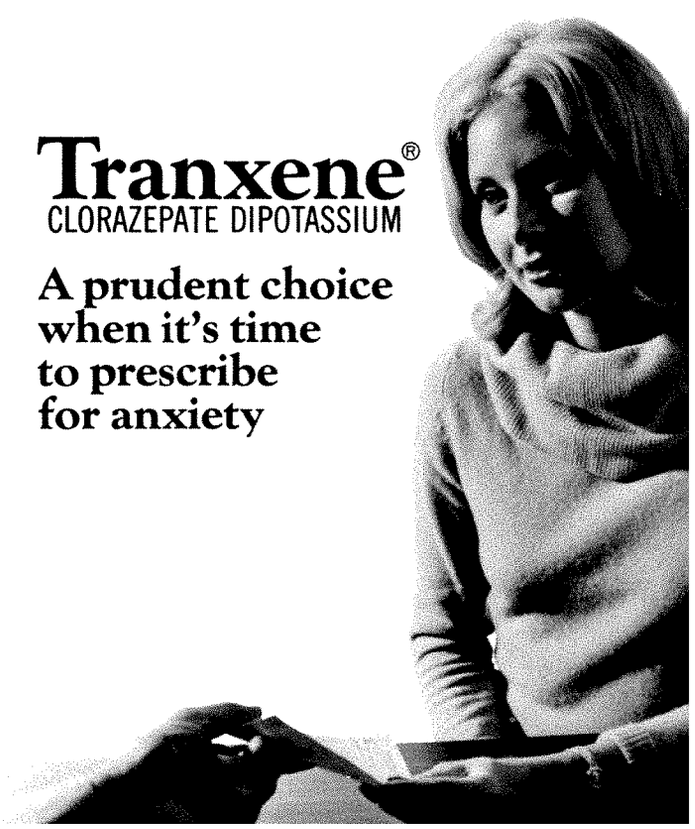
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Please see overleaf for brief summary of prescribing information.

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In this issue

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By Anne R. Somers

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Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

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Caution is recommended when 'Thorazine' is administered for the treatment of vomiting in children.

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Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

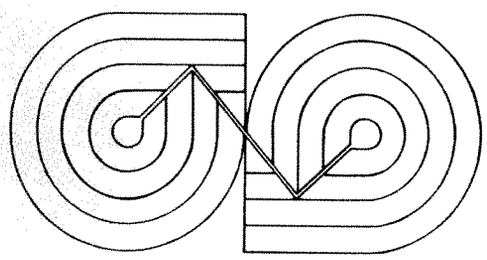
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1. Berne E: Principles of Group Treatment. New York, Oxford University Press, 1966, p 26
2. Schildkraut JJ: Tranylcypromine: effects on norepinephrine metabolism in rat brain. Am J Psychiatry 126:925–931, 1970
3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
4. Brosin H: Communication systems of the consultation process, in The Psychiatric Consultation. Edited by Mendel W, Solomon P. New York, Grune & Stratton, 1968, pp 1–12

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Actions. Navane is a psychotropic agent of the thioxanthene series. Navane possesses certain chemical and pharmacological similarities to the piperazine phenothiazines and differences from the aliphatic group of phenothiazines. Navane's mode of action has not been clearly established.

Indications. Navane is effective in the management of manifestations of psychotic disorders.

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy* – Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children–The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration – As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate–In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg, of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% w/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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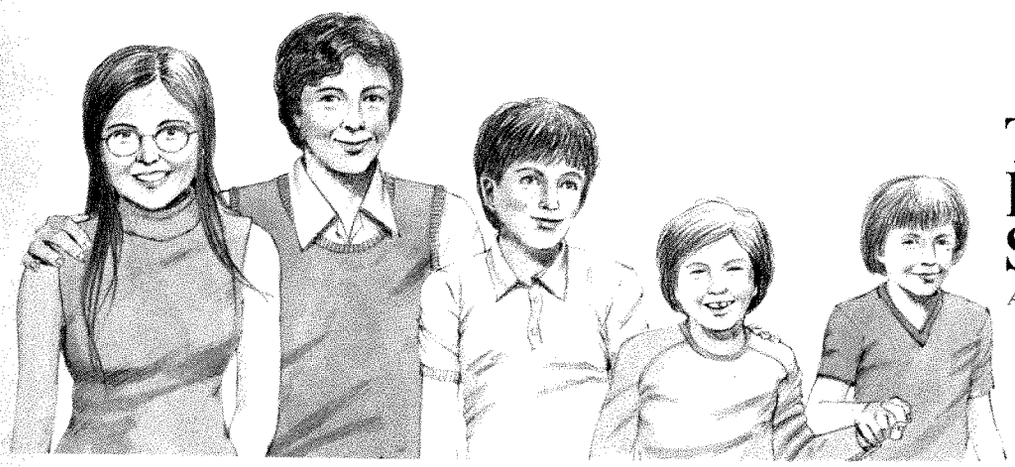
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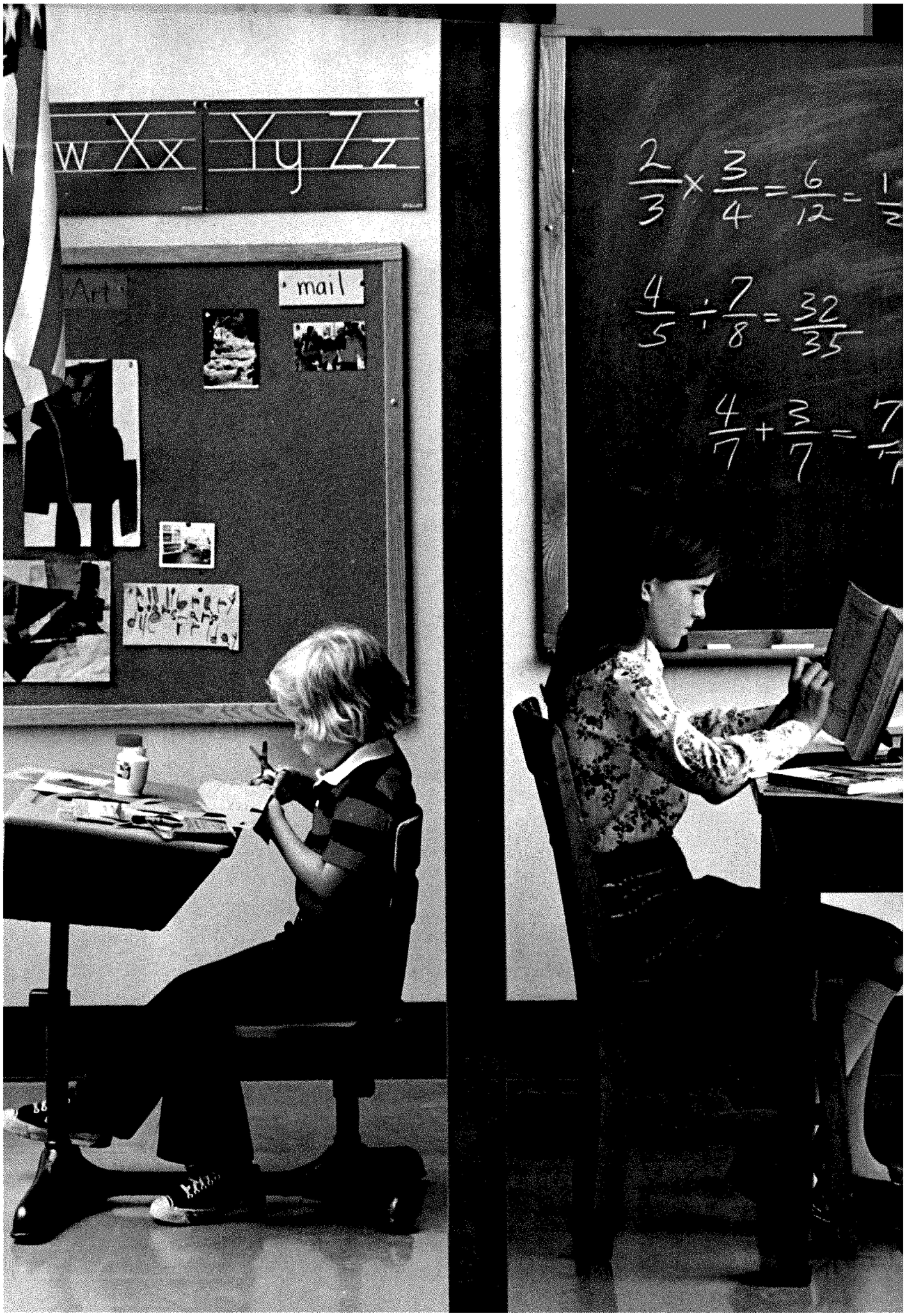
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Xx Yy Zz
Art
mail
$\frac{2}{3} \times \frac{3}{4} = \frac{6}{12} =$
$\frac{4}{5} \div \frac{7}{8} = \frac{32}{35}$
$\frac{4}{7} + \frac{3}{7} =$

The vulnerable ages

The first epileptic seizure is most likely to occur during early childhood and at the onset of puberty

About 9 out of 10 epileptics experience their first seizure before the age of 20—with the highest incidence between 5 and 7, when children start school, and at the onset of puberty, a time of physiological and psychic turmoil.[1] The most common type, grand mal, occurs in approximately 75% of epileptic children,[1] and more than 50% of patients who suffer initially from petit mal develop grand mal seizures before they reach the age of 16.[2]

Mysoline (primidone) for control of grand mal, psychomotor and focal epilepsy

At the onset and afterwards — used alone or as concomitant therapy, MYSOLINE may reduce the frequency and severity of major motor seizures—or even eliminate them. *Excellent* for control of grand mal. Valuable for control of psychomotor[1,3,4] and focal epilepsy as well.[5]

Add Mysoline when control with other anticonvulsants is inadequate — As concomitant therapy, MYSOLINE can improve seizure control in grand mal and psychomotor epilepsy. The combined use of phenobarbital, diphenylhydantoin, and MYSOLINE may have additive anticonvulsant effects without additive side effects.[6]

Change to Mysoline when other anticonvulsants fail — A changeover to MYSOLINE is frequently warranted when other anticonvulsants must be discontinued because of important side effects, or when grand mal seizures are refractory to phenobarbital, with or without diphenylhydantoin.[7]

Ayerst®

Mysoline® (primidone)

Tablets 250 mg.
50 mg.
Suspension 250 mg./5 cc.

May be the start of a better life for the epileptic

See following page of advertisement for prescribing information.

7538

Mysoline® (primidone)

may be the start of a better life for the epileptic

initial and maintenance therapy for
grand mal, psychomotor and focal epilepsy

BRIEF SUMMARY
(For full prescribing information, see package circular.)

MYSOLINE® Brand of PRIMIDONE
Anticonvulsant

ACTIONS: MYSOLINE acts on the central nervous system to raise seizure threshold or alter seizure pattern. The mechanism(s) of action of anticonvulsant drugs is not known.

Primidone has anticonvulsant activity *per se*. In addition, its two metabolites possess anticonvulsant qualities. The major metabolite is phenylethylmalonamide (PEMA); the other is phenobarbital. In addition to its own anticonvulsant potential, PEMA potentiates phenobarbital.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several days before it can be assessed.

Use in pregnancy: Recent reports strongly suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Reference has been made to primidone in several cases in which it was used *in combination* with other anticonvulsants; but its teratogenicity has not been conclusively demonstrated. The possibility exists that other factors, *e.g.*, genetic factors or the epileptic condition, may contribute to the higher incidence of birth defects. The data also indicate that the great majority of mothers receiving anticonvulsant medication deliver normal infants.

Anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and risk to both mother and the unborn child.

When the nature, frequency, and severity of the seizures do not pose a clear threat to the patient, good medical practice requires that the physician weigh the expected therapeutic benefit of anticonvulsant therapy against possible risk on an individual basis.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

The physician should weigh all of the foregoing considerations when treating and counseling epileptic women of childbearing potential.

PRECAUTIONS: The total daily dosage should not exceed 2 Gm. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid, 15 mg. daily, without necessity of discontinuing medication.

DOSAGE AND ADMINISTRATION: The average adult dose is 0.75 to 1.5 Gm. per day. The initial dose is 250 mg. Increments of 250 mg. are added, usually at weekly intervals, to tolerance, or therapeutic effectiveness, up to daily doses not exceeding 2.0 Gm. A typical dosage schedule for the introduction of MYSOLINE (primidone) is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg. daily at bedtime	*2nd Week* 250 mg. b.i.d.
3rd Week 250 mg. t.i.d.	*4th Week* 250 mg. q.i.d.

In children under 8 years of age, maintenance levels are established by a similar schedule, but at one-half the adult dosage. It is best to begin with 125 mg., with gradual weekly increases of 125 mg. a day, to a daily total usually between 500 mg. and 750 mg.

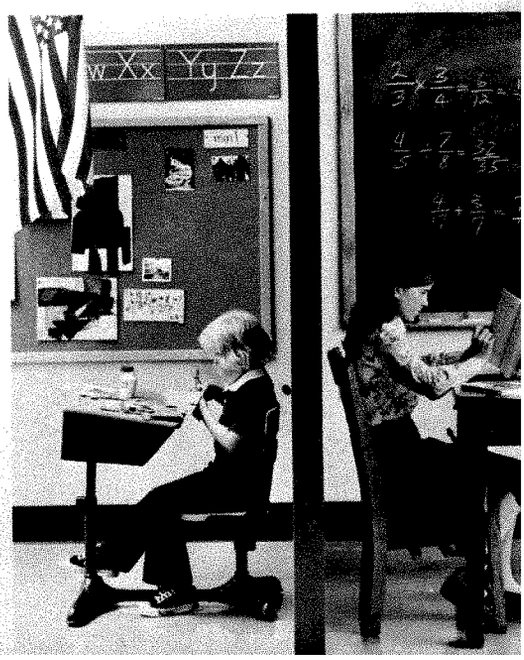
In patients already receiving other anticonvulsants: MYSOLINE should be gradually increased as dosage of the other drug(s) is maintained or gradually decreased. This regimen should be continued until satisfactory dosage level is achieved for combination, or the other medication is completely withdrawn. When therapy with this product alone is the objective, the transition should not be completed in less than two weeks.

MYSOLINE 50 mg. Tablet can be used to practical advantage when small fractional adjustments (upward or downward) may be required, as in the following circumstances:

- for initiation of combination therapy
- during "transfer" therapy
- for added protection in periods of stress or stressful situations that are likely to precipitate seizures (menstruation, allergic episodes, holidays, etc.)

HOW SUPPLIED: MYSOLINE *Tablets*—No. 430—Each tablet contains 250 mg. of primidone (scored), in bottles of 100 and 1,000. Also in unit dose package of 100. No. 431—Each tablet contains 50 mg. of primidone (scored), in bottles of 100 and 500. MYSOLINE *Suspension*—No. 3850—Each 5 cc. (teaspoonful) contains 250 mg. of primidone, in bottles of 8 fluid ounces.

References: 1. Livingston, S.: Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence, Springfield, Ill., Charles C Thomas, 1972, pp. 6, 7, 584. 2. Grossman, H.J.: Ill. Med. J. *135*:260 (Mar.) 1969. 3. Scholl, M.L., in Conn, H.F.: Current Therapy 1973, Philadelphia, Saunders, 1973, pp. 675-7. 4. Metrick, S.: C.M.D. *37*:491 (Jan.) 1970. 5. Forster, F.M.: Med. Clin. North Am. *47*:1579 (Nov.) 1970. 6. White, P.T.: Wis. Med. J. *68*:178 (Apr.) 1969. 7. Millichap, J.G.: Drug Ther. *1*:15 (Oct.) 1971.



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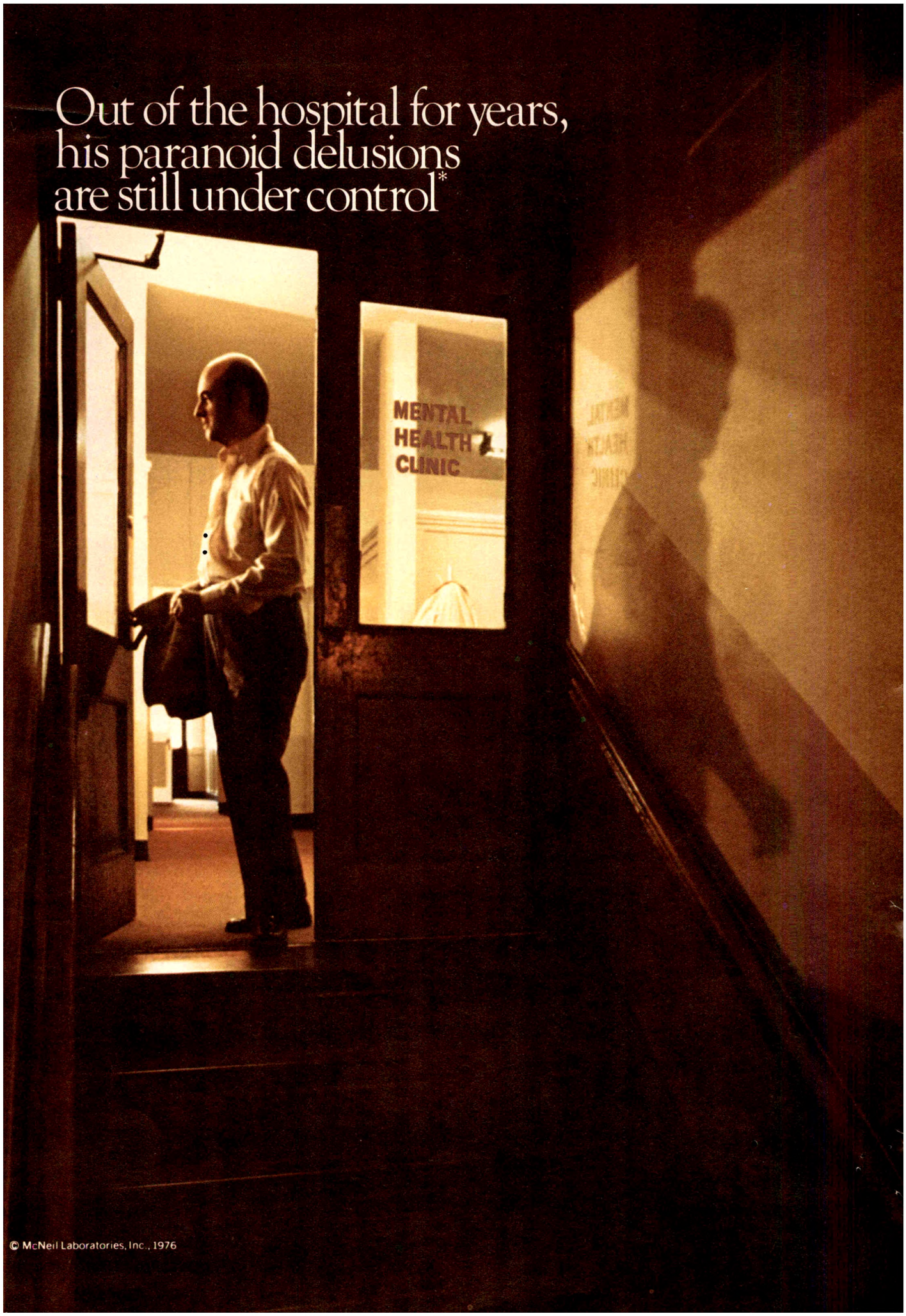
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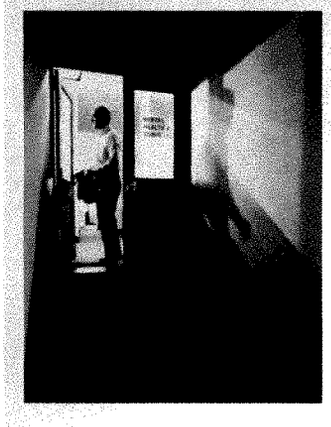
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Haldol® (haloperidol)

tablets/concentrate/injection

For long-term control of psychotic symptoms with minimal risk of toxicity

A Dosage Form for Every Need:

5 tablet strengths for convenience in individualizing dosage: ½ mg., 1 mg., 2 mg., 5 mg. and 10 mg.

A tasteless, odorless, colorless liquid concentrate for better patient acceptability: 2 mg. per ml.

A rapid-acting injection for psychiatric emergencies: 5 mg. per ml., with 1.8 mg. methylparaben and 0.2 mg. propylparaben per ml., and lactic acid for pH adjustment to 3.4±0.2.

Indications: HALDOL haloperidol is indicated for use in the management of manifestations of psychotic disorders.

It is also indicated for the control of tics and vocal utterances of Gilles de la Tourette's syndrome.

Contraindications: HALDOL haloperidol is contraindicated in patients who are severely depressed, comatose, have CNS depression due to alcohol or other centrally-acting depressants, have Parkinson's disease or are hypersensitive to this drug.

Warnings: ***Usage in Pregnancy:*** Safe use of HALDOL haloperidol in pregnancy and lactation has not been established; therefore, its use in pregnancy, in nursing mothers, or in women of childbearing potential requires that the possible benefits of the drug be weighed against the potential hazards. A case of phocomelia in an infant whose mother received haloperidol along with a number of other medications during the first trimester of pregnancy has been reported (a causal relationship was not established in this case). Animals receiving 2 to 20 times the maximum human dose of haloperidol orally and/or parenterally showed increased incidence of resorption, reduced fertility, delayed delivery, dose-related pup mortality (presumably due to lack of maternal care reflecting CNS depression).

Usage in Children: Safety and effectiveness in children have not been established; therefore, this drug is not recommended for use in the pediatric age group.

General: Cases of bronchopneumonia, some fatal, have followed the use of major tranquilizers, including haloperidol. It has been postulated that lethargy and decreased sensation of thirst may lead to dehydration, hemoconcentration and reduced pulmonary ventilation. If these signs and symptoms appear, especially in the elderly, the physician should institute remedial therapy promptly. Although not reported with HALDOL haloperidol, decreased serum cholesterol and/or cutaneous and ocular changes have been reported in patients receiving chemically-related drugs. HALDOL haloperidol may impair the mental and/or physical abilities required for the performance of hazardous tasks such as operating machinery or driving a motor vehicle. The ambulatory patient should be warned accordingly. The use of alcohol should be avoided due to possible additive effects and hypotension.

Precautions: HALDOL haloperidol should be administered cautiously to patients: (1)—with severe cardiovascular disorders, because of the possibility of transient hypotension and/or precipitation of anginal pain. Should hypotension occur and a vasopressor be required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur. (2)—receiving anticonvulsant medication, because HALDOL haloperidol may lower the convulsive threshold. Adequate anticonvulsant therapy should be maintained concomitantly. (3)—with known allergies, or with a history of allergic reactions to drugs. (4)—receiving anticoagulants, since an isolated instance of interference occurred with the effects of one anticoagulant (phenindione).

If concomitant antiparkinson medication is required, it may have to be continued after HALDOL haloperidol is discontinued because of the difference in excretion rates. If both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson agents, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used to control mania in cyclic disorders there may be a rapid mood swing to depression.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions*—Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which usually were mild to moderately severe and reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Administration of antiparkinson drugs may be required for control of such reactions. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases. *Persistent Tardive Dyskinesia*—Tardive dyskinesia may appear during long-term therapy or after therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear irreversible. There is no known effective treatment. All antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent. *Other CNS Effects*—Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms including hallucinations. ***Cardiovascular Effects:*** Tachycardia and hypotension. ***Hematologic Effects:*** Reports have appeared of mild and usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis. Agranulocytosis has rarely been reported and then only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice have been reported, although a causal relationship has not been established. ***Dermatologic Reactions:*** Maculopapular and acneiform skin reactions and isolated cases of photosensitivity and loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

Complete dosage information available in insert which accompanies each package (or on request).

The use of the injectable form is intended for the acutely agitated psychotic patient with moderately severe to very severe symptoms.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed. 9-74

References:

1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. **2.** Sugerman, A.A. et al.: Am. J. Psychiatry *129*:1190 (June) 1964. **3.** Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. **4.** Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. **5.** Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. **6.** Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973. **7.** Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966.

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Haldol®
(haloperidol)
tablets/concentrate/injection

For long-term control with minimal risk of toxicity

Highly effective in a wide range of psychotic symptoms,[1-7]
such as hallucinations, delusions, suspiciousness, hostility, mania, psychomotor agitation, etc., in both acute and chronic disorders.

Usually leaves patients relatively alert and responsive...[1-3]
better able to cope with job and family responsibilities...as well as easier to reach with supportive and rehabilitative measures.

Permits aggressive titration to effective dosage levels,[1,4]
up to 100 mg/day orally (with minimal risk of usual troublesome reactions) to achieve optimal response when patient is inadequately controlled at lower dosage...to help you rapidly control and stabilize new patients, promptly regain control during periods of exacerbation.

Common side effects easily controlled.[1,3-6]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Reduces risk of certain troublesome reactions.[2-5,7]
Transient hypotension occurs rarely and severe orthostatic hypotension has not been reported. Marked sedation is rare, although some instances of drowsiness have been reported. In addition, with chronic use, it is unlikely to cause hepatic damage, serious hematologic reactions, photosensitivity reactions and skin rashes...and has minimal effect on renal function.

Please turn page for information relating to Indications, Contraindications, Warnings, Precautions and Adverse Reactions.

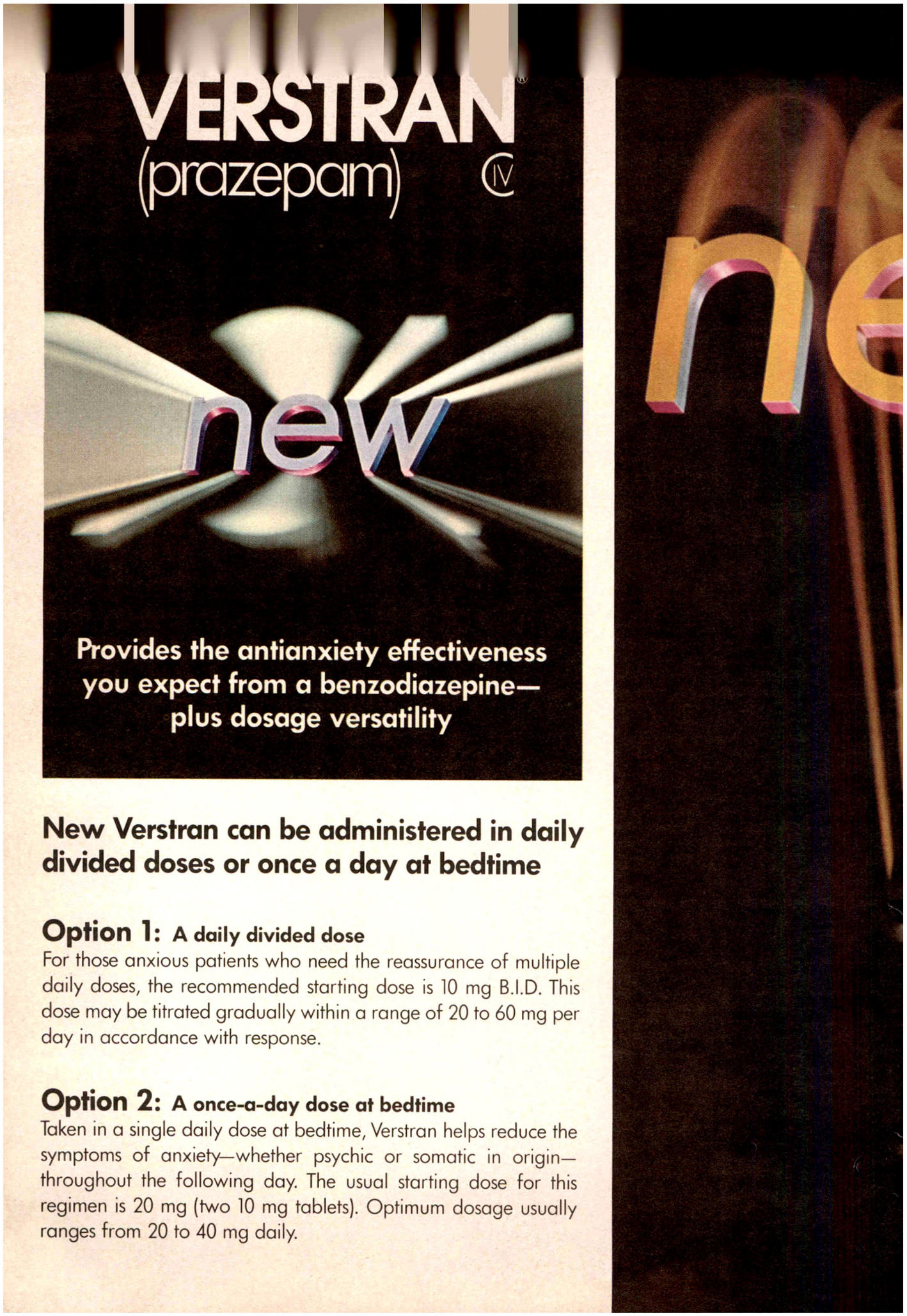
Important: Full directions for use should be read before HALDOL haloperidol is administered or prescribed.

Warner/Chilcott Introduces



VERSTRAN®
(prazepam) C IV

A new addition for the treatment of anxiety



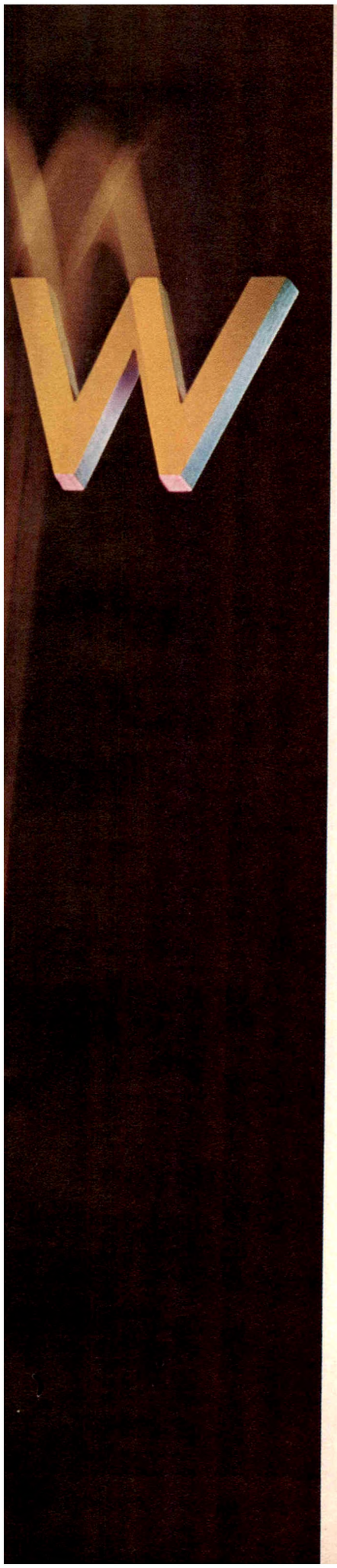
New Verstran can be administered in daily divided doses or once a day at bedtime

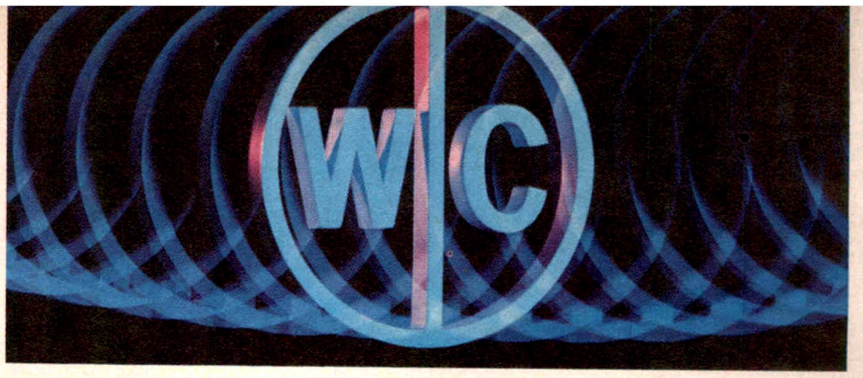
Option 1: A daily divided dose

For those anxious patients who need the reassurance of multiple daily doses, the recommended starting dose is 10 mg B.I.D. This dose may be titrated gradually within a range of 20 to 60 mg per day in accordance with response.

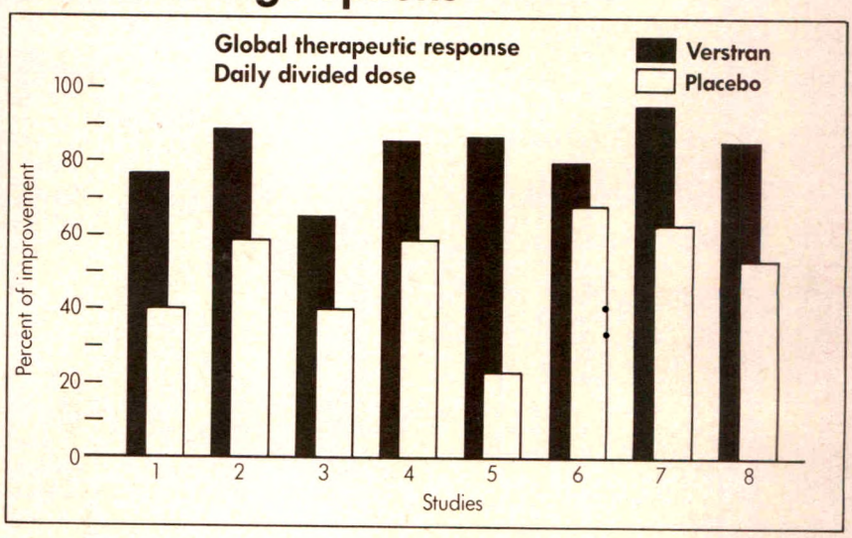
Option 2: A once-a-day dose at bedtime

Taken in a single daily dose at bedtime, Verstran helps reduce the symptoms of anxiety—whether psychic or somatic in origin—throughout the following day. The usual starting dose for this regimen is 20 mg (two 10 mg tablets). Optimum dosage usually ranges from 20 to 40 mg daily.



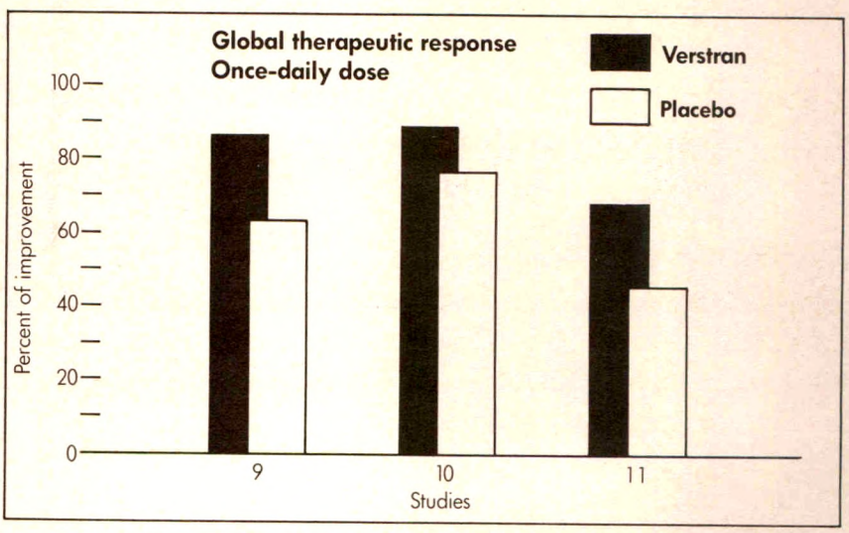


Eleven double-blind studies totaling 560 patients confirm the clinical efficacy of both dosage options*†



Four hundred and four patients suffering nonpsychotic clinical anxiety were given Verstran (228 patients) or placebo (176 patients) in a daily divided dose according to a randomized, double-blind design. Dosage of Verstran was within the recommended limits.

Target symptoms rated include: anxiety, depressive mood, irritability/hostility, tension and insomnia. Global Improvement Ratings were made at week 2, 3, or 4.



One hundred and fifty-six patients suffering nonpsychotic clinical anxiety were given Verstran (70 patients) or placebo (86 patients) once daily at bedtime according to a randomized, double-blind design. Dosage of Verstran was within the recommended limits.

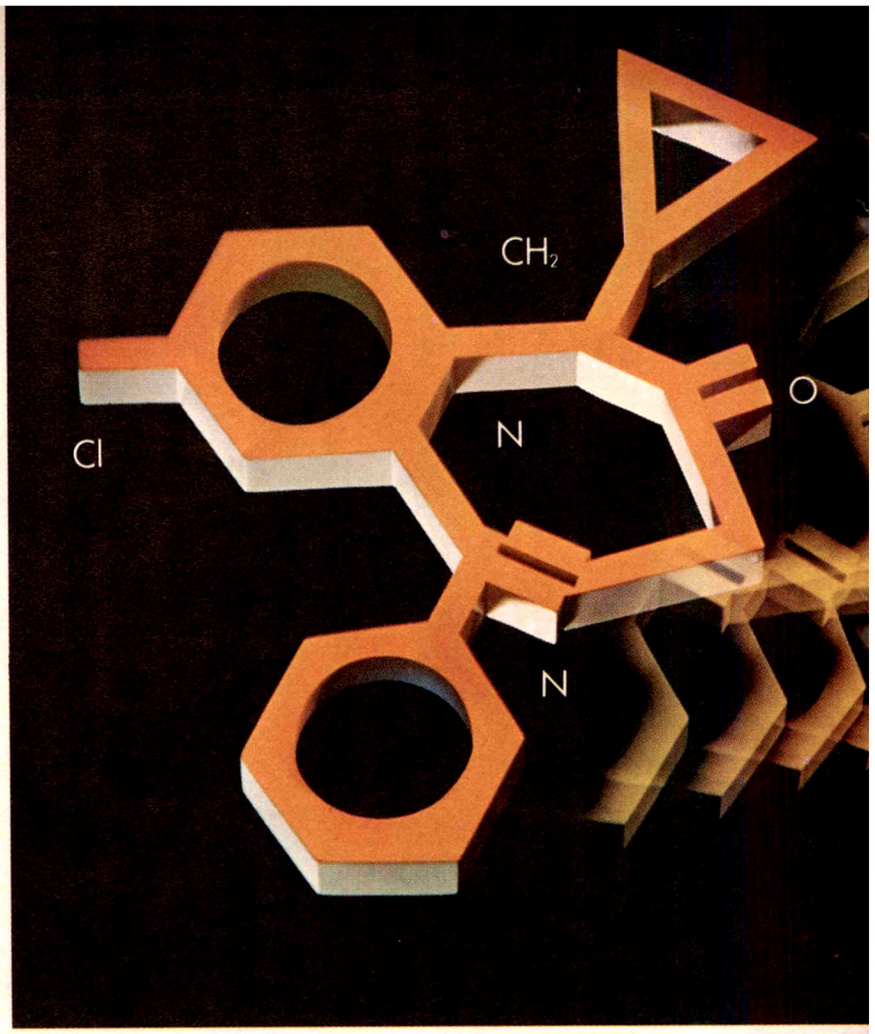
Target symptoms rated include: anxiety, depressive mood, irritability/hostility, tension and insomnia. Global Improvement Ratings were made at week 3 or 4.

*Data on file, Warner/Chilcott Medical Department.

†The number of patients cited represents those evaluated at final observation.

Please see last page of this advertisement for full product information.





New Verstran provides the simplicity of a single 10 mg tablet strength

Verstran is available in one 10 mg tablet strength only. Titration is simplified, and dosage is easy for the patient to remember. Scored tablets facilitate precise titration.



■ **New Verstran.**
Effective for the symptomatic relief of a wide range of anxiety conditions — whether psychic or somatic in origin.

■ **New Verstran.**
The option of fully effective divided daily or single nighttime dose.

■ **New Verstran.**
The simplicity of a single tablet strength.

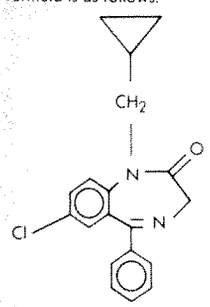
■ **New Verstran.**
Side effects are characteristic of benzodiazepine drugs.*

*Please see last page of this advertisement for warnings, precautions and adverse reactions.

Verstran® (prazepam) Ⓒ

Caution: Federal law prohibits dispensing without prescription.

Description: Verstran (prazepam), a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1,4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows.



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (± 15 s.d.) hours before and 70 (± 10 s.d.) hours after multiple dosing—a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:

Use in Pregnancy and Lactation:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible aberrations of liver function tests have been reported, as have been slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though unlikely, may be controlled with Levophed® (levarterenol bitartrate) or Aramine® (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection, USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

How Supplied: Verstran (prazepam) 10 mg light blue, scored tablets in bottles of 100 (N 0047-0276-51).

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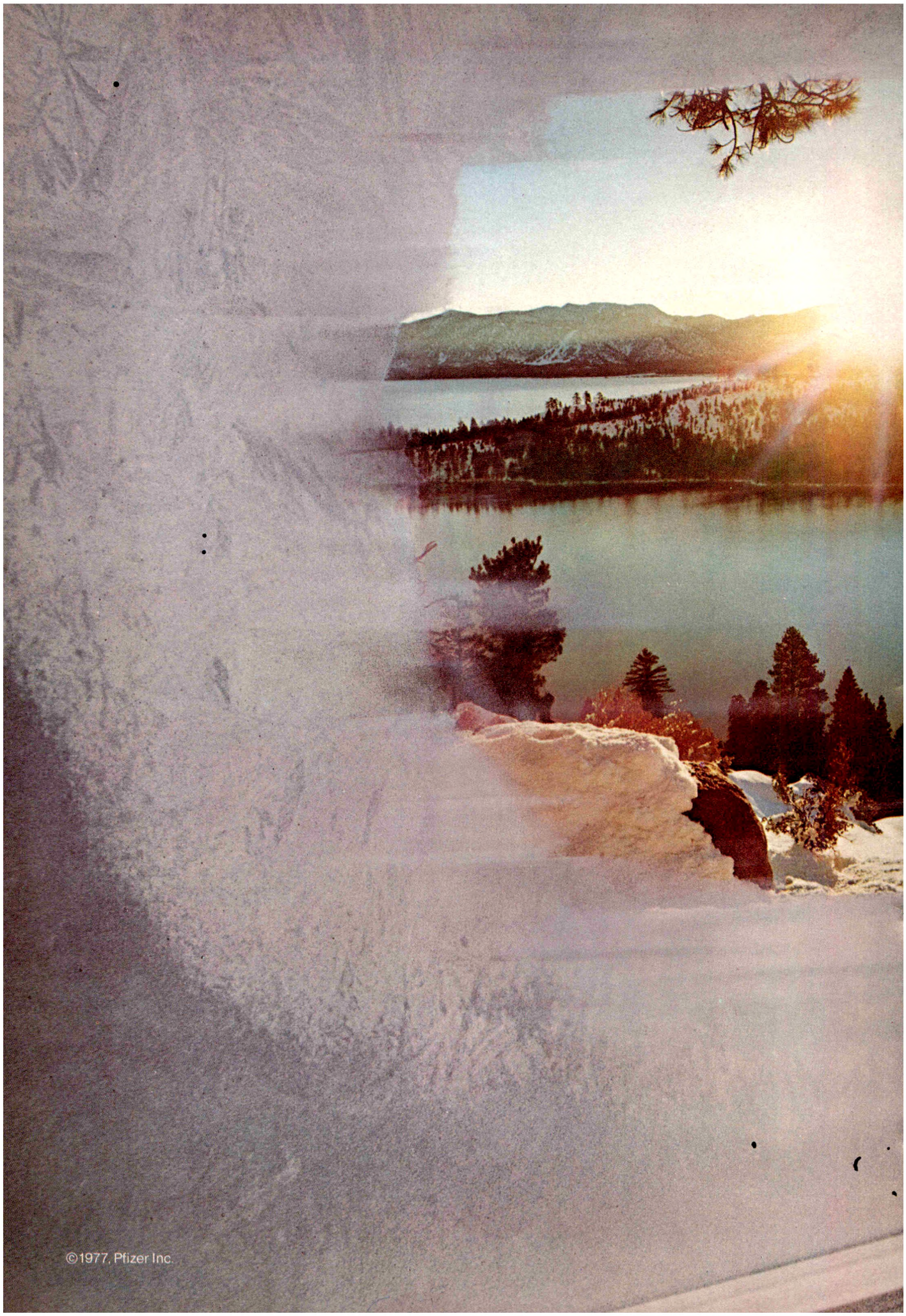
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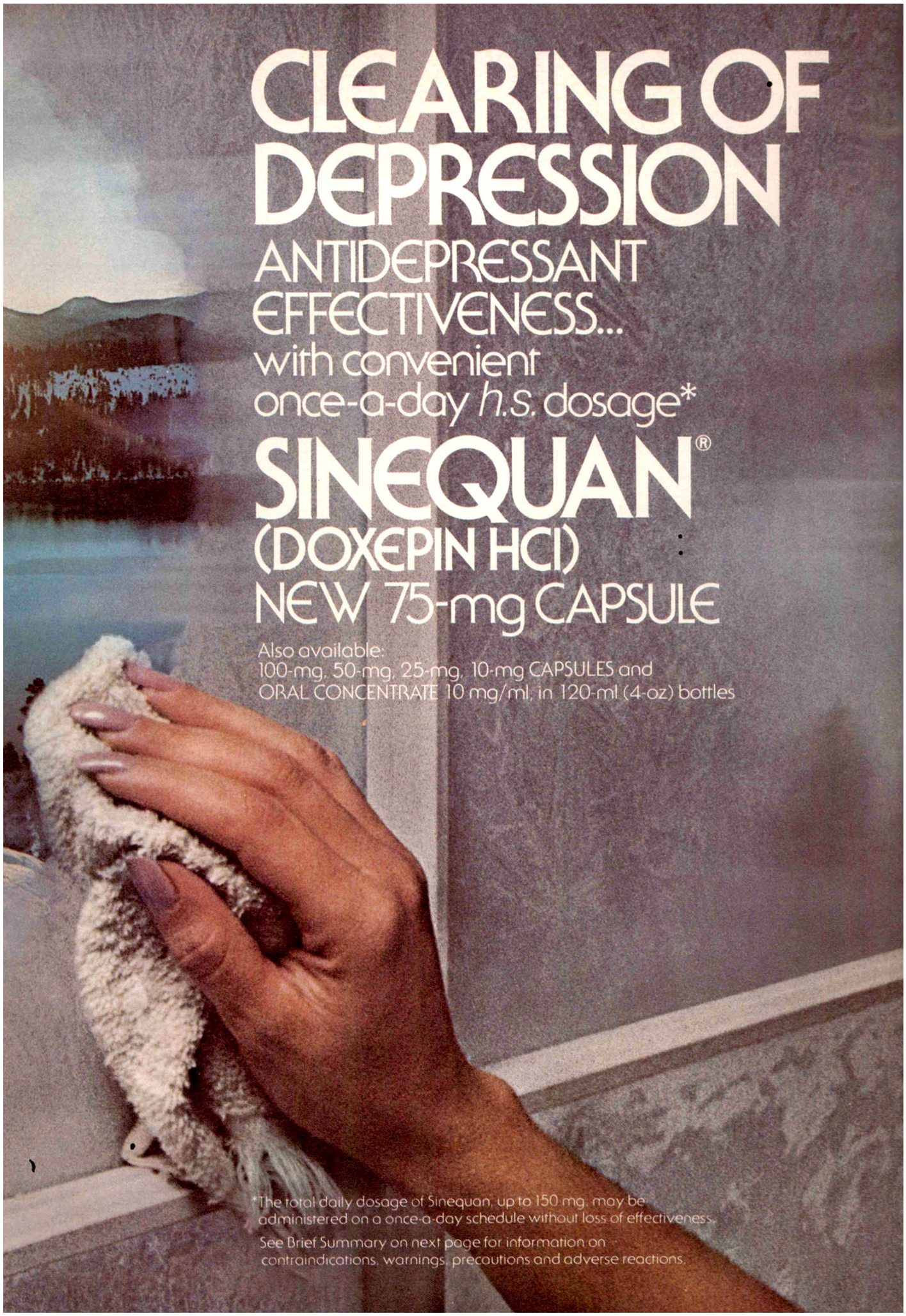


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See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have occasionally been reported. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug. Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting dose of 25 mg t.i.d. is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients an initial dose of 50 mg t.i.d. may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

As an alternative regimen, the total daily dosage, up to 150 mg, may be given on a once-a-day schedule without loss of effectiveness. This dose may be given at bedtime.

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.
A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. Available as capsules containing doxepin HCl equivalent to 10 mg, 25 mg, 50 mg, 75 mg, and 100 mg doxepin in bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). SINEQUAN 25 mg and 50 mg are also available in bottles of 5000. SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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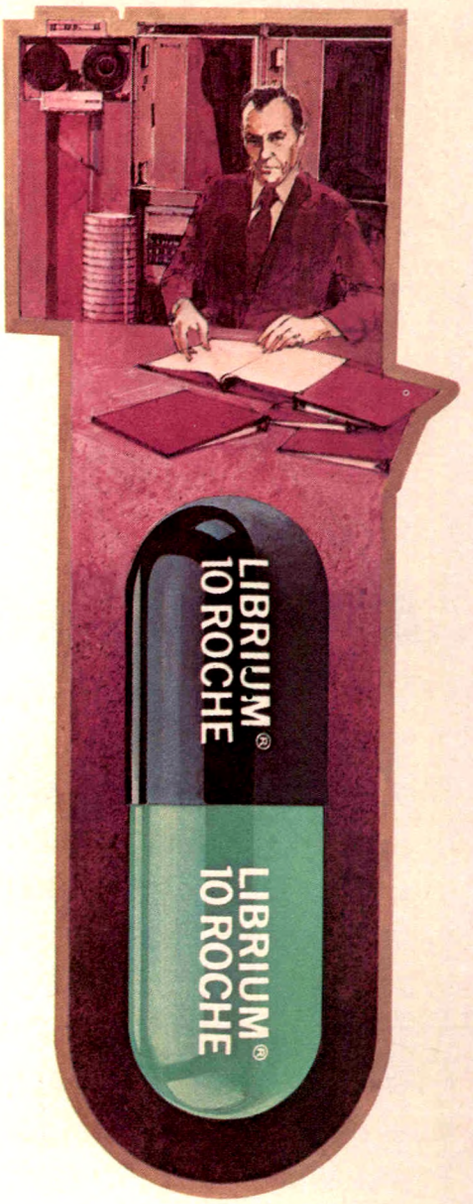
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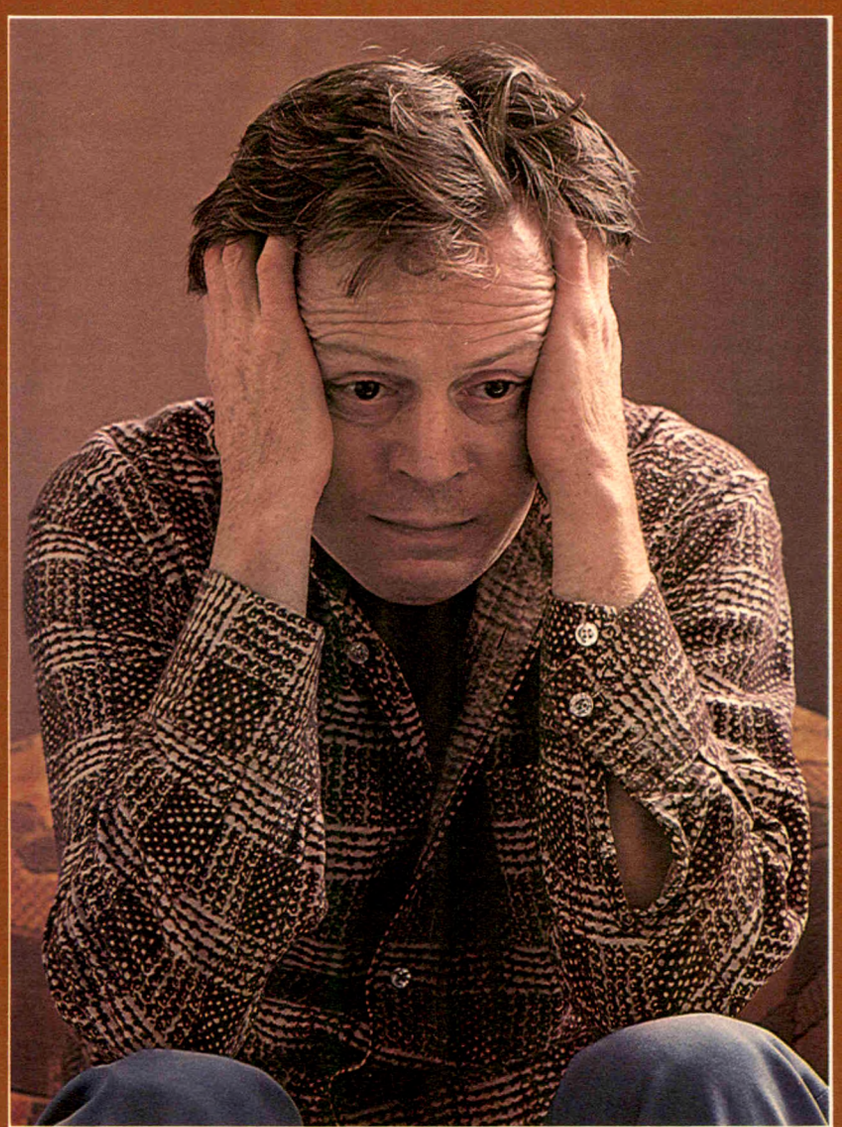
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The advantages of dual-action TRIAVIL in psychiatric practice.

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TRIAVIL simplifies therapy; encourages compliance.

A single tablet encourages patients to take medication properly and reduces the risk of dosage confusion and error. Cost of therapy to the patient is usually less than when the components are prescribed individually. To date, clinical evaluations of TRIAVIL have revealed no undesirable reactions peculiar to the combination.

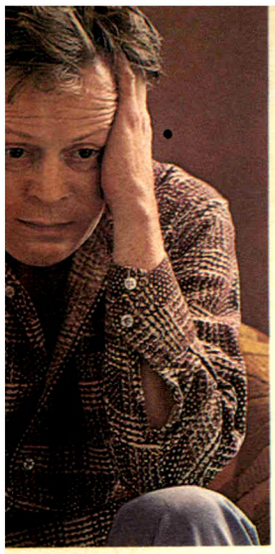
Treatment with TRIAVIL— a balanced view.

TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. It is not recommended in children or during pregnancy. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.

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CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric cris hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism have been reported and can usually be controlled by the concomitant use c effective antiparkinsonian drugs and/or by reduction in dosage, but som times persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy c may occur after drug therapy with phenothiazines and related agents ha been discontinued. The risk appears to be greater in elderly patients on hig dose therapy, especially females. Symptoms are persistent and in som patients appear to be irreversible. The syndrome is characterized by rhyt mical involuntary movements of the tongue, face, mouth, or jaw. Involu tary movements of the extremities sometimes occur. There is no know treatment for tardive dyskinesia; antiparkinsonism agents usually do not all viate the symptoms. It is advised that all antipsychotic agents be discontinued if the above symptoms appear. If treatment is reinstituted, or dosage c the particular drug increased, or another drug substituted, the syndrome ma be masked. Fine vermicular movements of the tongue may be an early sig of the syndrome. The full-blown syndrome may not develop if medication stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythem urticaria, eczema, up to exfoliative dermatitis); other allergic reaction (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions peripheral edema; reversed epinephrine effect; hyperglycemia; endocrin disturbances (lactation, galactorrhea, gynecomastia, disturbances of me strual cycle); altered cerebrospinal fluid proteins; paradoxical excitemen hypertension, hypotension, tachycardia, and ECG abnormalities (quinidin like effect); reactivation of psychotic processes; catatonic-like states; aut nomic reactions, such as dry mouth or salivation, headache, anorex nausea, vomiting, constipation, obstipation, urinary frequency or inco tinence, blurred vision, nasal congestion, and a change in pulse rate; oth adverse reactions reported with various phenothiazine compounds, but n with perphenazine, include grand mal convulsions, cerebral edema, pol phagia, pigmentary retinopathy, photophobia, skin pigmentation, and failu of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (panc topenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinopl lia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after lon term administration of some phenothiazines. Although it has not bee reported in patients receiving TRIAVIL, the possibility that it might occu should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia hav also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for th drug, but which have occurred with other pharmacologically similar tricycli antidepressant drugs and must be considered when amitriptyline is admini tered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitatio myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromu cular:* Confusional states; disturbed concentration; disorientation; delusion hallucinations; excitement; anxiety; restlessness; insomnia; nightmare numbness, tingling, and paresthesias of the extremities; peripheral neur pathy; incoordination; ataxia; tremors; seizures; alteration in EEG pattern extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (an diuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred visio disturbance of accommodation; increased intraocular pressure; constipatio paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Ski rash; urticaria; photosensitization; edema of face and tongue. *Hematologic* Bone marrow depression including agranulocytosis; leukopenia; eosinopl lia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric di tress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; paroti swelling; black tongue. Rarely hepatitis (including altered liver function an jaundice). *Endocrine:* Testicular swelling and gynecomastia in the mal breast enlargement and galactorrhea in the female; increased or decrease libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weaknes fatigue; headache; weight gain or loss; increased perspiration; urinar frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abru cessation after prolonged administration may produce nausea, headache, an malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosag should be admitted to a hospital as soon as possible. Treatment is sympt matic and supportive. However, the intravenous administration of 1–3 m of physostigmine salicylate is reported to reverse the symptoms of tricycl antidepressant poisoning. Because physostigmine is rapidly metabolized, th dosage of physostigmine should be repeated as required particularly if li threatening signs such as arrhythmias, convulsions, and deep coma recur persist after the initial dosage of physostigmine. On this basis, in seve overdosage with perphenazine-amitriptyline combinations, symptomat treatment of central anticholinergic effects with physostigmine salicyla should be considered.

J 6TR06 R3 (DC6613212)

For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486.

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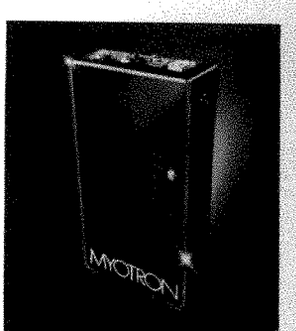
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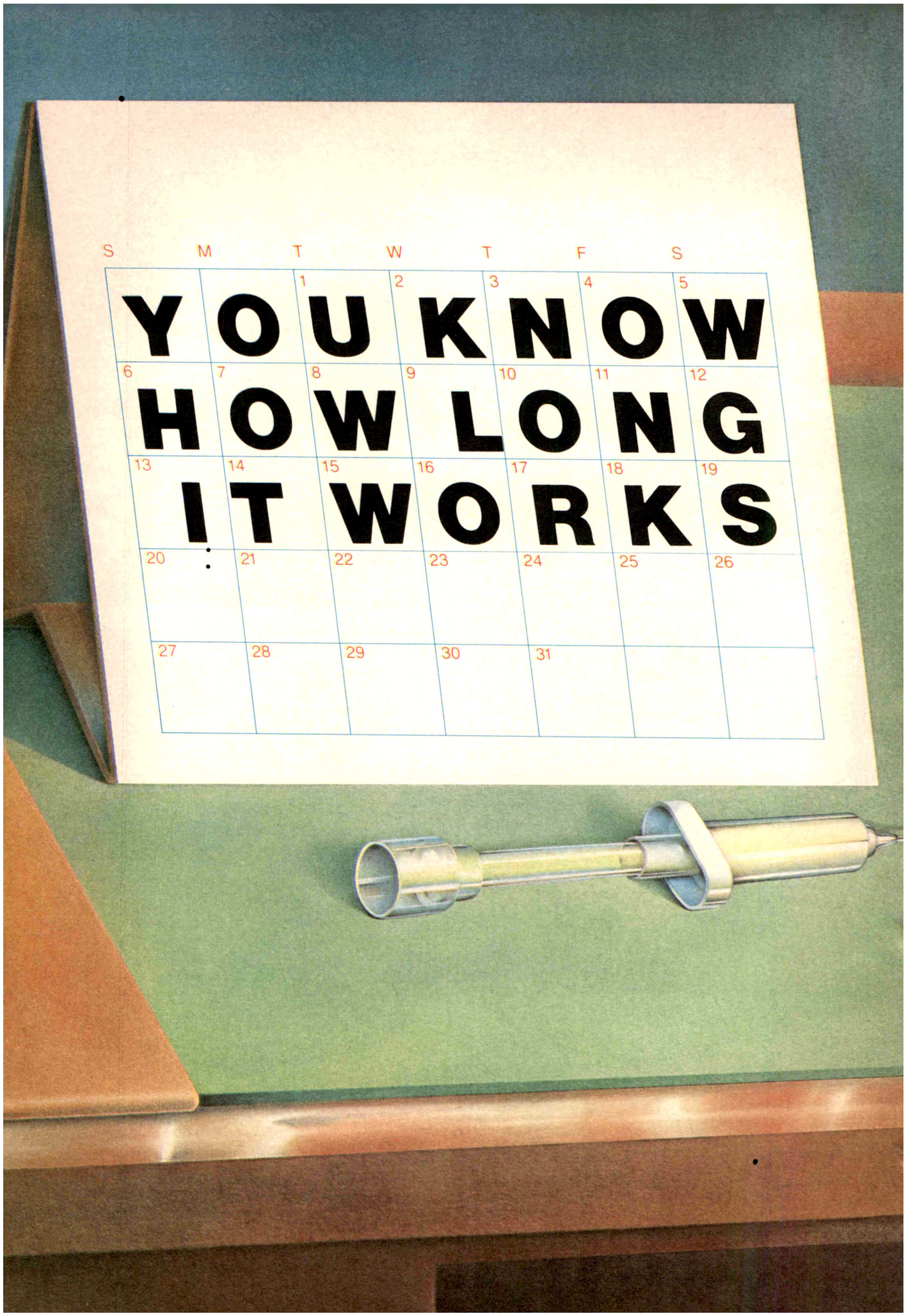
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Drug	Assumed adult dosage*	Form and potency	Cost per unit	Annual cost
Prolixin® Decanoate	12.5-100 mg every 28 days	5 ml vials 25 mg/ml	8.5¢† per mg	$ 13.81 – 110.50
Thorazine®(SKF brand of chlorpromazine)	300-600 mg per day	100 mg tablets bottles of 1000	5.1¢ per tab.**	55.84 – 111.69
Mellaril®(Sandoz brand of thioridazine)	200-800 mg per day	100 mg tablets bottles of 1000	12¢ per tab.**	87.60 – 350.40
Haldol®(McNeil brand of haloperidol)	5-50 mg per day	5 mg tablets bottles of 1000	17.1¢ per tab.**	62.42 – 624.15
Navane®(Roerig brand of thiothixene)	20-60 mg per day	20 mg tablets bottles of 500	24.7¢ per tab.**	90.16 – 270.47

*Dosage level and interval should be determined on an individual basis, in accordance with patient's response to the particular drug as well as the manufacturer's specific recommendations for the use of the product. **Based on prices listed in 1977 Red Book. †Based on Squibb list price of $10.65 per 5 ml vial.

1. Donlon PT, Axelrad AD, Tupin JP and Chien C-p: Comp Psychiat 17:369-376, 1976.
2. Christodoulidis H and Frangos H: Curr Ther Res 18:193-198, 1975.

PROLIXIN DECANOATE®
Fluphenazine Decanoate Injection

See following page for brief summary.

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CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. These include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, and hyperreflexia; most often these are reversible, but they may be persistent. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of antiparkinsonian drugs such as benztropine mesylate or intravenous Caffeine and Sodium Benzoate Injection U.S.P., and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may have to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection U.S.P. is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

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held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

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Clinical studies show that a single daily administration of Norpramin is as effective and well tolerated as the same quantity given as a divided dosage. This means that Norpramin may be given once daily, morning or bedtime, whichever is most appropriate to the patient's therapeutic need, drug response and activity patterns. A.M. dosage of Norpramin can be especially useful when your depressed patient feels more depressed in the early morning. Morning administration may also help reduce the potential for confusion when other agents are prescribed concomitantly at bedtime.

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For those patients who experience a sedative response to Norpramin, a bedtime dose is most appropriate. Evening administration of Norpramin rarely produces morning hangover.

Minimal daytime drowsiness —permits an active schedule
With Norpramin, problems of daytime drowsiness and morning hangover are largely avoided. Minimal daytime drowsiness can be especially important for patients who must perform daytime activities at home or at work.

Early therapeutic response— builds hope and provides encouragement
While full therapeutic effect may require two to three weeks, onset of action has often been observed in two to five days. Although results have been variable, the weight of scientific reports suggests a faster onset of action with desipramine than with either imipramine or amitriptyline.

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25 mg., 50 mg., 75 mg., 100 mg., 150 mg.

for the patient who must function during the day

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Norpramin®
(desipramine hydrochloride tablets NF)

Brief Summary

Indications: Norpramin (desipramine hydrochloride tablets NF) is indicated for the relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Desipramine hydrochloride should not be given within two weeks of treatment with a monoamine oxidase inhibitor. Contraindications include the acute recovery period following myocardial infarction and hypersensitivity to the drug. Cross sensitivity with other dibenzazepines is a possibility.

Warnings: 1. Extreme caution should be used in patients: (a) with cardiovascular disease. (b) with a history of urinary retention or glaucoma. (c) with thyroid disease or those on thyroid medication. (d) with a history of seizure disorder. 2. This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. 3. *Use in Pregnancy:* Safe use during pregnancy and lactation has not been established. 4. *Use in Children:* Norpramin is not recommended for use in children. 5. This drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. Therefore, the patient should be cautioned accordingly. 6. In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions: This drug should be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with drugs of this class. If possible, dispense in child-resistant containers. It should be kept out of reach of children. Reduce dosage, or alter treatment, if serious adverse effects occur. Norpramin therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates and may cause exacerbation of psychosis in schizophrenic patients. Use cautiously with anticholinergic or sympathomimetic drugs. Response to alcoholic beverages may be exaggerated. In the concurrent administration of ECT and antidepressant drugs one should consider the possibility of increased risk relative to benefits. The sedative effects of Norpramin and benzodiazepines (e.g., chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. Discontinue as soon as possible prior to elective surgery because of possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients on desipramine hydrochloride. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is neutropenia.

Adverse Reactions: *Cardiovascular:* hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke. *Psychiatric:* confusional states (especially in the elderly), hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis. *Neurological:* numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus. *Anticholinergic:* dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, hypotonic bladder. *Allergic:* skin rash, petechiae, urticaria, itching, photosensitization, edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs. *Hematologic:* bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue. *Endocrine:* gynecomastia, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels. *Other:* jaundice (simulating obstructive), altered liver function; weight gain or loss, perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia. *Withdrawal symptoms:* Though not indicative of addiction, abrupt cessation after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: *Usual adult dose:* 100 mg. to 200 mg. per day. Dosages above 200 mg. per day are not recommended. *Initial therapy:* Should be administered at a low level and increased according to tolerance and response. It may be administered in divided doses or a single daily dose. *Maintenance:* Lower adequate dose once daily to maintain remission. *Adolescent and geriatric patient dose:* 25 mg. to 100 mg. per day, in single or divided doses. Dosages above 100 mg. are not recommended.

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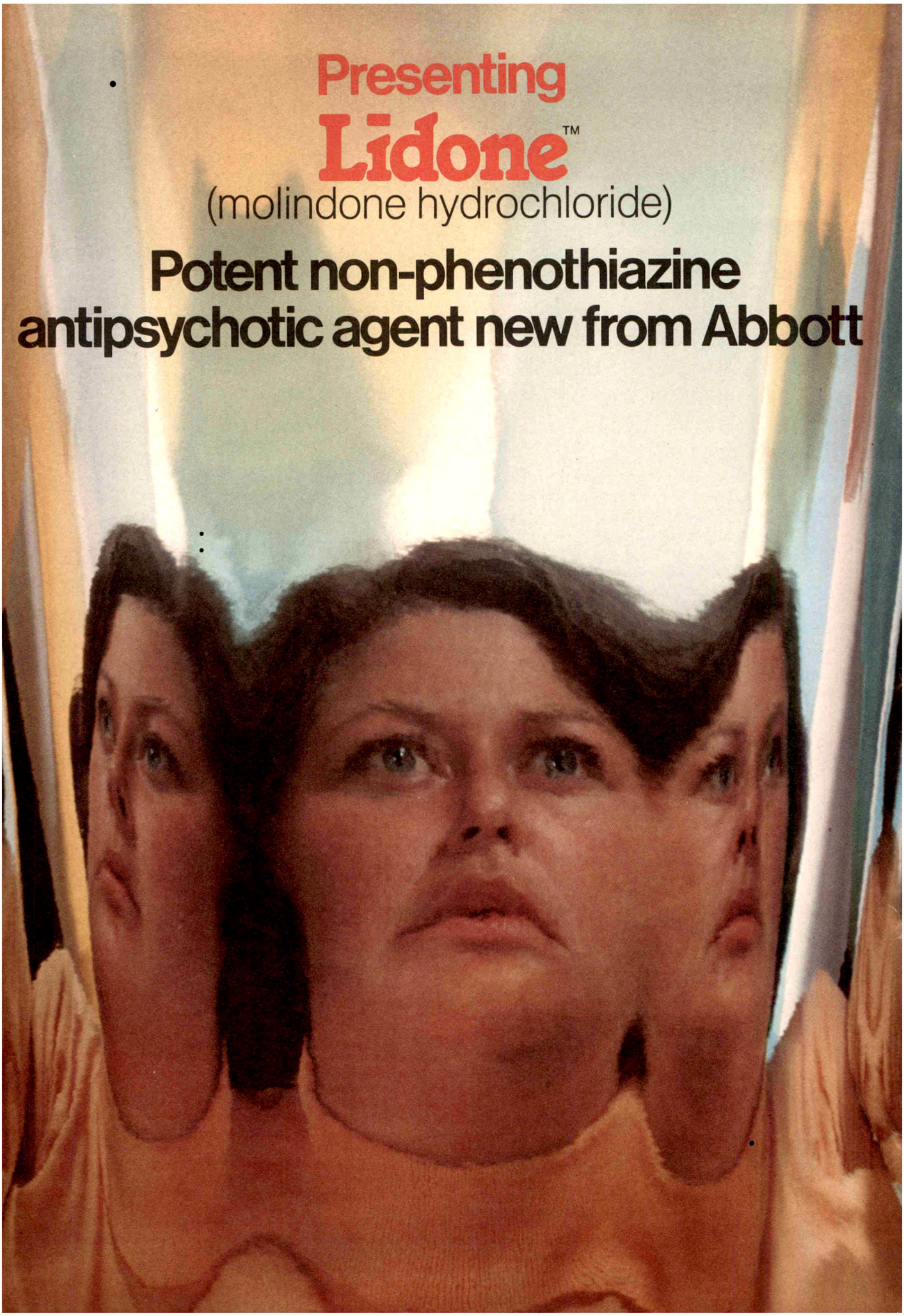
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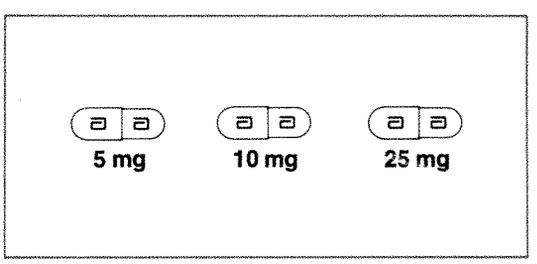
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Brief summary of prescribing information for

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DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose —
20 mg/kg/day — 2 weeks, no adverse effect
40 mg/kg/day — 2 weeks, no adverse effect

Pregnant Mice oral dose —
20 mg/kg/day — 9 days, slight increase resorptions
40 mg/kg/day — 9 days, slight increase resorptions

Pregnant Rabbits oral dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial and maintenance doses of LIDONE (molindone hydrochloride) should be individualized, and the minimal effective dose should be employed. Elderly and debilitated patients should be started on lower dosage.

DOSAGE SCHEDULE, BASED ON SEVERITY OF SYMPTOMATOLOGY

1. Mild — 5 mg three or four times a day; an increase to 15 mg three or four times a day may be required.
2. Moderate — 10 mg three or four times a day; an increase to 25 mg three or four times a day may be required.
3. Severe — daily dosage as high as 225 mg may be required.

DRUG INTERACTIONS — Potentiation of drugs administered concurrently with LIDONE (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when LIDONE is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE — Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of LIDONE (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of LIDONE (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by LIDONE in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized LIDONE from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of LIDONE is excreted unmetabolized in the urine.) However, poor response of the patient may justify use of these procedures.

While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized LIDONE in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED — LIDONE (molindone hydrochloride) capsules are supplied in bottles of 100 in the following dosage strengths and colors:

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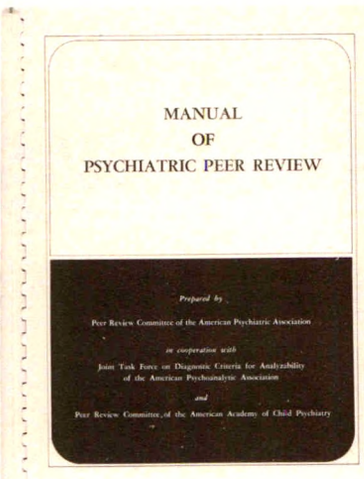
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in clinically significant depression...

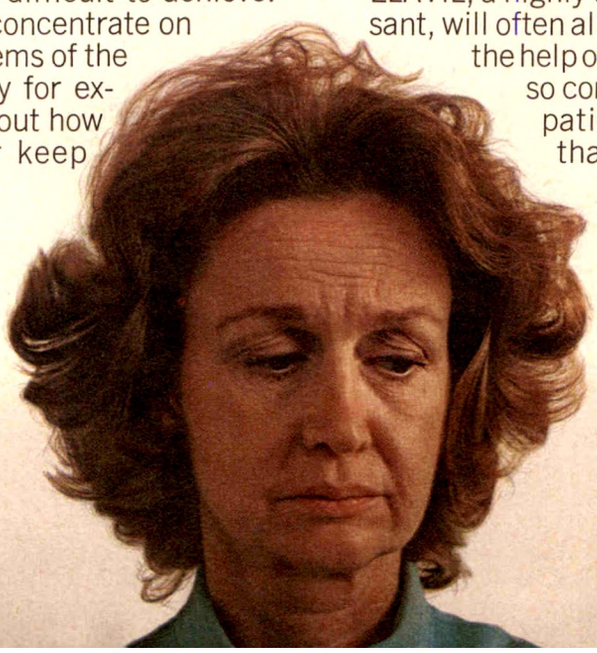
penetrating the symptom barrier to productive psychotherapy

symptoms often interfere with therapy

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ELAVIL, a highly effective tricyclic antidepressant, will often alleviate these symptoms. With the help of ELAVIL, sleep disturbance—so common in clinically depressed patients and often the symptom that distresses them most—as



well as other "barrier symptoms" may be lessened to the point where they no longer come between you and the patient.

psychotherapy takes direction

As these preoccupying complaints are lifted, psychotherapy sessions can become more productive. Patients are enabled to respond more fruitfully during therapeutic hours and to resume or pursue more efficiently the necessary activities in their personal lives.

meeting therapeutic goals

And as the antidepressant activity of ELAVIL (Amitriptyline HCl, MSD) takes hold, it helps relieve the depressive condition itself. Sometimes only a minimum of actual psychotherapy is required. The patient may experience a more rapid recovery—while you conserve valuable time.

Prescribe ELAVIL then, to help lighten the patient's burden in depression—and yours in its management.



ELAVIL should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks; or in children under 12. Patients with cardiovascular disorders should be watched closely. Safe use during pregnancy and lactation has not been established. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Hospitalize as soon as possible any patient suspected of having taken an overdose.

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, 100 mg, and 150 mg
INJECTION: 10 mg per ml

ELAVIL®

(AMITRIPTYLINE HCl | MSD)

an antidepressant with an anxiety-reducing sedative component to its action

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addendum

To further simplify once-daily therapy for many depressed patients

Tablets

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ONE TABLET, ONCE DAILY

—an appropriate way to prescribe for many depressed adult outpatients. Because of its simplicity, this regimen helps improve patient compliance. Of course, ELAVIL may also be prescribed in divided daily doses.

ONCE-DAILY DOSAGE SCHEDULE FOR ADULT OUTPATIENTS:

A single 75-mg tablet
Usual starting dosage



A single 100-mg tablet
Maximum starting dosage

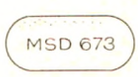


A single 50-mg tablet
Minimum starting dosage



Dosage may be increased by 25 or 50 mg as necessary until a total of 150 mg per day is reached.

A single 150-mg tablet
Maximum daily dosage



ELAVIL MAY ALSO BE PRESCRIBED IN DIVIDED DAILY DOSES

The 25-mg tablet



This strength may prove useful when therapy is initiated with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime dose.

The 10-mg tablet



This strength may prove useful for patients who require lower doses, e.g., adolescent and elderly patients. For these patients who can not tolerate higher doses, 10 mg three times a day with 20 mg at bedtime may be satisfactory.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.
When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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(AMITRIPTYLINE HCl, MSD)

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OFTEN ACCOMPANIES DEPRESSION

In 14 double-blind studies, of 339 patients with depressive neurosis who received Mellaril (thioridazine) over half had measurable relief within one week. Relief of the anxiety component was seen in 54.6% (185 of 339 patients) within the first week; relief of the depressed-mood component was seen in 53.4% (181 of 339 patients); relief of insomnia secondary to depression was seen within the first week in 50.2%, or 132 of the 263 patients with this symptom; and overall improvement was seen in 51.3% (174 of 339 patients) in the first week and in 86.4% (293 of 339 patients) by the end of the fourth week.

The most common side effects are drowsiness and dry mouth. Mellaril (thioridazine) is not habituating and usually does not cause euphoria or undue sedation. (The physician should, however, caution patients against participating in activities which require complete mental alertness, e.g., driving.)

in short-term therapy of moderate to marked depression
with variable degrees of anxiety in patients with depressive neurosis

MELLARIL® (THIORIDAZINE)

TABLETS: 10 mg, 15 mg, 25 mg, and 50 mg thioridazine HCl, USP

RESULTS OFTEN SEEN IN A WEEK

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For Brief Summary, please see following page.

SANDOZ
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in short-term therapy of moderate to marked depression with variable degrees of anxiety in patients with depressive neurosis

MELLARIL®
(THIORIDAZINE)

RESULTS OFTEN SEEN IN A WEEK

Before prescribing or administering, see Sandoz literature for full product information. The following is a brief summary.

Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient. In adults with depressive neurosis the usual starting dosage is 25 mg t.i.d. and the dosage ranges from 10 mg b.i.d. to q.i.d. in milder cases to 50 mg t.i.d. or q.i.d. for more severely disturbed patients; the total daily dose ranges from 20 mg to a maximum of 200 mg.

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THE AMERICAN JOURNAL OF PSYCHIATRY

Accountability, Public Policy, and Psychiatry

BY ANNE R. SOMERS

The author identifies the direction of changes in health policy in the United States that need to be taken into account by the health professions in their dealings with government. She discusses three areas in which these changes will call for adjustment on the part of the medical profession and major health care institutions and defines the special challenge to psychiatry implicit in the changes. She concludes that the public's need for attention to the psychosocial aspects of health and the renewed interest in prevention present psychiatry with an opportunity to provide leadership in health care delivery.

THE CONCEPT of "public accountability" involves two essential ingredients: 1) genuine participation by professions and institutions in the private sector in the formulation of public goals and 2) a commitment to work toward such goals in a fiduciary or stewardship role, including an ongoing "accounting" to the public for this stewardship. Stated a little differently, the concept presupposes not only the continued existence of the private sector but also a large degree of professional and institutional independence. The opposite side of the coin is that the professions and the institutions may expect to retain their private status only as long as they act as if they were public.

This may seem a meaningless distinction but, in fact, it is important. If we, in America, can achieve a combination of public goal-setting and private-sector implementation, avoiding the evils of overcentralization and overbureaucratization implicit in too much direct government control, we will have made a major contribution to the development of democratic self-government.

Public accountability might be likened to the "categorical imperative" propounded some 200 years ago by Immanuel Kant. At a time when philosophers were hotly debating the existence of God and the relevance of competing ethical principles, Kant concluded that, while His existence could not be proved, it behooved the individual to conduct himself "als ob" or "as if" the universe were ordered in accordance with universally binding moral law.

The concept is particularly relevant and useful in the health field. Considerations of professional responsibility and public trust are deeply embedded in the health professions and in hospital history. They are implicit in the Hippocratic oath and explicit in state licensing laws. It is obvious that the government cannot take over and run the nation's health care system or even a single hospital without the cooperation of the majority of professionals involved. It is equally obvious, in this day of multibillion-dollar health care costs, that private professions and institutions cannot function effectively without government participation and without implied acceptance of publicly defined goals.

I congratulate Dr. Gibson and the other officers of the American Psychiatric Association for their timely choice of theme for this meeting, "Professional Responsibility and Public Trust" (1). Public accountability is an essential aspect of this theme. After reading Dr. Marmor's 1976 Presidential Address (2), it is impossible not to conclude that the leadership of APA understood and accepted the implications of public accountability long before it became such a popular phrase. The challenge remains, however, to translate these lofty presidential ideals into the everyday thinking and practice of the 22,000 physicians who make up APA's membership. This, of course, is the real test of leadership, as well as of the viability of the concept.

Of the two components in my definition of public accountability, the second—procedures for accounting to the public with respect to professional performance or stewardship—has evoked the most discussion.

Revised version of the William C. Menninger Memorial Convocation Lecture, presented at the 130th annual meeting of the American Psychiatric Association, Toronto, Ont., Canada, May 2–6, 1977.

Ms. Somers is Professor, Department of Community Medicine and Department of Family Medicine, College of Medicine and Dentistry of New Jersey-Rutgers Medical School, Piscataway, N.J. 08854.

Professional Standards Review Organizations (PSROs) and other forms of quality control, utilization review, certification and recertification, and continuing education—the crucial implementing procedures—deserve all the attention they are receiving, and more. However, I intend to focus on the other component—public-private cooperation in the formulation of broad social goals, goals to which the private organizations will consider themselves committed and for whose implementation they will accept responsibility. I have chosen this focus because I believe that a great deal of the present antagonism between public and private sectors derives from failure to agree on mutually acceptable goals or even to appreciate the necessity of such goals.

Part of the problem is the inconsistency—sometimes to the point of direct contradiction—of the policies that the bureaucrats must implement. This inconsistency results from two major considerations: 1) the structure of our pluralistic form of government, which strongly favors special group interests rather than monolithic or holistic policies, and 2) the difficulty of eliminating old agencies even when their functions have become obsolete. Old bureaus never seem to die; sometimes they don't even fade away!

These are serious defects in our form of government and were probably among the conditions that led Winston Churchill to say, "Democracy is the worst possible form of government—except all others!" However, I doubt that any of us is prepared to exchange our often ambiguous pluralism for the clear-cut mandates of totalitarian government. We have learned to live with considerable ambiguity, and I suspect we will continue to do so. On the other hand, we could reduce the confusion considerably if we could learn to think about public policy as evolving in a dynamic, constantly changing context. Policies must and will change in response to changing scientific, technologic, socioeconomic, and demographic conditions. The more rapid the underlying shifts, particularly the scientific, which trigger most of the others, the more rapid changes in public policy have to be.

In this paper I shall try to do three things: 1) identify the direction of change in U.S. health policy today—providing a kind of forecast of the prevailing socioeconomic and political winds that the health professions would be advised to take into account in their dealings with government; 2) discuss briefly three specific areas in which these prevailing winds will probably call for considerable adjustment on the part of the medical profession and major health care institutions; and 3) provide some conclusions regarding the special challenge to psychiatry implicit in these trends.

THE DIRECTION OF CHANGE

There is no question but that the United States is in the midst of a new sociopolitical revolution. No one knows exactly how to define it, much less where it will take us. Some speak nostalgically of "the end of the American dream," meaning, usually, an end to economic growth, an ever-rising material standard of living, and maximum personal freedom—to do one's own "thing." Others, emphasizing the moral and political bankruptcy associated with Viet Nam and Watergate, see our present difficulties as an opportunity for a spiritual renaissance, an emergence from a long period of overemphasis on materialism.

President Carter's energy policy typifies the new mood. Although there is no abrupt break with the past, the differences in emphasis are considerable. Gone is the sense of limitless resources, replaced by a sober appraisal of limited resources. Gone is a one-dimensional approach to causation and cure, replaced by a complex multifactorial analysis and equally complex prescriptions for management. Gone is the assumption that the situation can be managed either by private enterprise alone or by government alone, replaced by a call for a difficult but essential working relationship. The two knee-jerk reactions that one might have heard in the past—"Nationalize the oil fields!" or "Get the government out of business altogether!"—are both conspicuously missing. Gone also is the idea that the individual consumer has no responsibility, replaced by a call for considerable responsibility and even some sacrifice.

It remains to be seen whether the President will be able to sell this policy to Congress and the American people, many of whom still prefer to attribute our problems to foreign plots, demons, and assorted personal villains, but at least the first reaction suggests a more mature, sensible public opinion than some pessimists had anticipated.

I mention energy because there are some important analogies to the situation in health. For a quarter-century after World War II the health field was characterized by the euphoric view that resources were limitless; all we had to do was design financing programs to remove barriers to access and use public money to subsidize the expansion of facilities and personnel. We now know that a national health policy keyed almost exclusively to greater access will result in vastly increased expenditures but not necessarily in proportionately better health. For a quarter-century health policy has been dominated by the one-dimensional view of illness and disability as resulting primarily from inadequate medical care, especially somatic care. We now know that good medical care is only one of the many factors affecting health; others include the individual's biological makeup and environmental and behavioral components. For a quarter-century people have been led to believe that some external body—the government, the hospital, the HMO, the neighborhood health center, the AMA—was responsible for their personal health, or the lack of it. We now know that, although responsibility has to be shared by those who can give or withhold information, care, and assistance when needed, the ultimate responsibility rests with the individual.

I do not know whether the new Administration will succeed or even try to translate our better understanding of health care into a new health policy more attuned to the realities of the late 1970s. A widespread groping for new directions has been evident for some time (3–6), even in the U.S. Department of Health, Education, and Welfare (7). Some of President Carter's campaign speeches indicated movement in this direction. Certainly the mental health field has reason to welcome establishment of the new President's Commission on Mental Health, with its broad mandate and composition.

The most carefully articulated official statement to this effect has come from the Canadian Government. The Lalonde report, *A New Perspective on the Health of Canadians* (8), has provided students of health care in the United States, as well as in Canada, with a challenging formulation of national health policy based on a new concept of the "Health Field."

Herman Somers and I have published a proposal (9, 10) that we hope will contribute to the formulation of a new American policy. We take our cue from the European epidemiologist, Dr. Rene Sand, who said, "Health cannot simply be given to the people; it demands their participation." Then we go on to say,

> Any new design that will prove effective must be based on two broad assumptions:
>
> 1. Responsibility for personal health rests primarily with the individual; not with government, not with physicians or hospitals, not with any third-party financing program. Meaningful national health policies must be directed to increasing, rather than eroding, the individual's sense of responsibility for his own health and his ability to understand and cope with health problems.
>
> 2. If the individual's responsibility is to be effectively discharged, it must be supported by social policies designed to provide him with essential environmental protection, health information, and access to health care when needed.
>
> In short, a policy of "individual responsibility" and a policy of "social responsibility" are complementary, not antithetical. Neither can be fully effective without the other. The term that seems best to suggest an approach that encompasses both assumptions is "health promotion." The overriding goal of such a national health policy may be stated as: The creation and maintenance of conditions under which individual Americans are most able and most likely to optimize their own health potential.

We then go on to classify the major conditions affecting individual and national health under several broad headings; i.e., genetic endowment, the environment, behavioral factors, access to good health services, and the scientific state of the art in all categories.

This does not reflect official U.S. health policy at the present time. Even the Lalonde report, which, unlike ours, is official, is not universally accepted, much less implemented, throughout Canada. However, the leadership of the health professions may wish to consider these new formulations and their implications for emerging public and professional policies. These implications may become clearer if we consider three specific issues: comprehensive care, cost controls, and prevention.

THREE AREAS OF CHANGE

Comprehensive Care

In theory, there is widespread agreement that Americans should have access to "comprehensive care," i.e., preventive, diagnostic, therapeutic, rehabilitative, and long-term services, as needed and with equal attention to physical and mental illness. The spectrum is sometimes defined more simply as primary, secondary, and tertiary care. Long-term care is sometimes subsumed under primary care, sometimes added as a fourth category.

The practice, however, is quite different from the theory. The realities of health care financing—both public and private—which reflect priorities of the past, prior to the emergence of chronic illness as the major cause of morbidity, strongly bias availability in the direction of the more specialized and expensive modalities associated with tertiary care. For example, although about 80% of the population under the age of 65 had some hospital and surgical coverage under private health insurance in 1974, only 62% had any coverage for physicians' office visits, and only 35% for nursing home care (11). Only 11% of the "working poor" had any coverage for out-of-hospital services (12, p. 21). A similar pattern can be seen in Medicare expenditures. In fiscal 1975, for example, this pattern-setting program paid for 72% of the hospital expenses of the elderly, 54% of the physicians' fees (mostly for in-hospital services), 3% of the costs for skilled nursing care, and 4% of all other costs (13).

Such financing and other government subsidies encourage the provider tendencies subsumed under the familiar maxims, "the technological imperative" and "the edifice complex." Today, most physicians are not in primary care. They are specialists or superspecialists, generally attuned to tertiary care. Most, sometimes all, of their clinical training takes place in hospitals. Prestige in the profession leans heavily to the accomplished diagnostician, who is greatly dependent on the imposing armamentarium of the tertiary institution. The "battery of tests" syndrome has recently found a new rationalization in "defensive medicine."

Hospitals are driven by similar stimuli. Each wants to be bigger, to offer a broader range of services, to be the "best." Prestige comes from attracting prominent specialists to the staff, and this requires more and better technological equipment and plenty of beds, all of which leads to expanded capitalization, higher operating costs, and pressure to "fill the beds." If money to pay for all this appears virtually assured—over 91% of all hospital costs were paid by third parties in fiscal 1977 (14, p. 10)—there is not much to restrain these "natural" tendencies.

Such developments led Dr. Alexander Leaf, physician-in-chief at Massachusetts General Hospital, to observe recently, "We are doing too much for too few, at too great a cost, and with too little benefit." In light of their training, it is often difficult to persuade physicians that postponement of death is not the sole purpose of health care. Matters such as pain, patient preference, medical expense, and social costs are competing considerations and, in some terminal contexts, may outweigh the prolongation of life. At the extreme, we have the appalling extent to which people in hopelessly comatose states are kept indefinitely alive by a panoply of mechanical and chemical interventions, dramatized in the celebrated case of Karen Quinlan (15).

While Medicare, Medicaid, and private health insurance pump tens of billions of dollars into the tertiary institutions, the public demand for adequate primary care and long-term care for the elderly and the chronically ill, including the mentally ill and retarded, remains unanswered. Some progress is being made in these areas. Congress (Public Law 93-641), DHEW (7, pp. 50–51), and the Robert Wood Johnson Foundation (16) have all declared that primary care, especially for underserved areas, is a top national health priority. Medical students are being bribed and bludgeoned to go into primary care for at least a year or two. Medical schools are exhorted to include more primary care in their curricula. The Institute of Medicine has had a Task Force on Primary Care, trying for 18 months to develop a workable definition. The American Board of Medical Specialties also has such a committee.

As long as the flow of national resources is so heavily in the direction of tertiary care, it is tempting to dismiss all such activities as meaningless or, at best, peripheral. Peripheral they certainly are at the moment, but it is my conviction, based on underlying demographic and socioeconomic trends, that they will become steadily more important. Just as chronic illness, including mental illness, has moved to center stage in the nation's morbidity and mortality picture, so the health care modalities that are directed at the prevention and management of chronic illness will, almost inevitably, become more important.

For the moment this constitutes a prime example of an area in which the health professions and the industry are receiving conflicting signals from the public. It is also an area in which the professions can help the government adjust its policies to the realities of current morbidity and mortality patterns and trends. Only after such adjustments are made and we come to some agreement on common goals can meaningful public accountability take place.

Cost Controls

Money is obviously a key issue in the provision of health services. Financial considerations influence both the direction of public policy and the interactions between public and private sectors. APA has put the proposition succintly: "The dollar flow is not merely a disbursement mechanism. It is a management tool which can be used for good or ill (17).

Prodigious inflation of health care costs has persisted for three decades, bringing health expenditures to well over $150 billion this year and moving rapidly toward 10% of the GNP (14, p. 3). The amount is so staggering that it is difficult to comprehend. Two comparisons may be helpful: 1) We are spending more for health care in the United States than the entire GNP of all but half a dozen other nations (18, p. 845). In 1973, the last year for which I have comparable data, we spent $95 billion for health care compared with $13 billion for the entire criminal justice system, including police, the judiciary, and prisons (14, p. 4; 18, p. 160). With homicide now our fastest rising cause of death (19, p. 26), the question can legitimately be raised whether the health of the American people might not now be better served by putting additional resources into law enforcement rather than health care.

Far from any sign of abatement, however, the rate of health care inflation has accelerated during the past few years. The rate during the past year is three times that of the general cost of living (20). Concern has understandably deepened. The causes are multifaceted and deeply rooted in both supply and demand factors. The phenomenon of rising costs prevails, in greater or lesser degree, in most advanced countries, regardless of varying methods of financing. This fact underscores the difficulty of establishing effective controls, particularly in a democracy.

There is a hen-and-egg relationship between the cost rise and financing mechanisms. The rise in costs led to the development of public and private health insurance, governmental health services, and other methods of minimizing the financial barriers to needed services. However, the very existence of such programs contributed to further cost rises. More programs were then added or existing programs expanded to meet the new hardships.

Most advanced countries have carried this process to the point of universal or near-universal coverage. Ironically, the United States, the richest of all countries, has stopped short of universality—some 25–35 million people short. That is the approximate number estimated to lack any reliable access to health care, either through private insurance, Medicare, or Medicaid, or whose coverage is so thin that it is useless except in cases of serious hospitalized illness (9).

For several years it appeared that this gap in coverage would be closed through some form of national health insurance that would assure universal access to at least the basic components of comprehensive care. However, the level of inflation, especially in hospital costs, that has prevailed since 1974 has dashed this expectation. As Dr. Gibson said in his Presidential Address, "Alarmed by runaway costs, legislators are hedging on the original declaration that health care is a right" (1). This appears to be a bipartisan judgment on

the part of both Congress and two successive Administrations.

It is hard to quarrel with the conclusion in terms of competing demands on national resources from other essential sectors of the economy that also affect health. Nevertheless, it has to be a matter of acute embarrassment to both public and private leaders of the health care "Establishment" that we in the United States have failed to hold the rise in costs within dimensions that would make universal coverage possible. It is, of course, the poor, whose clinics and neighborhood health centers are being closed; the aged, who are forced unnecessarily into inadequate nursing homes for lack of home care services; and the mentally ill, who still have to be institutionalized for lack of adequate community mental health programs, who are paying the price for the inordinate rise in costs and our inability to control it.

Here again, however, the medical profession is getting mixed signals from government. On the one hand, there are the daily scoldings and exhortations to control costs, the periodic release of names of physicians alleged to have profiteered from government payments for health care, and proposals to establish "caps" or ceilings on the annual increases. On the other hand, there is the failure to provide adequate support for primary and long-term care and the Medicare blank-check reimbursement policy for inpatient somatic illness.

This is not the place to undertake discussion of all that would be involved in establishing an effective program of cost controls; nor is it germane to my main thesis. Such a program would have to be as multifaceted as the causes of the inflation. Whatever the specifics, however, there are two essential components: 1) It will have to put an end to the current bias in favor of inpatient, technologically dominated care. 2) It will have to have input from the leadership of the professions and the industry, input that is not limited to protecting special group interests but is attuned to broad national needs and concerns, is prepared to think in terms of the public interest, and is prepared to live with the consequences without being constantly policed. This would be public accountability of the highest order.

Prevention

Despite the obvious "commonsense" proposition that preventing illness is good not only for health but also for the pocketbook, we have relegated prevention to a very low rank on the national health priority list. Estimates of the proportion of national health care expenditures going for prevention vary from 2% to 5%, depending in part on definitions (21, 22). For health education the proportion is probably less than half of 1%.

There are many reasons for this neglect, some of which have already been suggested: the skewing of resources toward tertiary care and the prevailing pattern of health care financing. The current financial crunch now gives rise to another major irony. Advocates of prevention have maintained for years that its neglect would lead to inordinate rises in the cost of curative medicine. They were correct. Now that that has happened, they are told that there is no money to help finance new preventive programs. This is true at all levels, federal, state, local, and institutional. Physicians who want to practice preventive medicine, who want to hold down hospital utilization and expensive therapies, are told by the U.S. Government: "Sorry, Doctor, but Medicare doesn't cover periodic health exams, or refractions, or preventive dental care, or preventive psychiatric counseling. . . . However, when your patient is hospitalized, that's a different story. We'll pay for whatever you want to do."

It is easy to make fun of the third parties. They are sitting ducks for such criticism. But what of the profession itself? Are most physicians really concerned with "health" or just with their special disease or condition? Perhaps just with the biomedical facets of that condition? I have already made clear that the individual bears the primary responsibility for his own health. But surely there is also a large role for the physician. Although he cannot be a specialist in all of the areas that affect individual health, he can be expected to have more than a layman's concern and knowledge as to how these areas affect his own patients. Applying the "health field" concept, this would call for some knowledge of biological, environmental, and behavioral factors in the patient's history, as well as a plan for incorporating preventive services—both primary and secondary—into the patient's regimen.

The "Lifetime Health Monitoring Program" (LHMP), developed by Dr. Lester Breslow and myself (23), represents an effort to move toward a practical, affordable program that could be incorporated in most primary care practices and in public and private health insurance coverage. Using clinical and epidemiological criteria, LHMP attempts to define broad health goals and professional preventive services appropriate to 10 different age groups covering the life cycle. For two of these age groups, infancy and older middle age (40–59), we suggest specific screening and counseling procedures intended to help the patient achieve the defined goals.

The response to this article from both the medical profession and a number of health insurers has been so good that I am led to believe this may indeed be an idea whose time has come. Several groups are considering demonstration and evaluation projects. This is a form of public accountability that goes beyond the current official definition of public policy and takes into account underlying needs and developing trends. Thus it helps to mold future policy.

THE CHALLENGE TO PSYCHIATRY

If the concept of public accountability and the general redirection of U.S. health policy suggested in this

paper are correct, it would seem—at least to an outsider like myself—that psychiatry should be in an exceptionally strong leadership position vis-à-vis the health professions as a whole. This should be true whether one thinks in terms of individual patient care or national policy direction.

There appears to be a clear affinity between the comprehensive, holistic, biopsychosocial approach to patient care, implicit in recent articles by Drs. Marmor (2), Eisenberg (24), and Engel (25), and the comprehensive, balanced approach to national health policy recommended by Lalonde, Herman Somers, myself, and others. A balanced system of comprehensive care would place equal emphasis on primary, long-term, and tertiary components; ambulatory as well as inpatient care; and physical and mental illness. Under effective cost restraints that do not favor one modality over others, specialties that are less dependent on expensive inpatient facilities and equipment would obviously be in a stronger position than they are now. Specialties that emphasize individual and community responsibilities rather than expensive technological interventions would also gain.

I believe this is consistent with the position of psychiatry, which, for two decades, has emphasized the importance of "getting out of the hospital and into the community." The national network of community mental health centers, whatever their current shortcomings, stands as a monument to this leadership and represents an essential aspect of primary care.

I am not suggesting any repudiation of scientific medicine, that indispensable storehouse of knowledge which has been so painstakingly and often painfully accumulated over the centuries. I am suggesting that the public need for more attention to the psychosocial aspects of health and the renewed interest in prevention present psychiatry with a golden, perhaps unprecedented, opportunity in such crucially important areas as drug and alcohol addiction, obesity, television and the "culture of violence," human sexuality, the disappearance of the extended—and now possibly even the nuclear—family, the loneliness of the aged and the dying, the grief of survivors, the increase in illegitimate births, and child abuse. Many of psychiatry's traditional and special concerns are now among the central health concerns of the American people.

Of course, concern is not enough. Again to cite Dr. Gibson, the fact that many existing procedures and therapies are accepted and reimbursed by society, without requirement as to cost-effectiveness, is no excuse for psychiatry to try to play the same game. The ground rules are becoming tougher all around, and psychiatry, like somatic medicine, has to face the demand for refinement of standards and criteria, more valid outcome studies, better peer review, and other proofs of efficacy. The emphasis being placed on peer review in recent APA publications and policy statements and APA's initiatives in this respect, if actually implemented, seem to me good examples of increasing accountability. The new multidisciplinary peer review contracts, currently being negotiated with the Department of Defense for the CHAMPUS program, could be an important precedent for the profession as a whole.

The other side of the coin is fair and adequate funding. Experience in Canada and under the U.S. Federal Employees Health Benefits Program indicates that equal treatment for mental and physical illness need not result in abuse of psychiatric benefits or exhorbitant costs. After some 15 years' experience, the proportion of total benefit payments (high and low options combined) spent on mental illness under the largest plan participating in the federal employees' program appears to have leveled off at a little over 7% (26, p. 12). In Canada, where mental disorders are covered fully on the same basis as other conditions, the per capita payments in the early 1970s varied from $0.43 to $3.15, depending on the province, and from 1.4% to 5.4% of total payments (26, pp. 13–14).

Despite such evidence, mental illness and psychiatry have been notoriously shortchanged in both public and private health insurance programs in the United States. According to APA figures, about 20% of all persons with hospital coverage and 15% of those with physician outpatient coverage still lack benefits for mental illness (26, p. 11). Moreover, when benefits are provided for mental illness they are generally more restricted than those for physical illness. This was true, for example, of 70% of the Blue Cross plans. Probably the most egregious example of discrimination is in Medicare: Outpatient psychiatric benefits are limited to an annual maximum of $250 or 50% of "reasonable charges," whichever is less, after a $50 deductible. This, of course, reflects Medicare's bias not only against mental illness but against preventive services in general.

Over the years general tax support has helped to compensate for the inadequacies of health insurance. This support will almost certainly continue to be necessary for certain types of mental illness. However, the unpredictability of such funds, as presently administered, offers little incentive to consistent professional accountability, including effective resource management. Moreover, it is essential for quality controls that psychiatry be considered part of the mainstream of health care, not separated by some financial iron curtain.

Obviously, there are problems in the coverage of mental illness, e.g., the definition, the specificity of diagnoses, and evaluation of results. As in all of medicine, there are special problems relating to primary care, prevention, and long-term care. However, there are even greater problems in trying to ignore them. The $150 billion health care bill is not the result of abuse of prevention and long-term care; it is primarily the result of abuse of secondary and tertiary care. It is at least partially the result of neglect of prevention and long-term care.

I shall not attempt to lay out a program of reform of mental health financing. APA's "Principles of Psychi-

atric Coverage under National Health Insurance" appear a sensible beginning (26, pp. 4–5). However, since there now seems little chance of implementing any form of national health insurance for the next few years, I would strongly suggest that the Association direct its immediate attention and energies to the reform of Medicare and existing private insurance. This will mean trying to establish benefit and payment standards at the state, as well as the federal level, a time-consuming process, but well worth the effort. If successful, it will not only improve coverage for millions of patients in the immediate future but help to set the pattern for national health insurance when it does come. In this respect, I know that APA will be working closely with the President's Commission and its special panel on financing.

CONCLUSIONS

The challenges facing psychiatry in the next decade are enormous. A great deal will depend on the basic direction of health care policy as well as on APA's stewardship under that policy. If we persist in following the current high-cost "technological imperative," I see a bleak future for psychiatry, along with prevention, primary, and long-term care in general. The inevitable cost controls will probably be harsh and arbitrary, and many consumers as well as providers will suffer. If, on the other hand, we move gradually to reorient policy and resources toward a less technological and more people-oriented system, with equitable cost restraints developed and applied in a democratic but unambiguous fashion, and if we can rely on public accountability by a freely cooperating profession and industry, the future could be bright indeed—not only for psychiatry but for a revitalized and rehumanized health care system.

In this field, at least, the cherished "American dream" could take on a new meaning. The "limitless frontier" of the future would relate less to geographic, technological, and financial considerations than to the vast potential of the human spirit, a spirit as bold and free as that of the astronauts who walked on the moon but wise enough and humble enough to appreciate the wisdom of Hegel's version of the "categorical imperative": "Freedom is the recognition of necessity."

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Diagnosis: Schizophrenia Versus Posterior Aphasia

BY STEPHEN N. GERSON, M.D., D. FRANK BENSON, M.D., AND SHERVERT H. FRAZIER, M.D.

Schizophrenia and posterior aphasia are easily cross-diagnosed, primarily because of similarities in verbal output, i.e., vagueness, looseness of association, and apparent confusion. Tape-recorded interviews with 8 posterior aphasics and 10 "loose" schizophrenics were transcribed and analyzed to provide guidelines for the clinician to differentiate the two conditions by monitoring verbal expression. Six major differentiating characteristics were identified. The authors present hypotheses that emphasize differing neuroanatomical loci and neuropsychological mechanisms to explain the differences in verbal output in schizophrenia and posterior aphasia.

SCHIZOPHRENIA and posterior aphasia are easily confused and cross-diagnosed, primarily because of similarities in verbal output, i.e., vagueness, looseness of associations, and "confusion." Although the verbalizations of the two patient populations have been characterized separately by many investigators, few studies have compared them in order to determine differentiating features.

In an earlier discussion we stated that

> When a patient with no apparent neurological disturbance produces words easily and in grammatically acceptable combinations, but the output is not comprehensible to the examiner, a diagnosis of abnormality of thought content may be entertained. If in addition, the patient reacts negatively or not at all to the examiner's verbalizations, he may be considered to be out of contact and a diagnosis of psychotic breakdown may be entertained. The above state, however, accurately describes a posterior aphasia. (1, p. 558)

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Because of this verbal behavior and absence of general neurological findings, the posterior aphasic is frequently referred to the psychiatrist, who must differentiate the neologisms, reiterations, pauses, comprehension disturbances, circumlocutions, verbal substitutions, and intrusions of the posterior aphasic from those seen in schizophrenia. Broca's and other anterior aphasias are easily distinguished (2), but separation of schizophrenic output from that of posterior aphasia is admittedly difficult and demands careful attention.

Descriptions of schizophrenic speech are abundant. Bleuler (3) described disordered quantity of verbal output (from press of speech to muteness), blocking, altered intonation, inappropriate affect, inexact word usage, neologisms, condensations, loose associations, word salad, inappropriate ironic juxtaposition of trivial and important concepts, and perseveration. Lehmann (4) noted that disordered verbal behavior in schizophrenia included being overly concrete and overly symbolic, cryptic meanings with language as a means of self-expression rather than communication, neologisms, intermittent muteness, echolalia, verbigerating (senseless repeating of a word or phrase), and being overly stilted, pedantic, and formal. There have been several other descriptions in the literature (5–9).

Diagnostic errors are not surprising because, by habit and training, psychiatrists generate their diagnostic impression from careful attention to the *content* of the patient's verbal utterances, rather than the *quality* in terms of syntax, verbal substitutions, patterns of pauses, agrammaticisms, coherence of verbalizations, etc. Because of the confusing similarities in verbal behavior, posterior aphasics may be misdiagnosed and confined to mental hospitals. It is the purpose of this report to discover and accent those signs of verbal output that would aid the psychiatrist in establishing the correct diagnosis.

METHOD

Interview

The data consist of 15-minute tape-recorded interviews conducted as follows. The interview began with 7–8 minutes of open-ended questions, e.g., "What are you here for?" "Tell me about your favorite hobby." The patient was encouraged to continue talking if he/she stopped verbalizing. Included in the 7–8 minutes were the following directed questions: "What is your

name?" "Where are we now?" "What kind of work do you do?" "Where were you born?"

Subjects

Two diagnostic populations were interviewed. All diagnoses were based on criteria listed below in addition to the diagnosis of the attending physician.

The posterior aphasic group consisted of 8 patients with fluent paraphasic verbal output, i.e., Wernicke's aphasia, transcortical sensory aphasia, or conduction aphasia (2). Criteria for the diagnosis of posterior aphasia depended on characteristic speech production plus confirmatory localizing information, such as neighborhood neurological findings, brain scan, or EEG localization of lesions in the appropriate areas of the dominant hemisphere.

Posterior aphasics produce abnormal verbal output characterized by fluent paraphasic speech. In addition, there may be problems in comprehension of spoken language, disturbances of repetition, serious word-finding problems, alexia, and/or agraphia to a greater or lesser degree. The onset is most often acute and accompanied by elementary neurological findings, but these may be transient, leaving abnormal language as the only residual. Cases with a slow, subtle onset of disordered verbal output are diagnostically treacherous. Appropriate laboratory tests may demonstrate pathology in the dominant temporal lobe or temporoparietal junction area. Each posterior aphasic in this series had both a diagnostic clinical picture and confirmatory laboratory tests.

The schizophrenic group consisted of 10 patients who were evaluated during acute schizophrenic episodes. Each patient was "loose" when tested, although most were medicated at the time of evaluation. Each patient had at least three of Schneider's first-rank symptoms (10). In cases where there had been laboratory tests such as brain scan and EEG, results were negative, and none of the subjects had evidence of elementary neurological dysfunction.

Characteristics of Verbal Output

The following features of abnormal verbal output (chosen from a review of the literature) were noted, tabulated, and compared: 1) appropriateness of response to the question; 2) syntactical pattern, i.e., blocking, pauses, verbigeration, stiltedness of language; 3) tendency to reiterate; 4) comprehension defect; 5) abnormal verbal content, i.e., a persistent theme controlling verbal output, such as an internalized pattern of thought; 6) circumstantiality; 7) circumlocution; 8) echolalia; 9) perseveration, i.e., repetition of a word, phrase, or action used in response to a previous question; 10) neologism; 11) paraphasia, i.e., the substitution of an incorrect word or letter in the output; 12) detailed responses, i.e., redundant, excessive, and inefficient verbalization; 13) condensation; 14) alliteration; 15) onomatopoeia; 16) clang association; 17) intonation defect, i.e., inappropriate affect for the content of the response; and 18) novel characteristics not otherwise described.

The tapes were analyzed independently by two judges who used the list of characteristics described.

RESULTS

Some of the 18 characteristics were of no help in differentiating the two disorders, but others did help in establishing the correct diagnosis.

Appropriateness of response to questions was not helpful. Both schizophrenics and posterior aphasics gave inappropriate responses about 40% of the time, presumably for different reasons. The schizophrenics appeared to be attending to an internal preoccupation, while the posterior aphasics perseverated or failed to comprehend the question.

Analysis of syntactical patterns was of only limited usefulness. Blocking was marked in both groups; stuttering (machine-gun repetitions of the initial phoneme of a word) was frequent in posterior aphasics but occurred only occasionally in schizophrenic output.

A tendency to reiterate or repeat words and phrases was noted in both groups. The schizophrenics tended to reiterate delusional themes when allowed to respond at length. Posterior aphasics repeated words and short phrases, e.g., "But actually, a real rattler has tiny, little, tiny little, uh be . . . be, bey, bey, uh, uh, things, you know." Comprehension problems were also present in both groups but were qualitatively different. Posterior aphasics often comprehended the first few questions but then appeared to "jam" or fatigue. If the examiner could gain the schizophrenics' attention, they comprehended.

Persistent themes appeared to modify and control the verbal outputs of both groups. Schizophrenics were often preoccupied with delusional concerns that intermittently penetrated their response; if the posterior aphasics stuck to one subject, it was a perseveration or failure to change set. The posterior aphasics were circumlocutory while the schizophrenics were circumstantial.

Echolalia was absent in both groups, as were condensations. Schizophrenics did not perseverate, whereas 70% of the posterior aphasics perseverated at some point in the interview. Neologisms were not found in any of the schizophrenics' output. Paraphasia was present in posterior aphasics and absent in schizophrenics. Informational detail was far less in posterior aphasics than schizophrenics, which probably reflected frequent pauses and relative paucity of substantive nouns in the posterior aphasics (substantive nouns accounted for only 3% of that group's output compared with 11% of schizophrenics' output). Alliterations and onomatopoeia were absent in both groups and only 3 of 10 of the schizophrenics demonstrated a rare clang association (1/3000 words). Both groups behaved inappropriately and gave inappropriate responses in ways that were different and characteristic.

Six striking differences in the verbal output of schizophrenics and posterior aphasics were noted. First, the responses of the schizophrenics to the open-ended questions were considerably longer. Individual responses of the posterior aphasics averaged between 20 and 30 words, whereas the schizophrenics' ramblings were typically 100 or more words. The posterior aphasics would pause or look to the examiner for a response or another question after a brief machine-gun-like output of fluent, empty phrases. In contrast, the schizophrenics often produced extremely long responses (700–800 words), stopping only when interrupted by the examiner's next question.

Second, the posterior aphasics demonstrated *some* awareness of their expressive difficulties and were disturbed by it, whereas the schizophrenics either presumed the interviewer understood their vague utterances or were unconcerned about the adequacy of communication. Even relatively recent (i.e., 1–2 weeks after a cerebrovascular accident) severe posterior aphasics demonstrated some awareness that something was awry. As they improved, they became increasingly aware of their problem. Conduction aphasics (the posterior aphasics with the best comprehension) were the most concerned, but even they demonstrated a mild, inappropriate euphoria (lack of concern and awareness), a trait that was most pronounced in those with Wernicke's aphasia.

Third, the posterior aphasics often used nonverbal substitutes or pauses to enlist the aid of the examiner in overcoming word-finding difficulties. In contrast, the schizophrenics' pauses were effortless and autistic, and the examiner did not feel a pressure to offer assistance. Thus, the examiner's subjective feeling was one of pressure to help or relate to the posterior aphasic, but the nonrelating style of the schizophrenic tended to lull the examiner into inattention. The impression that the posterior aphasics were more concerned about their ability to communicate is enhanced by their increased use of nonverbal gestures, i.e., body motion and facial movement.

Fourth, substitutions of incorrect letters, words, or even an occasional neologism were common in posterior aphasia but rare in schizophrenia. In fact there were no neologisms or other paraphasias in our samples of schizophrenic speech. This total absence of neologisms is striking, since most textbooks list neologism as a characteristic of schizophrenic output. All of our posterior aphasics produced neologisms, but they were uncommon (about 1/500 words); other paraphasic substitutions were common, occurring as often as 1/15–20 words.

Fifth, vagueness of response was prominent in both disorders but was clearly derived from different sources. Aphasic vagueness arose from word-finding difficulty. Posterior aphasics produced a comparatively voluminous output with relatively few substantive words. Brief pauses were frequent in the posterior aphasic's output, occurring most often when a substantive noun was indicated. This word-hunting quality differentiated the posterior aphasics' pauses from the thought blocking of the schizophrenics. Thought blocking was not frequent in our sample of schizophrenics, and pauses were less frequent in schizophrenics than in posterior aphasics. The schizophrenics produced a comparatively full lexicon, with shifts in subject matter during speech, apparently secondary to alterations of attention that resulted in incomplete, approximate, and vague responses.

Finally, the difference in content of the output was notable. The schizophrenics produced bizarre themes that were repeated; two or three persistent themes would be reiterated throughout the 15-minute conversation. In contrast, while the posterior aphasics' content presented many repetitions and intrusions, there was no persisting theme, and over the 15-minute interval many disparate themes were introduced.

DISCUSSION

The observations we have presented demonstrate basic differences in the verbal output in the two disorders. The differences can be readily noted by auditing verbal output and may be used to assist diagnosis.

In clinical practice the examiner often has anamnestic information to aid in making a correct diagnosis. For example, loose schizophrenics are usually younger, i.e., in their 20s, than posterior aphasics, most of whom are over 40. A premorbid personality aberration frequently precedes overt schizophrenia. Posterior aphasics may have no such history; rather, they suffer an acute onset of altered behavior. There are situations in which an adequate history is not available and, of course, both disorders can occur at any age. In problem situations attention to the following six characteristic features can prove helpful. In most cases when one differentiating feature was noted, the others were also present.

1. Length of response is shorter in posterior aphasia.
2. Awareness of communication problem is greater in posterior aphasia.
3. Efforts to enlist examiner's aid in communication occurs in posterior aphasia but not in schizophrenia.
4. Substitutions in language (paraphasias) are common in posterior aphasia and rare in schizophrenia.
5. Vagueness of response is present in both groups, apparently due to word-finding problems in posterior aphasia and to attentional disturbance in schizophrenia (circumlocution versus circumstantiality).
6. Content—bizarre themes are present in schizophrenia but absent in posterior aphasia.

Finally, some comments on the theoretical implications of the underlying mechanisms and neuropathological correlates of the two disorders may be heuristically useful. Expressed most simply, the verbal output of the posterior aphasic is a disorder of language, whereas that of the schizophrenic derives from a disorder of thought. Differentiation of abnormalities of

verbal expression into speech, language, and thought disorders (11) can be outlined as follows. A speech disorder is a distortion of otherwise adequate verbal output caused by neuromuscular disturbance involving some part of the speech mechanism. Patients with a speech disorder know exactly what they want to say, but mechanical disturbances interfere with output. Examples include stuttering, extrapyramidal dysarthria, etc. A language disorder is an abnormality of verbal output in which the patient has difficulty manipulating the semantic and syntactic structures necessary to express his ideas; all aphasics have language disorders. A thought disorder is the expression of abnormal ideas through intact language functions. Thus thought disorders represent abnormalities of ideation that are reflected in verbal output. Examples of disordered thought include overly concrete thinking, personalized thinking, idiosyncratic or bizarre thinking, obsessions, phobias, intellectualization, thought withdrawal, thought insertion, thought broadcasting, delusional percepts, overly symbolic thought, dereistic thinking, and magical thinking. Denial, projection, paranoia, circumstantiality, and tangentiality are disorders of thought that are often present in organic mental problems.

Maher (8) contends that normal coherent speech is the result of a successful and instantaneous inhibition of associations to each element in the utterance. A sentence is an extremely complex act demanding continuous inhibition of distracting associations, a process that breaks down in schizophrenia. On the other hand, disruption of syntax, almost a hallmark of aphasia, is rarely found in schizophrenia.

Stevens (12) proposed a subcortical neurological focus for disordered thought in schizophrenia. She hypothesized a breakdown in neostriatal and limbic striatal "gates" ("filtering structures") interposed between the cortical mantle and afferent fiber tracts of the motor, sensory, and visceral systems. Hyperattentional states may occur in schizophrenia when the gate is "overwhelmed" and normal inhibitory abilities are diminished or lost. The patient cannot keep competing stimuli and associations out of consciousness. Coherence of thought and, thus, the patient's sense of continuity and identity are disrupted. Disorders of thought appear to result from malfunction of subcortical CNS activities.

Unlike the disordered verbal output of schizophrenia, which appears to derive from subcortical dysfunction, aphasia results from damage to the cortex (of the dominant hemisphere). Geschwind elucidated the mechanism of posterior aphasia as follows:

> Broca's area is a way station between Wernicke's area on the one hand and the neurons which send axons to the motor nuclei of the speech organs on the other. However, it is more than an intermediary way station. It seems that it contains the rules for turning the sounds of speech into motor acts. It might be called an auditory-motor transducer. (13, p. 189)

Wernicke's area (the auditory association cortex) acts to classify the auditory input into patterns. These patterns, when passed forward, elicit activity in Broca's area to produce the motor activities for speech. Broca's area is, to some extent, controlled by stimuli from Wernicke's area. When the latter is destroyed or disconnected, Broca's area may run on freely, without normal inhibition. The resulting verbal output is fluent, well-articulated speech because many of the sequences of language are overlearned and Broca's area can run on without control. Thus, in a sense, the fluent output of posterior aphasia also results from decreased inhibition. In posterior aphasia, however, it is a lack of cortical control that leads to the disturbed verbal output.

Thus we can hypothesize that the abnormal verbal output of posterior aphasia derives from a cortical-cortical disruption while that of schizophrenia derives from a subcortical-cortical disruption. The six features that differentiate the output of schizophrenia and posterior aphasia would appear to be the differences between the underlying thought disorder in loose schizophrenia and the language disorder in posterior aphasia, which derive from different neuroanatomical disruptions.

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A Study of Treatment Needs Following Sex Therapy

BY ALEXANDER N. LEVAY, M.D., AND ARLENE KAGLE, PH.D.

The authors describe 19 couples who returned for additional treatment after traditional short-term sex therapy. Eight couples required additional short-term sex therapy, and 11 couples were treated primarily with individual psychoanalytic psychotherapy or conjoint therapy for an extended period of time. The authors identify three reasons for return to therapy: 1) treatment overload, 2) severity of symptoms, and 3) presence of psychopathology relevant to the sexual dysfunction. They point out that pleasure, intimacy, and cooperation dysfunctions need to be worked through if sex therapy is to have lasting effects.

SINCE THE PUBLICATION of *Human Sexual Inadequacy* (1) the Masters and Johnson method of rapid treatment of sexual disorders has become widely practiced and acclaimed. Several modifications of the basic technique have been introduced, notably Schumacher's variation of the duration and intensity of treatment (personal communication from S. Schumacher) and Kaplan's departure from dual therapy teams to single therapists (2). Follow-up studies of these therapies so far have indicated that short-term treatment is highly efficacious and efficient and that gains made are essentially maintained for as long as five years (1, 2). Although published studies indicate that approximately 80% of all patients experienced symptom reversal and that few of these relapse, our experience suggests a more cautious judgment and calls for a more complex and comprehensive assessment of treatment results. In particular there is a need for detailed study of long-term symptom reversal and improvement.

This paper will attempt to evaluate the effectiveness of treatment by describing and analyzing the results of the treatment of 19 of 45 couples who were originally seen for classical two-week Masters and Johnson therapy (1) and returned on their own initiative for additional treatment with one of us (A.N.L.) because their sexual adjustment had deteriorated or failed to progress. The first phase of treatment was conducted by a dual sex cotherapy team of psychiatrists who had received formal training in sex therapy at the Reproductive Biology Research Foundation in St. Louis. The second phase of treatment consisted of individual or conjoint therapy or both as indicated by the needs of the couple and was conducted by A.N.L. alone or by referral.

METHOD

Phase 1

Between June 1971 and June 1973, 45 couples referred by themselves or their physicians/therapists for sex therapy were seen for two weeks in Masters and Johnson intensive sex therapy. Criteria for acceptance into treatment included 1) presence of a sexual dysfunction, 2) willingness to work out the problem as a couple, 3) motivation to work out the problem for one's own sexual functioning and pleasure, 4) the absence of prohibitive psychopathology and/or intramarital conflict, and 5) the absence of overwhelming reality or organic factors. All patients were private patients, mostly of upper-middle class background, and ranged in age from 19 to 69 years. Presenting problems included all the categories of sexual dysfunction reported by Masters and Johnson in a comparable distribution. After the 2-week treatment period couples were seen for follow-up at least twice, at 2 weeks and 2 months. In addition they were asked to return for a 1-year follow-up. Only 4 couples did so; 3 are among the 19 reported here.

Of the original 45 couples, 10 cases were judged to be unimproved or deteriorated. In all the remaining 35 cases there was some degree of improvement or complete symptom reversal after the 2-week treatment period. Others in the field have reported similar results (1, 2).

Phase 2

After the termination of the original 2-week treatment period, 19 of the 35 improved couples (54%) returned of their own volition for further therapy with A.N.L. These couples left short-term treatment at all stages of improvement. In all cases the couples described a deterioration of sexual functioning or a fail-

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The authors wish to thank Virginia Lozzi, M.D., Associate in Clinical Psychiatry, Columbia University College of Physicians and Surgeons, cotherapist for all 45 cases described in this paper.

ure to progress in the period following treatment as their reasons for seeking additional help.

During this second phase of treatment couples divided into one of two distinct treatment groups. Eight couples were seen in short-term follow-up treatment, which consisted of additional sex therapy. This first group was characterized by the ability to progress as a couple out of treatment. In each case the deterioration or lack of further progress was accessible to treatment within 10 sessions or less. The other 11 couples failed to respond to a second course of sex therapy and were seen either together or individually for more extensive and traditional psychotherapy. This second group was notable in that in every case there was moderate to severe underlying psychopathology and/or unconscious conflict, which required additional therapy exceeding 50 treatment hours.

RESULTS

Since becoming sexually functional is structured as a step-by-step process in sex therapy, we have chosen to subdivide improvement into five major stages plus a sixth category for individuals who are totally dysfunctional: stage 0, nonfunctional—no sexual desire; arousal, or sensation; stage 1, the identification of pleasurable sexual sensations along with discernible arousal, i.e., erection for the man and lubrication for the woman; stage 2a, the ability to experience sustained arousal and orgasm by self-stimulation; stage 2b, the ability to experience sustained arousal and orgasm by self-stimulation in the presence of one's partner; stage 3, the ability to experience sustained arousal and orgasm by manual or oral stimulation by one's partner; stage 4, the ability to function satisfactorily and experience orgasm in intercourse; stage 5, the ability to integrate stage 4 level of sexual functioning into one's normal life routine on a regular basis.

All 19 couples were evaluated according to these stages at four different points: before treatment, after phase 1 treatment, before phase 2 treatment, and after phase 2 treatment. In terms of sexual functioning, the initial level did not prove to be a predictor of long-term treatment outcome, with the possible exception of a tendency for couples consisting of a sexually functional man married to an anorgasmic woman to require long-term additional treatment. There was also a trend which suggested that couples in which both partners could masturbate to orgasm before treatment had a simpler course of therapy. However, these findings may be an artifact of the small sample size.

An analysis of outcome performed after phase 1 and also before phase 2 and the results after phase 2 therapy were also not useful in determining whether a couple would require short- or long-term additional treatment. Thus, it seems clear that it is not possible to depend on the level of sexual functioning alone in developing a treatment plan for couples requesting sex therapy. What is predictive is the degree of psychopathology: moderate to severe underlying conflicts or marital discord were present in all couples who required long-term additional therapy.

DISCUSSION

Experience in the second phase of treatment (and with some 300 couples seen subsequently in modified sex therapy) indicates that for many couples the treatment format has to be individually tailored and integrated with one or more of the psychotherapies in order to bring about lasting results. Although the treatment of choice for some couples is sex therapy alone, others need additional help. Similar conclusions have been reported in the literature, notably by Kaplan and Sager (2–5).

Reasons for Return

We have subdivided the reasons for return to treatment by these 19 couples into the following categories that we feel are relevant to all couples seeking sex therapy:

1. *Treatment overload.* Some of the patients who returned had experienced sex therapy as too intensive and overwhelming. Two-week treatment had not allowed for the necessary assimilation and working through of basic concepts and new behaviors. As a result these couples were left with a combination of residual problems and uncertainty about treatment direction and goals that did not allow them to grow on their own or resulted in a relapse. Altering the rate and duration of treatment so that the assignment of tasks was more in harmony with the couple's ability to participate resulted in successful utilization of therapy.

2. *Severity of symptom(s).* When the presenting sexual dysfunction was severe and of long duration, the course of sex therapy was often, of necessity, extended. Even with a highly motivated and therapeutically adept individual, it is often asking too much to expect that all the developmental steps and assimilative tasks necessary to become fully sexual can be accomplished in a two-week period. Several couples returned for treatment primarily because they needed to have additional treatment time to work through these required stages. Needless to say, the situation is further compounded when both partners suffer from a dysfunction or when a spouse becomes dysfunctional near the end of treatment as a direct consequence of his/her mate's improvement. Given additional time, however, most couples can resolve these unforeseen problems.

3. *Presence of psychopathology.* Other problems occur if, in addition to the sexual dysfunction, psychopathology is present in one or both spouses. As was seen in the second group of couples who returned, unconscious, unresolved conflicts, various psychiatric syndromes, or emotional immaturity gravely influenced the course of treatment. In cases in which severe pathology is present, albeit latent, the sex therapy

can activate conflict, symptom formation, and even full-blown psychotic episodes. Similar experiences have been reported by Kaplan and Kohl (6) and Meyer and associates (7).

Treatment procedures can be seen as a series of diagnostic tests in which the therapeutic tasks bring out relevant problems with great intensity and clarity. For instance, the need to maintain a parent-child relationship is brought into focus when a couple is asked to interact on a reciprocal basis. Similarly, anxiety reactions have been experienced following a first orgasm by individuals who have clearly exceeded both their superego limitations and ego ideal expectations.

If psychopathology is present in one or both partners, one of three outcomes is possible. For some people the successful completion of a specific sexual task, combined with the powerful emotional backlash reaction that often occurs, somehow serves as a corrective emotional experience and leads to profound insight into the dynamics of the sexual dysfunction. From then on resolution of the unconscious conflict and integration of the newly found sexual gains take place with surprising ease and rapidity. It is important for the therapist not to mistake these dramatic events for an incipient decompensation of the individual or failure of treatment but to support fully and facilitate this process to a successful termination.

Unfortunately this does not occur often. Instead, even without a breakthrough the emotional backlash to the sex therapy is of such magnitude and severity that it requires the temporary or prolonged suspension of this phase of treatment while the intra- or interpsychic problems can be worked out. It helps if during individual or joint treatment the therapist is prepared to reintroduce the spouse and/or the sex therapy as soon as sufficient progress has been made. It is important that neither the patients nor the sex therapist sees this as a failure but, rather, understands that the sex therapy helped focus on problem areas that required work before further sexual progress could be made.

The third possible outcome in cases involving psychopathology is a successful and seemingly uneventful course of sex therapy. This occurs when the dynamic underpinnings of the sexual problem are bypassed without resolution because of the very favorable conditions that sex therapy provides, including therapist support, expected superego relief, and a "honeymoon" atmosphere. All of these provide an ideal setting for a "flight into health" type of symptom reversal. These couples subsequently relapse and require an extensive therapeutic intervention similar to the couples already discussed, but they have the advantage of having already experienced what their sexual lives and marital relationship could be like once they work out their problems.

As we have already pointed out, the effect of such pathology on the course of treatment should not be overlooked or minimized. Although some claims have been made that psychopathology can be successfully bypassed in sex therapy, our experience in long-term follow-up indicates that stable results are only accomplished when and if the relevant intra- or interpsychic problems are resolved or at least reduced. This finding reinforces the importance of competence in diagnosis and broad training in psychotherapy in order to adequately treat sexual dysfunctions.

It is interesting to speculate about the 15 improved couples and the 10 treatment failures who did not return for the 1-year follow-up or additional therapy. Although it is not surprising that the latter group did not reenter a therapy they did not find helpful, we are looking forward to the results of a follow-up study of the entire sample in order to determine their subsequent course of therapy, if any, and their present state of sexual functioning.

Problem Areas

Finally we would like to comment on three factors that have been present alone or in combination in all of the 11 couples who required extensive further therapy. In fact it is useful for treatment purposes to categorize individuals seen for sex therapy not only in terms of presenting sexual diagnosis but also in terms of these factors, since they seem to play a key role in perpetuating sexual dysfunction. We see these as impairments in specific ego functions and have labeled them as problems in the areas of pleasure, intimacy, and cooperation.

Pleasure dysfunctions are primarily associated with an inability or difficulty in identifying, experiencing, and/or enjoying pleasurable sensation, i.e., difficulty in having sex for oneself. Pleasure dysfunctions often respond well to standard sex therapy, at least initially, since the treatment offers techniques for dealing with the lack of attention (low arousal) or overattention (spectatoring) often associated with this condition and also provides superego relief. However, when sexual activity is no longer monitored by the therapists, patients often regress or relapse.

Intimacy dysfunctions are primarily associated with inability or difficulty in experiencing and/or enjoying closeness, binding, and union with another person. Such individuals often find it hard to carry out the prescribed activities of sex therapy that focus on the mutual aspects of sexuality. Here difficulty is associated with having sex with another person. Again short-term treatment provides techniques for dealing with these blocks, but when the couple's intimacy is no longer supported as well as diluted in treatment by the therapists, relapse is likely to follow.

Many variants and varieties of marital discord are subsumed under the label of cooperative dysfunctions. The primary aspect of this factor, however, is an inability to collaborate, act as a team, or agree on common goals, priorities, or values. Although pleasure and intimacy dysfunctions can be unilateral, cooperative disorders almost invariably involve both spouses. Often what underlies this condition is a need by both partners to compete with or deprive each other, usually accompanied by other manifestations of transfer-

ential rage and retaliation. Giving to the partner sexually, or in other ways, is experienced as a loss or defeat. This can be described as a difficulty with sex for another person. Consequently, during sex therapy, pleasuring the partner is seen as self-negating, and treatment often becomes stalled when the task involves reciprocity. For these couples conjoint therapy is often essential before any meaningful sex therapy can get under way.

When these dysfunctions are found in combinations, treatment is much more difficult and time consuming. In our experience, stage 5 sexual functioning is impossible until all these areas of difficulty are worked through to a considerable extent.

CONCLUSIONS

Sex therapy is a powerful, effective, and efficient therapy. However, it is not a panacea. Work with 19 couples who returned for additional treatment following traditional short-term sex therapy served to highlight the types of residual problems that necessitated additional treatment. The presence of the type of psychopathology that blocked progression or maintenance of gains in sex therapy proved to be a differentiating factor between the 8 couples who required only short-term intervention and the 11 couples who required long-term additional therapy.

It is useful for treatment purposes to categorize patients not only in terms of presenting sexual diagnosis but also in terms of the types of psychopathology (of pleasure, intimacy, and cooperation) that interfere with progression in sex therapy. We have found that these dysfunctions need to be worked through if sex therapy is to have lasting effects. When these factors are present in combination or alone, sex therapy is a helpful but insufficient means for successful resolution.

Our experience indicates that sex therapy has to be further modified in order to maximize and stabilize gains for each couple. The treatment format needs to be open ended; flexible in the rate and sequence of task assignments; and, when indicated, coordinated or integrated with other psychotherapies, principally psychoanalytic psychotherapy and conjoint therapy.

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On the Course of Anorexia Nervosa

BY REGINA C. CASPER, M.D., AND JOHN M. DAVIS, M.D.

The authors trace three phases in the course of anorexia nervosa and compare its physical and psychological symptoms with those of starvation. Phase I, which may occur months or years before the illness, usually includes precipitating events that result in loss of self-esteem and increased self-consciousness about physical appearance. During phase II patients develop the "anorectic attitude," an unreasonable fear of eating, and show pride in their ability to lose weight. By phase III patients are forced by the severity of starvation symptoms to admit that they are ill. Although many of the physical symptoms of starvation and anorexia nervosa are similar, anorectic patients, in contrast to victims of starvation, show high initiative, the ability to suppress hunger, restless hyperactivity, and body image distortion.

THE HISTORY of anorexia nervosa shows that it has not been difficult for the observant clinician to distinguish anorexia nervosa from starvation due to other conditions (1). A willful quality in the patient's refusal to eat and an insistance on thinness invariably associated with the emaciation led Gull (2) to conclude that "the want of appetite is due to a morbid mental state." Many physicians since the last century (3–7) have been fascinated by anorexia nervosa, yet few have seen and treated enough patients (8–11) to validly explore its psychological aspects.

When we were evaluating patients for a multicenter study designed to compare different treatment approaches to anorexia nervosa we noted that most of the patients' parents had initially mistaken the illness for a dieting fad, and many had remained unaware of a more serious problem until the child was markedly wasted. These observations led us to look closely into the natural history of anorexia nervosa. For this purpose we took a careful history of the sequence of events and systematically asked certain questions of both the patients and their families to trace the appearance and development of symptoms.

Furthermore, by talking to and treating hospitalized patients with anorexia nervosa we came to the conclusion that the effects of starvation are more intimately involved in the symptoms of anorexia nervosa than has been heretofore described. In order to define more clearly which symptoms are primary to anorexia nervosa rather than secondary to starvation, we compared the psychophysiological symptoms of starvation reported in the literature (12–18) with the symptoms shown by anorectic patients.

METHOD

Data were collected in evaluation interviews with approximately 30 patients who were referred for admission and their families and through observation of 20 hospitalized patients who were treated and followed after discharge in a larger collaborative treatment study supported by the National Institute of Mental Health (19).

Informed consent was obtained from the patients and their families after a detailed explanation of the treatment protocol.

Despite the fact that Feighner's diagnostic criteria for anorexia nervosa (20) contain some inaccuracies, such as classifying overactivity in the same category as bradycardia, they were used as an operational framework for the decision to include patients in this study (see appendix 1).

RESULTS

On admission patients had lost from 30% to 46% of their normal weight. Depending upon age and height, body weights ranged from a low of 26 kg to a high of 43 kg. The illness had lasted from 7 months to 6 years. All patients had been active in school, college, or work prior to admission. Only 2 patients were obese prior to weight loss. All patients were female.

We have found that conceptually dividing the course of anorexia nervosa into three phases is useful both in understanding the disorder and in the clinical management of patients. We believe that different symptoms and dynamics characterize each phase.

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Phase I

We systematically collected information on precipitating events and early symptomatology. External precipitating events that preceded the weight loss by months or years, such as siblings leaving the house, separation from or loss of friends, illness of a family member, going away to college, or getting married, could be identified in all cases. These events put the patient's self-esteem and self-concept to a severe test, resulting in painful self-doubt (e.g., "I felt I was not worth very much") along with feelings of powerlessness and loneliness.

This situation led patients to become preoccupied with themselves and more conscious of their physical development and appearance. Once they had attributed their dissatisfaction and general unhappiness to concerns about body size (most often about being "too chubby"), comments about a "round face" or a "tummy" were painfully remembered and dwelt upon and became the motive for the decision to diet vigorously. "If they don't like me as a person they cannot help but admire my body," said one patient; another stated, "This time I decided to really make it work." Although 3 patients lost weight accidentally through an illness and some patients used uneasiness after eating and vague sensations of fullness after meals as a pretext for reducing food intake, all patients stated that they had previously "had this idea in the back of my mind, to go on a diet."

Phase II

Most patients felt relieved and happier once they had begun to systematically and successfully reduce their food intake and had lost weight: "I had so much willpower, I felt so proud." It was also during this period that the anorectic attitude, an unreasonable fear of eating, became noticeable.

In order to highlight symptoms peculiar to the disorder, we wish to conceptually distinguish symptoms that could be secondary to starvation from those that occur only in anorexia nervosa. Anxiety related to food intake, expressed by the patient as a fear of becoming fat with each bite of food that effectively counteracts more or less continuous hunger feelings, is not observed in starvation but is pathognomonic for anorexia nervosa. Exactly when and how this fear develops could not be ascertained, although all patients said that they became aware of it after a certain amount of weight was lost.

Hunger awareness was reported by the majority of our patients (e.g., "I was hungry but it was so easy to pass up"). Patients could suppress hunger awareness quite successfully, particularly by exhausting themselves through exercise. Most patients complained after eating minimal amounts of food about feeling full and bloated yet not satiated or satisfied. Patients needed to consciously disregard this sensation of fullness in order to eat enough to gain weight. For many patients the sensation of an empty or full stomach, rather than normal feelings of hunger and satiation, seemed to be the primary regulator of food intake. One patient said, "The only feeling I wanted to feel was an empty stomach, and then I knew I was skinny." More important, all patients spoke about the pleasure and sense of achievement they gained from losing weight successfully; these feelings contrasted with their previous feelings of powerlessness and lack of enjoyment. As one patient put it, "I couldn't do many things, but I could do this."

Another symptom, which was first described by Bruch (11) and has never been reported in normal persons under conditions of starvation, is the failure of patients with anorexia nervosa to adjust their body image gradually to the progressive emaciation. Patients did not feel abnormally thin and consequently did not see any need for gaining weight. They typically said, "I don't see myself as really thin." It is surprising that patients without fail accurately perceive the emaciation of other patients with anorexia nervosa. As one said, "I can look at them, boy, they are really thin."

Appendix 2 compares behavioral and psychological symptoms observed during starvation states (12–15) with those of anorexia nervosa. Most of the information on starvation was gained during the Minnesota experiments on semistarvation (13). Although Keys and associates (12) used only young men recruited among conscientious objectors as subjects, the conditions under which the experiments were conducted came close to the situation involved in anorexia nervosa, in that the subjects were volunteers who were "starving in the midst of plenty." Most of the other literature on starvation (14–18) describes conditions in which subjects were not only deprived of food but also of their freedom and lived under severe duress.

As appendix 2 indicates, food restriction and a decreased caloric intake result in loss of strength and endurance accompanied by irritability and general discomfort. Subjects go through periods of increased worry and temporary depressive feelings, with a decrease in outside interests. This is not so in anorexia nervosa; when patients are lighter in weight they feel more vigorous, seem to have a reservoir of inexhaustible energy, and get involved in all kinds of activities. In fact, this restless hypermotility, which was present in all our patients, is a symptom regularly encountered in anorexia nervosa. Patients described a restless, sometimes exhilarating, feeling which prevented them from sitting still for a long time unless they were doing something. Restlessness and a quickening effect are sometimes encountered during food deprivation and are probably related to so-called appetitive behavior, which stimulates moving around. However, during starvation subjects generally attempt to conserve energy and reduce their activities, whereas anorexia nervosa patients continued to do exercises in order to lose weight. Anorexia nervosa patients pursue activities with a driven, almost obsessive quality; exercise programs are often highly structured and ritualistic, such

as doing exactly 150 rope jumps or 120 push-ups three times a day.

Another phenomenon that is typical in anorexia nervosa but is only reluctantly admitted by the patient is a constant preoccupation with thoughts of food or eating and difficulty concentrating on other matters. This has also been regularly experienced during prolonged fasting and is an expression of the strength of the hunger drive. Although the eating habits of both groups were strikingly similar (including the urge to handle food, cutting it into small pieces, picking at crumbs, and impulses to hide, hoard, or steal food), they were rationalized differently. During semistarvation these patterns were usually explained by a need not to lose out on any food. Anorectic patients, in contrast, usually complained that a meal was too large and stated that they found it easier to tolerate in smaller pieces; however, they could give no explanation for their urge to touch and handle food. Saving food during starvation seemed to give subjects an illusion of a safeguard in a situation over which they had little control. Anorectic patients called their hoarded food a "safety blanket" but felt ashamed and guilty about it, partly because they were aware that such behavior appeared senseless and unreasonable to others because they had access to an abundance of food. Another similarity between the two conditions is the occasional breakthrough of ravenous hunger feelings, sometimes with bizarre tastes and food preferences. Although daydreaming and fantasizing about food diminishes in starved persons when food intake is increased, in anorexia nervosa anxious ruminations about food and weight continue, with patients expressing fear that "one of these days I will wake up fat."

Phase III

Whereas patients who could accept treatment in Phase II were often successfully treated as outpatients, patients in phase III generally needed hospitalization. In this phase the patients' indifference to their bodily changes extended to all symptoms secondary to starvation and malnutrition. However, after a loss of 25% to 50% of normal body weight the effects of starvation (see appendix 3), such as hypotension resulting in dizzy spells, muscle wasting with weakness, hypothermia with blue and cold extremities, insomnia, amenorrhea, and hair and skin changes, had become so severe that patients eventually hesitantly acknowledged that there could be something wrong. At the same time, they still insisted that they did not feel thin. The arms were the only body parts perceived as wasted, probably because patients could readily inspect them and thus correct the mental image of their arms by visual perception.

Most patients during this stage made some ineffective attempt to eat more or stop regurgitation, but after a slight increase in meals and weight they panicked, frightened by the feeling that their bodies had become larger. They expressed the feeling that "Once you get started [eating], you don't know how to quit."

Despite their emaciation, patients denied any awareness or intention of self-destructiveness. Although some patients admitted that the thought that they might die had crossed their mind, all asserted that they did not want to die. Suicidal thoughts generally did not become conscious until the starvation process was reversed in treatment.

Since they were isolated from friends, struggling with their family, and weakened by malnutrition, patients had some difficulty in concentrating and in functioning; most, however, remained surprisingly mentally alert. We systematically asked anorexia patients during treatment if they had known beforehand that they would come to their present state by continuing to diet. They uniformly answered "no," thus indicating that they did not have full control over the process.

CONCLUSIONS

We have described three phases in the course of anorexia nervosa. The prodromal phase, which precedes the weight loss by months or years, is characterized by an emotional disequilibrium in reaction to traumatic events and eventually leads to the decision to restrict food intake. It is our impression that a process not unlike the one that precipitates the depressive syndrome may precipitate anorexia nervosa. Patients who develop anorexia nervosa make an active attempt to change their appearance instead of submitting to the depressive process. What apparently happens is that an initial disturbance in self-esteem eventually finds expression in a disturbed self-concept. Once the anorectic syndrome is fully developed, different psychological factors seem to be involved, but those contributing to phase I may again become important in the later stages of treatment.

Phase II marks the beginning of anorexia nervosa proper, with the development of an abnormal attitude, the "anorectic attitude," which is governed by fear on the part of the patient that she is in danger of overeating and becoming fat. The sequence of events reconstructed from the patient's history suggests that in predisposed individuals weight loss begun for psychological reasons could trigger anorexia nervosa through psychological and, possibly, biological mechanisms. Although the "hunger center" seems to be intact, a finding consistent with that reported by Garfinkel (21), the status of feelings of satiation is more difficult to ascertain. Thus there might be some dysfunction of the "satiety center." However, since disturbances in the perceptual transformation of the body image and the visceral sensations connected with hunger and satiety are so prominent, a suprahypothalamic disturbance appears more likely to be involved in these symptoms.

Despite many similarities between the psychological and behavioral manifestations of anorexia nervosa and starvation states due to other causes, characteristic features of anorexia nervosa such as fear associated

with food intake and the four areas of disordered body and self-perception described by Bruch (11) (absence of concern about even advanced emaciation, denial and disordered recognition of visceral sensations, lack of awareness of weakness and fatigue, and a paralyzing sense of ineffectiveness) have not been reported in starvation.

In phase III, starvation effects overshadow the anorectic symptoms. Although the absence of concern over the wasted appearance extends to all physical signs of starvation, there is also an increasing awareness of the abnormal physical state. This probably occurs because starvation effects reach awareness through bodily sensations; once these sensations become numerous and severe they cannot be repudiated by conscious experience as completely as the perception of the body image, which lacks a sensory equivalent. We found that in many cases directly pointing out to patients their true physical state was the only way to motivate them for treatment.

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APPENDIX 1
Criteria for Diagnosis of Anorexia Nervosa*

1. Weight loss of at least 25% of original body weight or at least 15% below normal weight for age and height.
2. A distorted attitude toward food, eating, and weight, in particular, a pleasure in losing weight that overrides hunger, admonitions, reassurances, and threats; persistence in an idealized slim self-image and fear of gaining weight.
3. Amenorrhea of at least 3 months' duration.
4. Presence of physical signs such as lanugo, bradycardia, periods of overactivity, vomiting, episodes of bulimia, and hypothermia.
5. Absence of known medical illness or psychiatric disorder such as schizophrenia or major affective illness.
6. Onset between the ages of 10 and 30 years.

* Modified version of Feighner and associates' criteria (20).

APPENDIX 2
Comparison of Psychological and Behavioral Changes in Starvation and Anorexia Nervosa

MOOD OR FEELING STATE

Starvation: Lack of initiative; labile mood, quarrelsomeness; indecisiveness; deterioration of personal appearance; continuous hunger.
Anorexia nervosa: Initiative high; labile mood, alternating with feeling good; "strong-willed"; pride in personal appearance, occasional exhibitionistic tendencies; hunger present but easily suppressed.

MENTAL CONTENT

Starvation: Thinking and dreaming about food; concentration of interest on food with narrowing of unrelated interests; daydreaming, reading, and conversing about food.
Anorexia nervosa: Same as in starvation, but preoccupation with thoughts of food continues after weight gain.

EATING BEHAVIOR

Starvation: Bizarre tastes; preference for bulky foods and hot meals; dwelling a long time over meals; picking up crumbs; bulimia.
Anorexia nervosa: Same as in starvation, but low-calorie foods and fluids are preferred, and carbohydrates are avoided.

ACTIVITY LEVEL

Starvation: Fatigue; avoidance of physical exertion; restlessness and periodic quickening effect.
Anorexia nervosa: Seemingly inexhaustible energy; physical exercise sought; overactivity; restlessness with periodic quickening.

SEXUAL ACTIVITY

Starvation: Decrease in sexual fantasies, feelings, and interest; impotence; amenorrhea.

Anorexia nervosa: Same as in starvation; amenorrhea often precedes weight loss.

APPENDIX 3
General Effects of Starvation

Wasting of subcutaneous adipose and muscle tissue
Hypotension
Bradycardia
Hypothermia and increased tolerance to heat
Acrocyanosis
Loss of hair
Growth of "lanugo" hair
Constipation
Edema
Nocturia, polyuria
Sleep disturbances with early morning awakening

The Manfred S. Guttmacher Award

The American Psychiatric Association invites applications for its annual Manfred S. Guttmacher Award for 1978. The award is given for any outstanding contribution to the literature of forensic psychiatry in the form of a book, monograph, paper, or other work, including audiovisual presentations, submitted to or presented at any professional meeting or published during the period from November 31, 1976, to December 1, 1977. The award, which includes an honorarium of $250 and a plaque, will be presented at the Convocation of Fellows at the 1978 APA annual meeting in Atlanta, Ga.

Anyone who wishes may apply to receive the award by submitting five copies of the work as well as five abstracts to Carl P. Malmquist, M.D., Chairperson, Guttmacher Award Board, 1700 18th St., N.W., Washington, D.C. 20009. Entries will be acknowledged but will not be returned.

The deadline for submissions is December 1, 1977, and any entry to be considered must be in Washington by that date.

Revolutions and the Rights of Man

BY G.M. CARSTAIRS, M.D.

The author describes the dialectical struggle in psychiatric treatment between those who advocate maximum liberty in the management of the mentally disordered and those who believe such patients will benefit more from firm guidance and discipline. He uses illustrations from British psychiatric history to illustrate this struggle. He also points out and discusses two current challenges to psychiatry worldwide: the unconstructive hostility of the various schools of antipsychiatry and the political abuse of psychiatric diagnosis and treatment, particularly in the USSR.

THE HISTORY OF PSYCHIATRY is punctuated with revolutions and counterrevolutions. This is due to the fact that in both the area of theoretical understanding and the area of treatment policy antithetical schools have vied with each other for dominance. The contest has been between those who believe the basic underlying forces in mental illness to be spiritual, if not supernatural, and those who believe that ultimately all mental illnesses will be intelligible in pathophysiological terms. We all remember Hippocrates' challenge to the description of epilepsy as the sacred disease; he wrote, "It seems to me to be no more divine and no more sacred than other diseases but, like other affections, it springs from natural causes."

This dialectic continues in the rather sterile antithesis between those who place undue emphasis on either organic or psychological causes—as if either aspect can meaningfully be excluded. In this paper, however, I wish to concentrate on the dialectical struggle in the area of treatment between those who advocate maximum liberty in the management of the mentally disordered and those (not necessarily less benevolent in their motives) who believe that such patients will benefit from firm guidance and discipline. I will discuss this struggle with illustrations from British psychiatry.

FREEDOM VERSUS CONTROL

The antithesis between freedom and control can of course be recognized not only in psychiatry but in many other human activities as well. In the history of art it appears and reappears as the conflict between romanticism and classicism, between the Dionysian and the Apollonian schools. In education, it manifests itself in the contrast between clearly structured, disciplined teaching and the type of schooling that concentrates on eliciting the child's own interests as the motivation for study.

Until very recently the debate between advocates of "traditional" and "progressive" schooling was based almost entirely on the protagonists' personal preferences. In 1976, however, a controlled comparative study of primary schools in England (1) showed that the more traditional ones were significantly more successful not only in teaching children reading, writing, and arithmetic but also in eliciting imaginative work. The study also found that children in progressive schools showed higher levels of anxiety than did those in the more structured settings. All this was a rude shock to the advocates of permissive education, although they derived some consolation that the most progressive school studied was also the one whose students performed best on the formal tests of learning.

The debate about freedom or control in the management of the mentally ill has shown remarkable ups and downs. The eighteenth century, the Age of the Enlightenment, was a period in which educated people cherished unfettered intellectual inquiry as man's highest activity. Therefore, mental illness, which interferes with clarity of thought, was regarded as the supreme affliction. The eighteenth century was also the age in which two nations experienced a new crystallization of their views as to what are the essentials for human freedom and carried out successful revolutions in order to assert these values. The Declaration of Independence of the United States was dedicated to democracy. In spite of all its tribulations that ideal still thrives in the United States. The French Revolution took "Liberté, Egalité, Fraternité" for its slogan, but even before the revolution had run its course the revolutionaries were inflicting appalling cruelties on their fellowmen.

Thomas Paine, whose eloquence in defense of the rights of man contributed significantly to the events of 1776 in the United States, found that the Parisian revolutionaries initially welcomed his support, only to imprison him when he drew attention to their own violations of human rights. Robespierre himself had Paine's name put on the list of those to be purged. Even when in prison and threatened daily with a trip to the guillotine, Paine was able to write,

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> Painful as the want of liberty may be, it is a consolation to me to believe that my imprisonment proves to the world that I had no share in the murderous system that then reigned. That I was an enemy to it, both morally and politically, is known to all who had any knowledge of me; and could I have written French as well as I can English, I would publicly have exposed its wickedness and shown the ruin with which it was pregnant. . . . Imprisonment with preservation of character is preferable to liberty with disgrace.

(Have we not heard very similar assertions in recent years of the importance of human freedom from contemporary prisoners of conscience—Bukovsky,[1] Plyushch, Solzhenitsin, Fainberg, Sakharov, Roy and Zhores Medvedev, and so many others?)

In spite of its excesses, the French Revolution's initial drive for the liberation of the oppressed provided a favorable climate of opinion for Philippe Pinel's great reforms, symbolized by his striking off the chains from patients in the Bicêtre and the Salpêtrière in Paris. These reforms were applauded by liberal thinkers.

THE COURSE OF REVOLUTIONS

It is a curious facet of history that so many revolutionary movements begin with generous proclamations of brotherly love and sharing of all worldly possessions but end in bloodshed and injustices as bad as those which they set out to reform. This has been well documented in historian Norman Cohn's book *The Pursuit of the Millennium* (2). The millennial movements Cohn described all followed a similar course. They all began with sanguine prophecies of a new era of justice, equality, and brotherly love, in which plenty would be enjoyed without effort by all mankind or, at least, by the elect. As reality supervened and these magical expectations were disappointed, the movements became angry and violent and usually ended in bloodshed.

Cohn showed that irrational revolutionary movements have always occurred at moments in history when large groups of people found their settled mode of existence disrupted by sudden social or economic change. As a result, they were filled with uncertainty about their own personal future and that of their families. Cohn hypothesized that this sudden undermining of secure expectations made such groups receptive to the wish-fulfilling prophecies of mentally unstable leaders. However, Cohn's account is biased because by focusing on the recurrent fantasy of a new golden age he excluded other forms of adaptation to rapid change.

There have been many instances of organized groups, and sometimes whole nations, that have reacted to sudden change by adapting themselves realistically to the new social environment. Among such populations one must include the Russian and the Chinese people, both of whom have brought about new regimes that offer their citizens much better standards of living than they had before—but at the cost of enforced social conformity.

In Russia, those who are so rash as to dissent from the values of the regime run the risk of being declared insane and compelled to undergo psychiatric treatment. In China, according to the reports of visitors (3), the pressure to conform is evident even in psychiatric hospitals, where the mentally ill are involved in daily group discussions with staff and fellow patients. They are fully informed about their illness and about the treatments that have been prescribed, all of which seems commendable. However, they are also urged to view their lapse from rationality as an antisocial state from which they must try to emerge by self-examination and by studying the precepts of Chairman Mao.

LIBERTARIAN MOVEMENTS IN BRITISH PSYCHIATRY

It occurred to me to consider to what extent fluctuations of emphasis can be seen in the history of libertarian movements in psychiatric care in Britain during the last 200 years. The landmarks of any such review must be the new regimen of moral treatment, established in the Retreat at York in 1796; Dr. John Conolly's campaign for no restraint; the mid-nineteenth-century enthusiasm for occupational therapy; and the advocacy of open doors in the new county asylums built in the 1850s and 1860s.

Each of these landmarks was the work of a minority of enlightened individuals. The general standard of patient care in Britain left much to be desired, as was shown by Dorothea Lynde Dix when she toured British mental hospitals in the 1860s. Without exception the libertarian reforms lost most of their original impetus before the end of the nineteenth century, although one of the finest buildings in Edinburgh's Bangour Village Hospital, built as late as 1906, had carved in stone over its porch the brave words "curative workshops."

It can perhaps be argued that moral treatment, occupational therapy, and open doors declined because exaggerated expectations had been associated with their introduction. When expectations for reforms were more realistic the reforms continued to form part of the hospital regimen (as at the Lincoln Asylum, under Charlesworth and Gardiner-Hill, and at the Chrichton Royal Hospital in Scotland, under W.A.F. Browne).

Work in the fields and vegetable gardens attached to the nineteenth-century mental hospitals also formed part of the patients' regimen, but its therapeutic effect was blunted by the low level of skill expected of the patients, who found it a monotonous and unstimulating chore. However, in the 1930s interest began to revive in the effectiveness of a busy, well-planned pro-

[1]Since this paper was written, Bukovsky has been released to the West in exchange for a Chilean political detainee. He has made several public appearances; he has shown no evidence of being psychiatrically disturbed in any of them.

gram of work, as the German psychiatrist Hermann Simon had advocated and demonstrated during and after World War I.

Toward the end of the 1930s George Bell, a young Scottish psychiatrist, was so dismayed by the prison-like atmosphere of the first mental hospital in which he worked that he made it his ambition to direct a hospital in which there would be no locked doors. He was appointed superintendent of Dingleton Hospital in Melrose in 1939 and at once started to open its locked wards. In spite of the interruption of six war years, he persevered, educating his nursing staff and the surrounding community into a new tolerance for harmless eccentricity on the part of his patients. By 1949, the last ward was unlocked; by this time the Melrose rugby team had come to appreciate the presence of a strong body of supporters from Dingleton Hospital whenever they played on their home ground.

Two other contemporaries of George Bell, Duncan MacMillan of Nottingham and T.P. Rees of Croydon, soon followed suit in opening all their wards, but for them this was only one aspect of very comprehensive rehabilitation programs aimed at remedying and, if possible, preventing the apathy associated with prolonged hospital stay. Together with Arthur Bowen of York, they were also pioneers in linking the work of the psychiatric service closely with that of the social services, which would become responsible for the patients' aftercare. By their own exertions, each of these succeeded in creating a network of community care that has not yet been matched in the country as a whole.

Two other outstanding figures in modern British psychiatry deserve special mention, Maxwell Jones and R.D. Laing. I find their contributions fascinating because they exemplify the contrasting accomplishments of two charismatic figures, one of whom always kept one foot on the ground while the other took off into the clouds. Maxwell Jones's special contribution was to teach us, by his example, how to create a genuinely therapeutic community in which the contributions of nurses, occupational therapists, social workers, relatively untrained "social therapists," and, above all, the patients themselves were all employed in the task of understanding and helping those who were experiencing mental or emotional problems. Here I speak as one who has tried to copy Maxwell Jones's techniques in my own clinical work: imitation is the sincerest form of flattery.

R.D. Laing will be remembered for his remarkable intuitive insight into the experience of the schizophrenic individual and for the generosity and compassion that have inspired his form of treatment. However, he will be remembered less flatteringly for his flights of speculation (less in evidence now than in the late 1960s) that seemed to claim that a schizophrenic breakdown can be a rewarding experience, a "journey to the interior of the self" from which one can return with one's personality enhanced. This sounds wonderful, especially in Laing's eloquent prose, but there are all too few returned travelers who can vouch for the authenticity of his claims.

A sketch of British psychiatry over the past two centuries would not be complete without referring to the striking improvement in the quantity and quality of research and training in the field during the past 30 years. This was due in very large measure to the work of Sir Aubrey Lewis and his collaborators at the Maudsley Hospital, London. For more than a generation practically every leading psychiatrist in Britain had part or all of his/her scientific training at the Maudsley. Today Sir Aubrey's former students chair most of the country's university departments of psychiatry. The other main source of research and new ideas has been the Tavistock Clinic and its associated Institute of Human Relations. Its role as a center of psychoanalytic training and for applying psychoanalysis in industry and in life in general was the more important because of Lewis' hostility to psychoanalysis.

Some critics have argued that Sir Aubrey's influence was largely negative. By stressing the importance of the scientific method and, above all, of the critical evaluation of etiological theories and therapeutic claims, he and his followers succeeded in clearing away a great deal of dead wood. Today we are much more modest in our claims of understanding the processes underlying mental illness, but at least our modicum of knowledge has a firm scientific base; this was not so before Sir Aubrey's contribution.

In this paper I have paid particular attention to fluctuations in the humane or libertarian care of the mentally ill. I have indicated that fluctuations and, indeed, regressions in the standard of care have been most evident when the advocates of particular reforms became overenthusiastic, made excessive therapeutic claims, and raised hopes that in the long run were doomed to disappointment. There is an interesting paradox here. The very enthusiasm and confidence of those who conduct any therapeutic regimen have repeatedly been shown to contribute to its success, but this apparently continues to hold true only as long as the confidence is reality-based. In other words, enthusiasm alone is not enough; it has to be underpinned by procedures that of themselves (not merely because of the personality of the therapist) are capable of effecting change.

This calls for a combination of two rather different qualities of temperament. Zealous reformers tend to be emotionally committed, in a hurry to do good, and impatient of criticism as to whether their innovations are going to be helpful to patients in the long run. They want action without delay and are convinced that any action that is generous in intention must be beneficial. Unfortunately, this has often proved to be a false assumption.

On the other hand, those with a bent for verification are less concerned with action for its own sake than with action that has been proved to be efficacious. There is a genuine conflict between these two temperaments that can find expression in mutual hostile criticism. It can also find more constructive expression

when the enthusiasts recognize the need to verify and, perhaps, in the light of operational research, to modify their expectations and when the evaluators give due credit to reformers for the initial impetus without which improvements in care could never be achieved.

Only when the enthusiasts and the evaluators work together will we see a lessening in the fluctuations of emphasis in patient care. Reforms of proven worth will then become accepted as standard practice instead of relying on some individual's personal leadership and drive. The surest defense against false hopes and relapses into inhumanity and neglect lies in our willingness to tolerate criticism and to apply it where we see the need for it.

TWO CHALLENGES TO PSYCHIATRY

Two major challenges confront the discipline of psychiatry at the present time: 1) the unconstructive hostility of various schools of antipsychiatry, whose denial of the reality of mental illness and of the physician's role in helping the mentally ill has raised great hopes among the general public, and 2) the political abuse of psychiatric diagnosis and treatment, particularly in the USSR.

Antipsychiatry

In antipsychiatry we see an example of a generous, liberating movement that has turned sour because it was partially based on unreal expectations. When this happens the "liberators" very often seek a scapegoat, some group of individuals who they believe are actively thwarting the achievement of their humane goals.

Antipsychiatry offers us an opportunity to put into practice that readiness to listen to criticism to which I referred above as the best defense against lapsing into dogmatism and error. When I say listen to criticism, however, I do not necessarily mean to accept it; rather, we should examine the critic's arguments as dispassionately as we can and remain open to change when we find them well founded. I believe there are many genuinely compassionate, generous, and well-motivated people among the antipsychiatrists. They are playing an important role in compelling us to reexamine many of our assumptions and practices, and the history of psychiatry shows us how important it is that such reexaminations be carried out—whether by psychiatrists such as Philippe Pinel or R.D. Laing or by concerned laypeople such as the Tukes (the founders of the Retreat at York) or Dorothea Lynde Dix.

The Political Abuse of Psychiatry

The abuse of psychiatry for political ends is, unhappily, only one element in a worldwide regression toward authoritarian, tyrannical regimes that increasingly impose the suppression of criticism, torture, and arbitrary imprisonment without trial in order to buttress their rule. We see particularly gross examples of such regimes today in Chile and in South Korea, but the denial of basic human rights is flagrant in many other countries.

It is principally in the USSR that political dissent has come to be equated with insanity and dissenters forcibly subjected to psychiatric treatment. In a way, this represents the survival of an old Russian tradition. More than a century ago Tsar Alexander I had a military cadet named Zhuko declared insane because he wrote verses about freedom. His successor Tsar Nicholas I decreed the philosopher Chaadaev (a friend of Pushkin) to be insane because he published an article criticizing certain tyrannical actions of the tsar's regime.

In recent years the Soviet regime has been emulating the tsars. Men such as General Grigorenko, Ivan Yakimov, Leonid Plyushch, and Victor Faiberg, who have dared to criticize such Soviet actions as the invasions of Hungary and Czechoslovakia, have not been tried in public but have been secretly declared insane and incarcerated in prison mental hospitals. When the young psychiatrist Semyon Gluzman tried to draw attention to the lack of medical evidence to substantiate the psychiatric diagnosis in several of these instances he was savagely persecuted and is still in prison. Another young man, Vladimir Bukovsky (now 35 years old) was responsible for delivering to the West a 150-page documentation of cases of the wrongful detention in psychiatric hospitals of political dissenters; he has suffered grievously for this highly principled action.

In 1971 Roy and Zhores Medvedev published *A Question of Madness* (4), a most spirited account of their joint struggle to effect the release of Zhores, who was carried off to a mental hospital on insufficient grounds. This book is at once a horror story in its depiction of a Kafkalike regime, and a heartening story, in its revelation of the courage, forthrightness, and sincerity of men like Sakharov, Solzhenitsin, and many others who were—and still are—prepared to speak up in condemnation of such violations of human rights. This struggle is still going on; we owe it to our colleagues in the USSR to support them in their fight against the perversion of psychiatry.

The World Federation for Mental Health was among the first to protest such actions; it has done so at each of its biennial world congresses since 1971. The agenda of the VI World Congress of the World Psychiatric Association includes a discussion of the Declaration of Hawaii, which is a code of psychiatric ethics with such statements as "The psychiatrist must never use the possibilities of the profession for maltreatment of individuals or groups" and "The psychiatrist must not participate in compulsory psychiatric treatment in the absence of psychiatric illness."

To support such declarations is not to be anti-Russian. On the contrary, it is to align ourselves with a courageous, patriotic group of Soviet citizens who have reproached their own country for having misused psychiatry to deprive citizens of basic human rights. The dissidents appeal to the same high ideals and re-

spect for human freedom and human dignity that inspired their own twentieth-century revolution and that in earlier centuries inspired England's Magna Carta, the French Revolution, and the Declaration of Independence of the United States. Freedom of the human spirit has had to be defended many times in the past. Today those Russians who, at great personal risk, protest the abuse of psychiatry to suppress dangerous thoughts are in the forefront of this struggle. They deserve our support.

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Referrals Sought for NIMH Program

The Intramural Program of the National Institute of Mental Health is seeking referrals of patients with anxiety states, depression, mania, amphetamine and cocaine addiction, acute and chronic schizophrenia, and tardive dyskinesia; also, anorexia nervosa, Huntington's chorea, presenile dementias (e.g., Alzheimer's and Pick's), Korsakoff's syndrome, Gilles de la Tourette's syndrome, hyperactivity, and enuresis in children.

Patients with these diagnoses are sought to participate in clinical-research studies designed to elucidate the role of biochemical, genetic, familial, and socioenvironmental factors in psychiatric illness and to assess treatment modalities. A goal is the significant integration of knowledge regarding central nervous system functioning in animals with that of human beings, with the hope of increasing its applicability to humans for improved diagnosis and treatment of psychiatric and neurologic disorders.

A physician referral is a prerequisite for admission, and a written medical summary is helpful. Referrals are made through Stanley I. Hirsch, L.C.S.W., Room 3N-250, at NIMH Clinical Center, 9000 Rockville Pike, Bethesda, Md. 20014; telephone (301) 496-5111. Mr. Hirsch will arrange with the appropriate unit to schedule a screening date.

Teaching Psychological Medicine to Family Practice Residents

BY ERWIN R. SMARR, M.D., AND ROBERT BERKOW, M.D.

The authors describe the development of a curriculum focused on teaching residents in family practice how to recognize and deal with the psychological issues presented by patients and by their own reactions to patients. They describe how teachers in a medical school's divisions of family practice and psychosomatic and liaison psychiatry collaborated to develop an integrated training program and the changes this program effected in the attitudes and understanding of residents. They found that residents in this program shifted their emphasis from a narrow view of traditional medical diagnosis and treatment to a broader psychological-biological understanding of patients.

THE RENEWED EMPHASIS on family practice and the development of new training programs in primary care stress a humanistic approach and comprehensive patient care, both of which recognize the need for skills in psychological medicine. The standards for accreditation of family practice residencies require substantial psychiatric training, but neither those standards nor any current body of pedagogic knowledge provides a generally accepted core curriculum. This paper will describe our approach to curriculum development at Hahnemann Medical College and Hospital, our teaching processes, and the changes we have observed in our residents.

In July 1974 the Division of Family Medicine of Hahnemann Medical College approached the Division of Psychosomatic and Liaison Psychiatry with the idea of collaborating to develop an integrated training program. The traditional rotation through psychiatric inpatient services, psychiatric case consultations, and formal lectures had many defects. A new approach, based more on actual family practice, was needed.

Family practitioners need to learn about psychosis, neurosis, and behavior disorders. However, they usually see patients with overt psychopathology at different stages and in different forms and contexts than the psychiatrist or the psychiatric service sees them. Family practitioners often deal with the psychological problems of everyday life, which are often interwoven with a variety of illnesses, rather than with overt psychiatric symptoms.

Psychiatric consultants, on the other hand, respond only to preselected cases of the referring physician. Thus they become the problem solvers rather than the teachers of physicians in the processes of diagnosis, evaluation, and management in physician-patient situations. Psychiatric advice is usually directed to only the most immediate problems and tends to be tailored to basic levels that can be understood and applied with certainty, considering the limitations of the referring physician.

PROGRAM DESCRIPTION

Personnel and Schedules

Although the family medicine division knew what it did not want for its 9 residents, it was much more difficult to define what was needed. Out of the dialogue between the two divisions of family medicine and psychosomatic and liaison psychiatry came a commitment to create a new clinically oriented program of psychosomatic medicine. The program's basic design was that of a preceptorship centered on family medicine patient-physician interactions.

A liaison psychiatrist made inpatient family medicine rounds 2 hours a week with the medical chief and a medical sociologist who was a full-time staff member in family medicine. The outpatient precepting and conferences were conducted by two part-time staff members of the liaison psychiatry division, one of whom (E.R.S.) is a psychiatrist with special interest and experience in general medical practice and the other (R.B.) an internist with subspecialty training in psychosomatic medicine. Each contributed half of the teaching time required. Nine residents were involved in this program in its first year; the number has increased to 17 in the third year, with no change in the number of teachers.

In the outpatient Model Family Practice Unit, all residents were precepted individually during their patient interviews and participated in one of two separate group conferences (each lasting at least 1½ hours) a week; the family medicine faculty and the medical sociologist participated. A total of 12 hours of teaching a week, supplemented by monthly didactic lectures for the full 3 years, was planned. The family medicine di-

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vision, whose faculty consisted of two full-time and two half-time staff members, funded the salaries.

Curriculum and Program Design

Decisions on curriculum content were deliberately not made because of our conviction that as long as the work focused on the problems of residents and patients the appropriate curriculum would emerge. Additional presuppositions that contributed to our basic approach included the following: that it was mandatory to examine the psychological elements observed in each and every patient contact without prejudging whether or not these elements were important; that effective modeling by psychologically sophisticated faculty is a sine qua non of learning by identification; that unlearning of role models developed during medical school would be necessary; and that teaching would be associated with unconscious resistances and biases. The latter problem was to be dealt with through teaching rather than by psychotherapy.

Teaching focused on three broad areas: 1) psychological-biological interactions, i.e., when and how people become diseased and modifications in the course of disease; 2) psychic stress, processes, and pathology in different modes of illness, i.e., somatic, cognitive, emotional, and behavioral; and 3) physician and patient roles, i.e., their separate individual defenses, their interactions, and sociocultural aspects of illness and medical care systems explicitly analyzed, defined, and then modified according to particularized aims rather than stereotyped.

The general philosophical and pedagogic background for this approach to teaching an integrated psychosomatic perspective lies in the earlier work and writing of others (1–5). Our efforts were directed to developing practical applications in nonpsychiatric residency training. The program as a whole was continuously monitored through regular 2–3-hour faculty conferences held every 6–8 weeks as well as through interim informal discussions, which allowed modifications and provided concurrent evaluations of residents, teachers, and the program. Later, an evaluation form became the basis of more objective measurement of each resident and periodic review of the goals and progress of each resident. We estimated that it would take about 3 years for the faculty to delineate and transmit the relevant curriculum and requisite skills and for the residents to genuinely incorporate them into routine practice. After 3 years the residents should be capable of continued growth on their own. However, the total effect could not be known until the postresidency years of practice.

We wish to emphasize that preplanning a curriculum would be contrary to the goals of the program. After the first year and a half a curriculum emerged and substantial changes in attitudes and practices of the residents were apparent. This paper will describe the content that emerged and the important processes observed during outpatient precepting and conferences.

PROGRAM RESULTS

Curriculum Development

In evaluating curriculum development, the initial tabulation of discussion subjects presented by our residents from their experiences with patients yielded almost 100 subjects within less than a year (see appendix 1). The list represents a typical range of psychologically significant outpatient problems seen by family practitioners. Although the two conferences held each week were led by two teachers with different backgrounds, there was about 80% correspondence in content between them in the first year. Content differences between the two conferences were related to the composition of the resident groups, presentations of patients, and differences in background of the teachers. The items enumerated in appendix 1 can be broadly grouped in many ways, and it should be recognized that each subject was usually discussed from several perspectives. For example, in discussing obesity, the etiological psychodynamics of this disorder are important, but evaluation and management must also take cognizance of social factors and the physician's attitudes, reactions, bias, and interviewing techniques.

Among the medical problems per se, the following appeared most often: hypertension, headache, obesity, ulcer, alcoholism, insomnia, death and dying, problems of pregnancy, cancer, and impotence. Because Hahnemann is an urban teaching center, the social problems of poverty, ghetto life, racial and subcultural influences, religious effects, unemployment, and divorce naturally arose.

Although many clinical syndromes and psychopathological processes were discussed in the conferences, an equally large number of subjects dealt with the physician's role, attitudes, and resources. These subjects could possibly have been preplanned and approached in a didactic program, but the advantages of having the topics raised by the residents themselves and providing timely critical discussion in direct response to immediate clinical needs were especially valuable.

Most subjects could not have been taught as effectively didactically or from a curriculum isolated from the residents' experience. Active versus passive, authoritarian versus laissez-faire, medical model versus psychiatric model styles of doctoring; the moral and ethical issues that underlie so many fine points in case management; and unconscious decision making continually emerged as topics for discussion and were dissected in conferences. All aspects of interviewing technique were examined, changing the concept of the interview from a purely medical history taking to a conscious psychodynamic analysis from which appropriate psychotherapeutic management could proceed. The physician's own feelings and reaction patterns could be perceived as influences affecting his/her style and impact in different situations.

Ordinary family practice yielded a full range of clinical psychopathology and ample opportunity to teach

differential diagnosis and management. Developmental psychology, although also taught separately in child psychiatry, came under review in case discussions. Learning techniques of supportive psychotherapy as well as principles affecting referral for more specialized treatment helped residents define their own appropriate psychiatric skill levels and limits.

Teaching Processes

In precepting, several processes were conceptually and operationally important. For example, interviewing techniques were looked at closely (by direct observation and with the aid of tape-recordings), and medical histories were reexamined with a psychiatric focus, sensitizing residents to emotional nuances and hinted-at conflicts that are routinely excluded from attention by the standard medical interview. Our residents were impressed when they discovered what they had been missing. However, unless they were taught how to collect the relevant data themselves, this new awareness led only to frustration. This often required that they become aware of their own prejudices and defensive mechanisms that predetermined not only what they perceived but how they acted.

As the residents learned to gather psychosocially significant data, the preceptors were challenged to help them organize and synthesize them with psychosomatic concepts. This involved the two prevalent types of logic in medical practice: diagnostic reasoning and management (or therapy) planning. We also found it important to implement reasonable conclusions and follow the subsequent events.

A pertinent and important question is just how much is appropriate for nonpsychiatric physicians to learn regarding such specialized aspects of psychology as defenses, transferences, and psychodynamics. These processes came up for discussion quite naturally in the course of teaching how to interview and understand patients and their problems in broader perspective.

As in every group process, resistances to change or to program features were manifest either actively or passively. The group conference format provided the means of bringing these issues into direct discussion.

Resident Responses

Through the group process our residents have become more analytical with themselves and each other. Emphasis has shifted from the narrow traditional medical diagnoses and usual treatments to a much broader psychosocial, biological inquiry in which patients and their problems are viewed as uniquely human organisms responding to stresses with a variety of mechanisms (defenses) to maintain or achieve homeostasis (2).

A formal evaluation form has been designed and will be described in a subsequent paper. Preliminary data indicate that without teaching our residents psychiatry per se they have learned the following: 1) an appreciation that looking at psychosocial data saves time and is effective in dealing with medical problems; 2) how to use spontaneous verbalizations rather than cut them off; 3) to be aware of transferences and countertransferences that affect what patients say and do, the course of the physician-patient relationship, and the outcome of treatment; 4) how to recognize patient defenses and deal with them; 5) to be aware of their own emotional reactions (not due to countertransference) affecting the interview; 6) the psychodynamics of object-relationship conflicts behind such phenomena as somatizations, depressions, and neurotic symptoms; 7) to avoid being caught up in neuroses, manipulations, or acting-out of problems; and 8) to incorporate psychodynamic logic in the diagnosis and management of medical disorders.

The residents have come to use diagnostic procedures and treatment methods differently. They have learned that ordering laboratory tests is part of the communicative process and mutual search and how to use these elements in the diagnostic process to enhance basic trust and confidence in the physician. Instead of seeing tests and prescriptions as purely medical or scientific processes, the residents have come to understand these in the context of the transference. They have also recognized that the quality of the physician-patient relationship affects the treatment result. After first dealing with their own anxiety and defenses, residents have been relieved by being able to interact more naturally. Patient confidence has been established on the basis of genuine respect rather than authoritative awe or seduction. Equally important, the resident has learned to do what is important and necessary to cope with emotional aspects of patients' problems rather than avoiding them out of fear or ignorance.

Program Modifications

The program has changed only slightly since its inception. We have become even more convinced that precepting family practice residents in their usual patient care settings, using the group process methods, and providing new role models for identification are effective methods of teaching. A curriculum suitable to the program needs has evolved spontaneously. The precepting process supplanted formal lectures. In the second year of the program didactic lectures were dropped except as they were requested by the residents. A more structured presentation of the emerging core curriculum may be added at a later date.

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APPENDIX 1
"Curricular" Subjects of Conferences

PHYSICIAN'S AND PATIENT'S ROLES AND PROBLEMS

Self-Image and Role Model

Active versus passive
Authoritarian versus laissez-faire
Medical versus psychiatric

Interviewing Technique

Setting: inpatient, outpatient department, emergency room, etc.
Open-ended versus directive
Crisis, pseudocrisis, limit setting
Data from minimal clues
Brief psychotherapy
Ventilation
Reassurance
Family therapy
Marital counseling
Family practitioner as psychiatrist
Referral and communication

Psychological Issues

Anger, hostility
Anxiety
Guilt
Denial
Seductiveness
Manipulation
Transference
Countertransference

Social-Ethical-Moral Issues

"Union doctor"
Certifier
Malingering
Confidentiality
Placebo
Medical mismanagement
Birth control
Death and dying
Rounds' effects on patients
Abuse of patient's time

PSYCHOLOGICAL-BIOLOGICAL INTERACTIONS

Separation-depression (disease onset)
Obesity
Hypertension
Ulcer
Psychophysiological symptoms
Alcoholism
Insomnia
Death and dying
Pregnancy
Dysphagia, hysterical
Cancer
Impotence

PSYCHIC PROCESSES AND PATHOLOGY

Somatization
Anxiety
Defense mechanisms
Developmental psychology
Child psychiatry
Adolescent development and sex
Denial and pregnancy
Regression
Depression
Conversion
Obsessive-compulsive personality
Obsessive ideation
Religious cults and psychosis
Homosexuality
Sadomasochism
Hypochondriasis
Dependent-hysterical personality
Paranoid processes
Addiction
Schizophrenias
Diagnostic labeling
Borderline conditions

SOCIAL PROBLEMS

Poverty
Urban ghetto
Religion
Referral services and religion
Cultural psychosis
Racial and ethnic stereotypes
Adolescent pregnancy
Contraceptives
Marriage
Divorce
Jehovah's Witnesses and medicine
Child-rearing instruction
Unemployment
Common law marriage
Use of social worker
Maturity/immaturity

RESISTANCES

Scheduling problems
"Patient's resistance"
Behavior modification
Lateness
Silence and passivity
Authoritarianism
Resentment
Didactic teaching versus case presentation
Defenses and learning

PROGRAM ITEMS

Coordinator's role in family medicine
Psychosomatic rotation
Scheduling conflicts
Research
Faculty changes
Role of social worker

Twin Relationships and Depression

BY MARIA PALUSZNY, M.D., MELVIN L. SELZER, M.D., AMIRAM VINOKUR, PH.D., AND LARRY LEWANDOWSKI, M.A.

The authors investigated closeness and other variables measuring depression in 22 identical and 13 fraternal twin pairs. Each twin rated him/herself on a two-part questionnaire; part 1 included questions on demographic characteristics and the twin relationship, and part 2, the Zung Self-Rating Depression Scale, measured depressive symptoms. There was a high degree of agreement across all variables for the total sample, and degrees of depression were comparable to that in the general population. Closeness was found to be inversely correlated with depression. The authors suggest that future studies include other groups (siblings, spouses, etc.) and follow-up data.

TWIN STUDIES frequently focus on the negative aspects of the intertwin bond. Problems with development of self-identity (1), prolonged mutual dependency (2), and intertwin rivalry (3) have been described. In addition, the twin relationship can enhance a preexisting psychiatric disturbance, as has been noted in psychotic twins (4) and in selective mutism (5). Conversely, it appears that the intertwin relationship can serve as a special support during stress (6). Keeping these factors in mind, we were interested in comparing the relationships of a sample of identical and fraternal twins and correlating the data with depressive symptoms in the twin population. We wondered if our data would enable us to ascertain the importance of closeness (and other variables) in the twin relationship. The questions we were interested in include the following:

Does closeness in the twin relationship promote dependency and loss of self-identity and enhance depressive symptoms? Or does the twin serve as an important support object, diminishing the likelihood of depression?

To assess the twin relationships, we compared and contrasted self-rated variables that measured the intrapair relationships and similarities in a twin population; we then compared these variables with depressive symptoms.

Adequate exploration of intertwin relationships and depression would necessitate individual psychiatric interviews to quantify depression (7) and to delineate precisely the intertwin bond, and extensive data gathering would be necessary to obtain a control sample. Because we were unable to do such an involved study at this time, we decided to use self-assessment methods to obtain preliminary data.

The Zung Self-Rating Depression Scale (SDS) is a simple 20-item questionnaire that measures depressive symptoms (8, 9). It has been used frequently in depression research and according to Blumenthal (10) "appears to have the validity, reliability, and replicability so important in epidemiological research." Although the scale's uses are limited and it has been criticized as a diagnostic tool, especially in controlled drug studies (7), the SDS appeared completely adequate for the purpose of our study.

METHOD

Subjects

Our sample consisted of 70 twins (35 pairs); 22 pairs were identical and 13 were fraternal. In previous studies it has been established that twins' perception of zygosity is highly accurate (11). Thus, we accepted the twins' rating of whether they were fraternal or identical without verification by blood typing. All the twins were adults, 18 years or older, and 70% were in the 18–44 age group; 50 were women and 20 were men. Many of the twins were located through twin organizations and others through personal contacts. The questionnaires were either completed in social settings or mailed to the twins. Hence, no strict criteria were adhered to in administering the questionnaire except that the subjects were asked not to compare ratings with their twin.

Questionnaire

The questionnaire consisted of two parts. Part 1 included questions on such demographic characteristics as age, sex, type of twinship, education level, and marital status. These data were used to ensure that our

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sample was representative of the general population on these variables. Each twin also independently rated his/her relationship to his/her twin according to closeness (rated on a 5-point scale from "like part of me" to "distant") and frequency of contact with the twin (rated on a 5-point scale from "few times per week" to "less than once per year").

Similarities to twin were rated on an 11-point scale for appearance, likes and dislikes, hobbies, and feelings about having a twin.

In part 2 the twins rated themselves and their perception of how their twin would rate him/herself on the SDS.

From these data we assessed whether 1) a close relationship to another person (in this case the twin) correlated with lower levels of depression as measured by the SDS, 2) depression in both twins was equally evident, and 3) twins were able to see their twin's symptoms accurately or would tend to project, i.e., see their twin as being as depressed (or nondepressed) as themselves. We also wondered if there was a significant difference between identical and fraternal twins.

RESULTS[1]

Twin Comparisons

Intrapair analysis revealed a strong level of agreement within twin pairs regardless of whether they were identical or fraternal. The one exception was that fraternal twins rated themselves as significantly different in likes and dislikes than their twin partner ($p<.05$).

Despite the within-pair agreement, further comparisons were made to explore possible differences between identical and fraternal twin pairs. Because both the identical and fraternal twin groups are composed of paired observations, it was necessary to take the average rating of each twin pair before making any between-group comparisons. This statistical manipulation offered a less biased measure by creating one observation for every twin pair. Comparisons were then made between 22 observations for identical twins and 13 observations for fraternal twins across six variables.

Significant differences were found between identical and fraternal groups on all but one variable, frequency of contact. On five other variables the identical twin versus the fraternal twin ratings showed that 1) identical twins feel closer to each other ($p<.01$); 2) identical twins like having a twin more than fraternals do ($p<.05$); identical twins are more alike in appearance ($p<.01$); identical twins have more similar interests ($p<.01$); and identical twins tend to like and dislike similar things more than fraternal twins do ($p<.01$).

Furthermore, with the identical twins, a strong correlation was found between closeness and liking having a twin ($r=.8016$, $p<.001$). Thus, it appeared the more the identical twins liked being a twin, the closer they felt to each other. The fraternal twins did not show a significant correlation between closeness and liking being a twin. However, a significant correlation was found between how often the fraternal twins saw each other and similarity of interests ($r=.9590$, $p<.01$). One could speculate that frequency of contact was prompted by sharing activities that both fraternal twins enjoyed rather than being prompted by other variables.

We found no significant difference in a comparison of single versus married twins in terms of feelings of closeness to their twin. Although single twins tended to see their twin slightly more often than married twins, this difference was not significant.

Depressive Symptoms

Interesting data emerged when we correlated the 6 variables with the SDS. The mean SDS score for the total sample was 31.5, very close to the SDS score of 33 that Zung found in his normal control groups (8). In this regard the twins appear similar to a normal population and comparable to the "normal" range as described by Blumenthal (12). No correlation in the twins' perception of their relationship was found between the SDS and similarity in appearance, similarities in likes or dislikes, or frequency of contact. Weak but statistically nonsignificant correlations were found between SDS and similarities of interests (hobbies) and how the twins felt about having a twin. The only significant correlation was between the SDS and the ratings of how close the twins felt to each other ($r=.26$, $p=.03$). Thus, it appears the closer the twins felt to each other, the less likely they were to show depressive symptoms as measured by the SDS.

Evaluation of the similarity of SDS scores between twin pairs revealed an SDS correlation of .70 ($p<.01$). When we divided our sample into fraternal and identical groups, we found that the correlation was much higher for the identical group ($r=.80$, $p<.001$).

We assessed how much the twins tended to project their own depressive symptoms onto their twin by comparing difference scores between twin ratings with a rating for accuracy. This method has been described previously (13). Basically, a difference score was obtained between the twin's own SDS and the SDS the twin filled out for his/her twin. This score was then compared with the difference of the SDS score of the twin and the person's own SDS score. In calculating projection in this manner, we found that the twins saw their twin as equally depressed (or nondepressed) as themselves. However, even though the total sample showed this tendency, we found a major difference when we looked at identical and fraternal twins separately. Identical twins significantly projected their own perceptions of depression ($p=.02$), but projection of depression by the fraternal twins was not significant.

[1] Statistical tables are available from Dr. Paluszny on request.

DISCUSSION

Our sample of 70 twins appeared to be very similar in how they rated themselves and their twins. There was a high degree of agreement across all variables for the total sample. The identical twins, however, liked being a twin more, felt closer to their twin, and rated themselves as more similar to their twin in appearance, interests, likes, and dislikes than the fraternal group. In assessing the negative and positive effects of this closeness, we found closeness to one's twin did not correlate with frequency of contact or with staying single. Thus, closeness did not seem to interfere with the twins separating from each other.

The total sample showed degrees of depression comparable to that found in the general population. The twin pairs, especially the identical twins, showed a similarity in their depressive scores. Likewise, the identical group, more than the fraternal group, tended to "project" their own ratings on the SDS, viewing their twin as similarly depressed (or nondepressed) as themselves.

The degree of depression showed an inverse correlation with closeness, i.e., the closer the twins rated themselves, the less likely they were to show depression. A word of caution is necessary pertaining to this finding. The correlation between feeling close to one's twin and low level of depression might suggest a causal relationship between the two phenomena, i.e., closeness of feeling decreases depression. Such a conclusion, however, is unwarranted at the present time. It is quite possible that the experience of depression in itself leads a person to feel quite distant from those significant others who surround him. This finding, however, does seem to indicate that closeness in twins does not lead to psychiatric problems that become clinically evident as depression.

Future studies to investigate closeness and other pertinent questions regarding depression (including the role of hereditary factors) should include such additional groups as siblings, spouses, and friends and should involve longitudinal (follow-up) data collection.

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Editor's Note: We are aware that there will be criticism about the publication of these papers. The editors are especially concerned about their unevenness. However, in view of the widespread interest in and strong differences of opinion about the subject, we decided to give it a full airing. F.J.B.

Myths of "Shock Therapy"

BY MAX FINK, M.D.

The author discusses the myths of the ECT process—that shock and the convulsion are essential, memory loss and brain damage are inescapable, and little is known of the process—and assesses the fallacies in these ideas. Present views of the ECT process suggest that its mode of action in depression may best be described as a prolonged form of diencephalic stimulation, particularly useful to affect the hypothalamic dysfunctions that characterize depressive illness. The author emphasizes the need for further study of this treatment modality and for self-regulation by the profession.

"SHOCK THERAPY" is a treatment that has been in clinical use since 1937. It is one of many treatments that evolved after the success of fever therapy in relieving CNS syphilis led to a hope that mental illness was not necessarily incurable.[1] Of many similar heroic measures that were tested amid this enthusiasm—insulin shock, camphor, pentylene tetrazol (Metrazole), cardiozole, sleep therapy, lobotomy, and ECT—only ECT continues to be used frequently. Despite improvements in its application, the use and study of ECT are hampered by some widely held beliefs that are often contradictory and confused.

In a lead article in the *British Medical Journal* in 1975 it was stated that "Shock and convulsion, even if the latter is modified almost to the point of invisibility, are of the essence" (1). Also in 1975, a psychiatrist writing in *Psychiatric News* said,

> ECT has been abused. It has been given too often, too broadly, and for whom the treatment was not indicated. . . . The skill that it takes to administer the actual shock to a patient is negligible. It is about as involved as the skill required to take a patient's temperature. True, there are variations in the placement of electrodes on the patient, and there are different ways of administering the current, but none of these techniques require the background of a psychiatrist's intensive training or would take more than a few minutes for *anyone* to master. (2)

An internationally known psychologist, in an interview in the newsletter of the American Psychological Association, said, "I'd rather have a small lobotomy than a series of electroconvulsive shock. . . . I just know what the brain looks like after a series of shocks, and it's not very pleasant to look at" (3). A resident neurologist, attacking the clinical use of ECT in a lay journal, stated that "Electroconvulsive therapy (ECT) . . . is remarkably widespread, demonstrably ineffective, and clearly dangerous. It causes brain damage manifested in such forms as severe and often permanent loss of memory, learning disability, and spatial and temporal disorientation" (4).

Critics assert that "shock" and the convulsion are

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[1] The Nobel Prize for medicine in 1927 was given to Dr. Julius Wagner-Jauregg "for his discovery of the therapeutic value of malaria inoculation in the treatment of dementia paralytica."

essential to the treatment, that memory loss and brain damage are severe and inescapable, that ECT "burns the brain," that its effects are like a Band-Aid to a wound, i.e., are palliative rather than relieving the cause, that so little is known of the mechanism of ECT that it should be discarded, as cupping, burning, leeching, and bleeding have been discarded.

EXAMINING THE MYTHS

The widely held beliefs discussed above are unfounded—they are the myths of electroconvulsive therapy. Neither shock nor convulsion is essential. Memory loss, which does occur, is neither permanent nor essential and can be minimized by treatment modifications so as to be a calculable risk. Brain damage, like death, is a rare event in ECT, and even its low incidence may be reduced further. Much is known about the ECT process—possibly as much or more than is known about any other treatment mode in psychiatry—and there is good evidence that cerebral seizures affect specific pathology, particularly in depressive illness.

Let us examine each ECT myth individually.

1. *"Shock and convulsion are essential":* Shock is defined as the perception of the electrical currents used to induce a seizure or as the evocation of fear, panic, and anxiety that may accompany any stressful experience. Neither type of shock is essential to the ECT process.

In the early days of ECT, seizures were unmodified by sedatives or muscle relaxants. Fear and panic often accompanied such treatments, and their prominence was believed to be essential to the treatment process (5). Today, however, treatment is given to patients who are asleep after receiving an intravenous barbiturate; the patient experiences neither panic nor pain. Seizures may also be induced by an anesthetic inhalant, flurothyl, with no electrical currents, and these treatments are as effective as ECT (6–8).

The convulsion is defined as the motor and autonomic events that accompany the cerebral events—the patterned electrical discharges that are termed the central "seizure." ECT given under complete muscle paralysis following the intravenous administration of succinylcholine is as therapeutically effective as unmodified seizures, which indicates that the motor and autonomic aspects of the convulsion contribute little to the therapeutic process. At one time, electric currents were applied without inducing a seizure. These subconvulsive currents were significantly less effective in treating depressive states and schizophrenia and were soon discarded (9–11). Trials with subconvulsive levels of metrazole and flurothyl also found that clinical efficacy was dependent on the seizure (12).

An interesting aspect of the role of seizures in ECT is seen in the "missed seizure." With threshold currents or unilateral electrode placement, incomplete seizures occasionally occur and are associated with a poor clinical response. Such incomplete seizures occur often enough for various authors to have suggested that EEG monitoring of the seizure is necessary to assure efficacy in ECT (7, 8). These observations are a further indication that electrical currents alone, without the cerebral events, are clinically ineffective.

Not only is the cerebral seizure essential, but its length is related to clinical efficacy (13, 14). When the duration of the seizure is reduced by lidocaine, ECT efficacy is also lessened (15). The present view is that the seizure is either the therapeutic event itself or a direct reflection thereof, and that neither an electric current ("shock") nor a convulsion is central to the therapeutic process.

2. *"Memory loss is permanent, and improvement depends on the degree of memory loss":* Each seizure produces changes in brain functions which persist for minutes to hours after each treatment. With successive seizures, the mental changes persist for longer periods, often for many weeks after a series of treatments. The syndrome is characterized by confusion, memory difficulty (both anterograde and retrograde), disorientation, and changes in psychological and physiological tests (16). These deficits gradually improve after the last seizure. Nevertheless, memory impairment is a frequent complaint, with 18%–67% of patients reporting defects after treatment (17–19). Examination of memory processes by objective tests, however, reveals that performance is usually at pretreatment levels or better within weeks of the last treatment. Long-term follow-up studies have failed to find persistent memory defects after six months (18, 19).

It was once thought that memory loss was central to the therapeutic process, but recent studies suggest otherwise. Depressed patients improve after few seizures, without evidence of memory loss. When electrodes are placed over the nondominant hemisphere, as in unilateral ECT, the therapeutic response is sustained, but memory loss is reduced (20, 21). The type of memory that is affected is related to the path of currents (i.e., location of electrodes), which suggests that the changes in memory functions are largely the direct effects of the current, rather than an effect of the seizure or the accompanying biochemical events (22).

Our beliefs about the role and significance of memory loss in the treatment process are complicated by the many changes in treatment methods and treatment attitudes during the past decades. In the early days of shock therapy, changes in memory were believed to be important to the therapeutic process, and memory impairment was encouraged by allowing the patient to remain apneic and cyanotic until normal breathing recurred after each seizure. Today, sedation, hyperoxygenation, unilateral electrode placement, and control of current characteristics make memory impairment and other features of the organic mental syndrome no longer a factor in the ECT process.

3. *"Shock therapy causes permanent brain dam-*

age": Brain damage is best defined by pathologic study. The data concerning sustained pathologic changes with ECT come from the examination of brain tissues of patients who died after ECT, from epileptic patients who died in status epilepticus, and from tissues of animals subjected to repeated seizures. Inferences are also made from the psychological and physiological tests that are usually interpreted as measures of an organic mental syndrome in man (16).

The definition of the pathology of ECT is complicated by the time between ECT and the time of death in published reports. Some tissues were examined after seizures that were within hours of death, but in other reports, ECT is a distant event that may or may not be related to the pathology examination. Brain tissues have been reported to show increased gliosis (23), diffuse degeneration (24), petechial hemorrhages in the brain stem with fat embolism (25), and edema and subarachnoid hemorrhage (26–28). The brain of a patient who died 15 minutes after the 12th ECT was found to be edematous and showed neuronal damage and increased lipofuscin pigmentation (29). In four autopsies of patients who died after ECT, one author reported death due to intraventricular hemorrhage in one and to cardiovascular disease in three (30). Each of these studies was done before 1956, when seizures were usually induced without succinylcholine, oxygen, or barbiturates.

The incidence of death in ECT has been reported variously from none in 8,500 treatments in 870 patients (31) to .06%, .08%, .03%, and .8% of patients treated (32–35). In most instances, death is ascribed to cardiac complications, usually after the seizure during the recovery period and rarely during the seizure itself. Higher death rates were reported for the 1940s, when treatments were usually given without drug modifications and "missed" or incomplete seizures were more common. Patients frequently experienced pain, fear, and panic—psychological events that can contribute to a cardiac death (36). In the pathology reports, missed seizures were a prelude to a number of the deaths studied. Missed seizures and subsequent panic, fear, and excitement are no longer a feature of clinical ECT. Pretreatment sedation and anesthesia are routine and probably contribute to the reduction in the number of deaths occurring with ECT.

It is difficult to negate the global judgment that ECT causes permanent brain damage. Except for persistent subjective complaints of memory defect, however, the evidence for such injury or its persistence is lacking, despite extensive study.

4. *"ECT is at best palliative; its efficacy is not equal to other therapies":* Among the beliefs with little foundation, the most pervasive is the professional myth that psychotherapy gets at the root of the patient's problems, removes the cause of the illness, and thereby prevents recurrence, while ECT and drug therapies suppress or bury the cause, so that the illness will recur. In fact, comparisons of the results of ECT with other therapies show it to be superior to drug therapies and psychotherapy in both immediate and long-term effects in depressive illness and at least equal to drug therapies, milieu therapy, and psychotherapy in schizophrenia (31, 35, 37).

The results of ECT are directly related to clinical diagnosis (31). ECT is most effective in psychotic depressive syndromes and mania; success rates are 60%–90% of cases selected. Patients with diagnoses of bipolar depression, unipolar depression, involutional depression, and depression of old age are rapidly responsive, usually requiring 2–4 seizures spaced at 48-hour intervals to elicit an elevation of mood, relief of suicidal preoccupation and agitation, and reversal of the symptoms of hypothalamic dysfunction. In most instances, the "course" is completed with 6 to 8 seizures, a number that seems constant regardless of whether seizures are induced electrically using bilateral or unilateral electrode placements (20, 21) or by flurothyl (8). Maintenance seizures are rarely needed, although subsequent treatment with antidepressant drugs sustains improvement better than the use of sedative drugs (38) or no maintenance drugs (39, 40). ECT is effective compared with placebo (41–43), psychotherapy (44), simulated ECT or subconvulsive treatments (10, 11, 45), and antidepressant drugs (41, 46–54).

The data regarding suicide are impressive. A shorter hospital stay and a lower suicide rate for ECT-treated patients compared with those treated by psychotherapy alone were reported in one early study (44). Another study reported 9 deaths in 109 patients treated by psychotherapy alone compared with 1 death in 88 patients treated with ECT (55).

In a 3-year follow-up study of 519 depressed patients treated by ECT, antidepressants, or neither in the decade 1959–1969, Avery and Winokur (56) reported a lower fatality rate for the ECT-treated patients than the other two groups.

The efficacy of ECT also is seen in the comparisons of different methods of inducing the seizure. Studies comparing ECT with unilateral and bilateral electrode placements find the two methods have essentially similar efficacy (20, 21), as do comparisons of seizures induced electrically with those induced by an inhalant, flurothyl (8). The efficacy of repeated seizures is related more to the clinical diagnosis than to the type of seizure induction.

The usefulness of ECT in the treatment of schizophrenia is cited in standard psychiatric texts (35, 37). Controlled studies, however, find little difference between ECT and psychotropic drugs, milieu therapy, or insulin coma therapy in type of outcome, duration of hospitalization or illness, or in incidence of recurrence (31, 57).

In 9 of 10 open clinical studies of acute schizophrenics, an average of 75% improvement in clinical ratings and in discharge rate has been reported, with one study reporting a 30% rate (31). Few studies are controlled. Twelve studies used a historical control, comparing treatment results with those of treatments be-

fore ECT was introduced. In 10 of these reports, ECT was found to be superior, with a 50%–70% improvement rate contrasted with a 30% rate for the earlier period, but 2 reports found no advantage for ECT (31,56). Other studies compared ECT and psychotropic drugs and found similar discharge rates (31, 56, 58).

In chronic schizophrenia, clinical efficacy is less well defined. In 4 controlled clinical trials, no differences were reported between ECT and 1) anesthesia alone, 2) ECT with anesthesia, or 3) subconvulsive currents (59); between ECT and simulated ECT (60); between ECT alone and ECT with succinylcholine (Anectine), ECT with sodium thiopentol (Pentothal), sodium thiopental alone, or nitrous oxide (61); or between ECT and milieu therapy, psychotherapy alone, or psychotherapy combined with psychotropic drugs (62). In a follow-up of the last mentioned study, May and Tuma indicated that the long-term results with ECT were equal to, and often better than, the other therapies (63). These evaluations of ECT in chronic schizophrenia are seriously flawed, however, by inadequate dosage. Despite the clinical experience that schizophrenic patients require an average of 20 seizures for clinical efficacy, each of these controlled studies used fewer than 20 treatments. In some early reports (64) and in more recent assessments, intensive ECT has been reported to elicit better clinical effects than drug therapy alone as measured in work record, number and duration of rehospitalizations, and need for further treatment, suggesting that dosage may be a critical factor in the results of chronic schizophrenia (65).

Part of the confusion in evaluating efficacy results from the paucity of well-designed studies and the complexity of the outcome criteria. The principal evaluations of ECT were done between 1940 and 1960, when mental hospitals were in the transition from closed asylums to open community hospitals and the science of control studies and independent evaluations was yet to be developed.

Every new therapy is broadly applied initially; later its role is refined to a limited number of mental conditions. For ECT, the claim that its use in depression is purely palliative and less effective than other therapies is contrary to the published data. For other psychiatric states, where its efficacy is poorly defined, ECT may or may not be as effective as other therapies, and there is a need for further assessments of its efficacy and safety.

5. *"The mode of action of ECT is unknown; hence, it should not be used":* There are two aspects to this belief—that ECT's mode of action is unknown, and that this is a reasonable basis for it to be discarded.

If a lack of understanding of the mode of action were a sufficient basis for the withdrawal of a medical therapy, we would have to discard not only ECT but all therapies used in psychiatry, including psychotherapy, the drug therapies, and behavior therapy.

But our knowledge of ECT is not so meager, and we know much about its mode of action (66). It is well defined that the seizure—the cerebral and biochemical events that accompany and follow the fit—is essential to a therapeutic result, whereas the convulsion itself or the mode of induction of the seizure contributes little to the treatment's efficacy.

Oxygenation, sedation, and muscle paralyzing agents reduce the complications of ECT without significantly altering its efficacy. Consideration of current strength, frequency of seizures, and electrode location reduce side effects without significantly affecting efficacy. Changes in memory, the organic psychotic reaction, and fear bear no direct relation to the treatment process.

We also know that the best predictor of the response to ECT is individual psychopathology. The efficacy of ECT is most clearly defined in depressive psychoses. Depressed patients require the fewest seizures (an average of 4–7) to achieve a reduction in both their psychosis and in the symptoms of hypothalamic dysfunction that frequently accompany depression. In acute and chronic schizophrenia and in mania, more seizures (an average of 15–30), often with greater frequency, are necessary to obtain clinical benefit. The response to ECT in such patients is often associated with an organic mental syndrome.

Changes in language, memory, EEG, and performance on a wide range of psychological tests accompany each seizure and persist for longer periods with each additional treatment. These changes gradually revert to pretreatment levels within weeks of the last seizure. While the measured changes are features of the seizure process, they are associated with, but not causally related to, the treatment outcome.

Extensive biochemical events accompany and follow the seizure. Almost any index in blood, CSF, tissues, or urine that can change with stress changes with each seizure. How is one to define the *significant* biochemical events? One suggestion is to focus on those changes which have a time course and intensity that parallel the behavioral events (67).

Such events may include persistent increase in catecholamines, reflected best in the increase in brain and CSF monoamine oxidase (68). Cholinesterase levels increase, suggesting that acetylcholine may be a factor in ECT (69, 70). Protein synthesis is inhibited, and blood constituents show the changes of the stress response, which are probably nonspecific with regard to the ECT process (71, 72). Hypothalamic functions, which were abnormal during the illness, show a return to normal levels: sleep, appetite, and libido improve; weight increases; and menstruation returns. Few chemical measures of hypothalamic function have been taken, but the diurnal curve of cortisol excretion was found to be flat during depression and to be more like a normal diurnal curve after an ECT-induced recovery (73).

There is a similarity between the status of ECT today and that of psychotropic drugs in 1957–1962, a period when scientists were finding that the distinctions among the antipsychotic drugs could be re-

lated not to the chemical structures of the compounds but to clinical diagnosis and physiological response. The diverse actions of thymoleptics, stimulants, MAO inhibitors, lithium, and antipsychotic drugs in the treatment of depressions and psychoses do not allow a unitary theory of their mode of action; a pluralistic view is required. The ECT process, while superficially similar in different cases, should also be viewed as being different for different psychopathologies. In schizophrenia, in contrast to depression, the prevailing data do not allow a biochemical formulation but only a psychologic one, in which organic mental changes play a central role that is as yet poorly defined.

SUMMARY AND CONCLUSIONS

At this juncture we can review the argument. From a group of heroic therapies developed in the 1930s, many have been discarded and one, ECT, remains clinically useful. It has undergone many modifications, so much so that the treatment today is significantly different from that first introduced more than 40 years ago. However, misconceptions about the process, its efficacy and safety, and its mode of action, persist (74). The myths and attitudes have contributed to the legal restrictions in the use of ECT, to its infrequent use in public institutions, and to a decreased interest among researchers. Perhaps the psychiatric profession should be grateful to the California legislature and that state's young activists, and to the criticism of Friedberg (4), Pribram (3), and Viscott (2), who have alerted our profession to the need for a reassessment of ECT.

We cannot ignore allegations of abuse of ECT, for we are committed to a policy of self-discipline that can operate only if each allegation is investigated and corrected. Medicine has failed to use its inherent powers of self-discipline to police its practitioners and its institutions, and the present malpractice crisis is the result. The recent effort by the Massachusetts Department of Mental Health to set ECT guidelines and its annual review of the use of ECT is one approach (75, 76). Standards for ECT practice must be set by the profession, and each allegation should be investigated promptly. Where abuse is found, reeducation or censorship should be implemented.

Relinquishing our myths and changing our clinical attitudes toward ECT can only improve the present unrest and common allegations of abuse. By so doing, we can reduce the call for legal proscriptions and encourage more controlled clinical and basic research into this interesting and useful method of psychiatric therapy.

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ECT and Memory Loss

BY LARRY R. SQUIRE, PH.D.

The author reviews several studies that clarify the nature of the memory loss associated with ECT. Bilateral ECT produced greater anterograde memory loss than right unilateral ECT and more extensive retrograde amnesia than unilateral ECT. Reactivating memories just before ECT did not produce amnesia. Capacity for new learning recovered substantially by several months after ECT, but memory complaints were common in individuals who had received bilateral ECT. Other things being equal, right unilateral ECT seems preferable to bilateral ECT because the risks to memory associated with unilateral ECT are smaller.

MEMORY LOSS has long been recognized as a prominent side effect of ECT. For a decade or two after the introduction of ECT, loss of memory was often believed to contribute to ECT's therapeutic effect (1). Today the view is considerably different. Several investigators have demonstrated that the extent of memory impairment is not correlated with clinical improvement (2–4). In addition, although right unilateral ECT (5) results in markedly less memory impairment than conventional bilateral ECT (6–9), it is clinically as effective, or nearly as effective, as bilateral ECT (10). Thus the available evidence supports the contention that memory loss is an undesirable side effect of ECT that is not related to therapeutic efficacy.

Like the organic amnesias that result from head trauma (11), Korsakoff psychosis (12), diencephalic tumor (13), and temporal lobe dysfunction (14), the amnesia associated with ECT is both anterograde and retrograde. Several general reviews of ECT's amnesic effects are available (15–17). In this paper I will describe recent studies that further clarify the nature and extent of memory impairment.

The first study compared the anterograde amnesic effects of bilateral ECT with the effects of right unilateral ECT. The second described retrograde effects of ECT on remote memory. The third reported findings involving the reinstatement procedure. (Results with this procedure in animal studies suggest that learned material not ordinarily affected by electroconvulsive shock may be forgotten if a reminder of the material is presented just before the shock is administered (18–20). These reports raise the possibility that eliciting depressive ideation just before administering ECT could be therapeutically advantageous because ECT might produce amnesia for such ideation.) The final study considered memory capacity in terms of objective and subjective estimates of ability many months after ECT.

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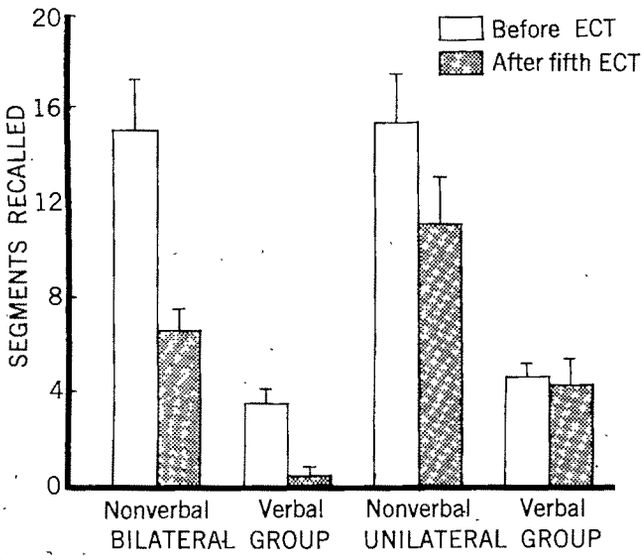
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ANTEROGRADE AMNESIA FOLLOWING BILATERAL OR RIGHT UNILATERAL ECT

It has been demonstrated repeatedly that bilateral ECT produces a greater impairment of new learning capacity than right unilateral ECT (6–9). In virtually all of these studies, however, learning ability has been assessed with verbal memory tests that are particularly sensitive to dysfunction of the left cerebral hemisphere. It is therefore possible that if memory were assessed with nonverbal tests designed to detect dysfunction of the right hemisphere the amnesic effect of right unilateral ECT might be similar to or greater than the amnesic effect of bilateral ECT. In two studies of patients receiving bilateral or unilateral ECT (6, 21), impairment of nonverbal memory associated with bilateral ECT was found to be slightly greater than the impairment associated with right unilateral ECT. However, in the absence of information about how patients with identified unilateral cerebral lesions perform on these nonverbal memory tests, it is difficult to be sure how specifically sensitive they were to right unilateral dysfunction.

My associates and I have recently assessed verbal and nonverbal memory before and after ECT in patients receiving bilateral or right unilateral treatment. To assess verbal memory, patients were read a short story and then immediately asked to recall as much of it as possible. Delayed recall was tested again 16–19 hours later. Patients with identified dysfunction of the left temporal lobe are known to perform more poorly on this test than patients with similar dysfunction of the frontal, parietal, or right temporal regions (22). To assess nonverbal memory, patients

FIGURE 1
Delayed Retention Scores on Verbal and Nonverbal Memory Tests for Patients Receiving Bilateral ECT (N=15) or Right Unilateral ECT (N=9).



were asked to copy a complex geometric design (23, 24). Without forewarning the same patients were asked to copy the design from memory 16–19 hours later. Patients with right temporal lesions are known to be deficient on this memory task, but patients with left temporal lesions exhibit no impairment (25). Tests were administered 1–2 days before ECT and again with equivalent forms 6–10 hours after the fifth treatment of the series.

Figure 1 shows that before ECT patients in the bilateral group and the unilateral group were nearly identical in delayed recall of the story and in delayed reproduction of the geometric figure. After ECT, patients in the bilateral group showed greater impairment in both verbal and nonverbal memory than patients in the unilateral group. Delayed recall of the story was significantly impaired in the bilateral group ($p<.01$) but not in the unilateral group ($p>.3$). (Differences between and within groups were assessed with t tests.) The scores of the bilateral and unilateral groups were significantly different ($p<.01$). Delayed reproduction of the geometric figure was significantly impaired by bilateral ECT ($p<.01$), and the difference between the scores of bilateral and unilateral groups was just short of significance ($p<.09$).

It has sometimes been assumed that right unilateral ECT causes as much memory impairment as bilateral ECT in aspects of memory function identified with the right hemisphere. Our present results clearly indicate that bilateral ECT impairs memory to a greater extent than right unilateral ECT, regardless of whether the tests used to assess memory are more sensitive to left or right hemispheric dysfunction.

This finding may mean that after unilateral ECT the unaffected hemisphere can always contribute to some extent to performance. This idea is supported by the observation that bilateral medial temporal surgery affects both verbal and nonverbal memory to a greater extent than unilateral temporal surgery (25).

RETROGRADE AMNESIA FOR REMOTE EVENTS

It has been reported that convulsive therapy can cause retrograde amnesia for events that occurred close to the time of treatment (15–17). It is now clear that retrograde amnesia can also extend to events that occurred many years before treatment (8, 26, 27). My associates and I have recently assessed remote memory with objective tests that ask about relatively familiar past events. In one test patients were asked to recognize the names of television programs that were broadcast for a single season from 1957 to 1972 (28). The programs, selected from different time periods, were exposed to national audiences to about the same extent, and memory for these programs was acquired close to the time the programs were on the air. Patients who had had a course of bilateral ECT took one form of this test before ECT and another form 1 hour after the fifth ECT.

ECT caused a temporal gradient of impairment in long-term memory (8, 29). Programs broadcast 1–3 years before ECT were forgotten; programs broadcast 4–17 years before ECT were remembered as well after ECT as they were before. Further work indicated that right unilateral ECT caused no deficit in remote memory as measured by this test. Finally, the memory loss associated with bilateral ECT was largely recovered 1–2 weeks after the completion of treatment.

We have also confirmed the clinical impression that in amnesic patients memory for temporal order is more impaired than other aspects of memory (27). Patients were shown sets of three names of television programs and were asked to choose which program was broadcast most recently. In each set of three the correct program name had been broadcast for one season from 1962 to 1973 and the two incorrect program names had been broadcast from 1957 to 1968. After five bilateral treatments, patients developed a marked impairment in their ability to make temporal judgments about this material. The impairment was temporally graded, extending to events that occurred 4–7 years before treatment but not to events that occurred 8–16 years before treatment. The deficit for temporal order information was more persistent than the deficit for recognition of program names and remained unchanged 1–2 weeks after the completion of treatment. Further work is needed to determine how long this deficit remains.

These results indicate clearly that the amnesia associated with bilateral ECT affects not only recent events but also events that occurred many years before treatment. They also indicate that bilateral ECT produces greater retrograde amnesia than right unilateral ECT. Right unilateral ECT caused no measurable loss of memory for remote events; in contrast, a stan-

dard course of bilateral ECT led to an impairment in memory for remote events that persisted for at least 2 weeks.

REINSTATEMENT

Normally, the severity of retrograde amnesia is inversely related to the time interval between learning and amnesic treatment. However, several animal studies have suggested that material not ordinarily affected by convulsive stimulation may be forgotten if a reminder is presented just before treatment (18–20).

To assess the reinstatement phenomenon with human subjects my associates and I conducted a study in which inpatients receiving bilateral ECT learned material 18 hours before ECT or about 10 minutes before ECT (30). Alternatively, patients learned material 18 hours before ECT and then were given a reminder a few minutes before ECT. Retention was tested 6–10 hours after ECT. The upper portion of figure 2 shows the results for a 32-item recognition task; the lower portion of the figure shows the results for 18 paired associates. Patients who learned material 18 hours before ECT consistently showed better retention than patients who learned material only a few minutes before ECT ($p<.05$ by t test). Patients given a reminder just before ECT of material learned 18 hours before retained this material as well as or better than patients not given a reminder. Thus, recalling material from memory just before ECT did not produce amnesia. If anything, the reminder procedure improved retention.

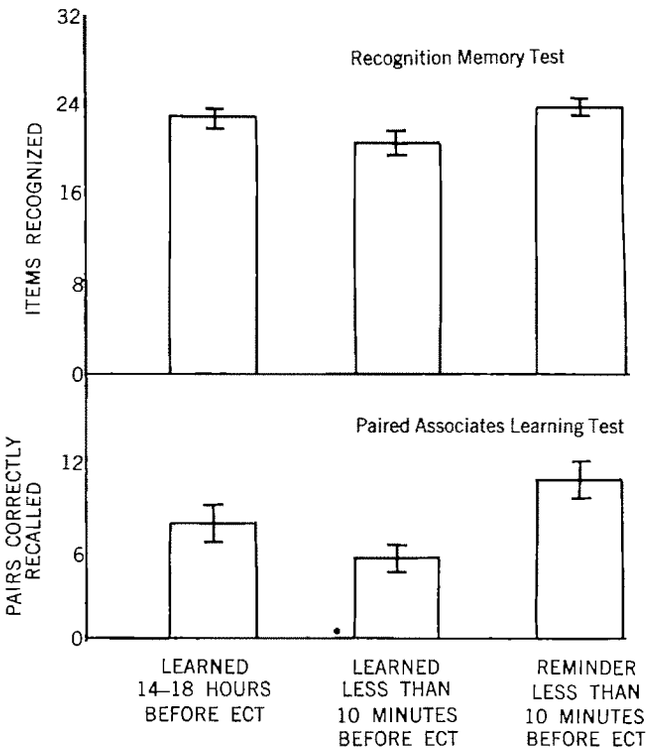
These results cannot rule out the possibility that amnesia might have occurred if the interval between the reminder and ECT had been shorter than the 3 minutes required for administration of medication. Nevertheless, it is clear that amnesia need not occur even when a reminder is given at a time before ECT when memory for newly learned material is disrupted. This finding is of clinical interest because of the possibility that the reminder procedure might be used advantageously with depressed psychiatric patients to improve the effectiveness of ECT. The results of this study provide no evidence that such a procedure would be effective in a clinical population.

LONG-TERM EFFECTS OF ECT ON MEMORY

My associates and I have conducted a long-term follow-up study of patients who had received bilateral ECT, right unilateral ECT, or hospitalization without ECT 6–9 months previously (31). Memory functions were assessed with five different tests of new learning and a test of remote memory capacity; self-ratings of memory functions were also obtained from all subjects. A group of inpatients who at the time of testing were receiving a course of bilateral ECT was also included.

The three follow-up groups did not differ from each

FIGURE 2
Retention Scores for 12 Patients on a Recognition Memory Test and a Paired Associates Learning Test

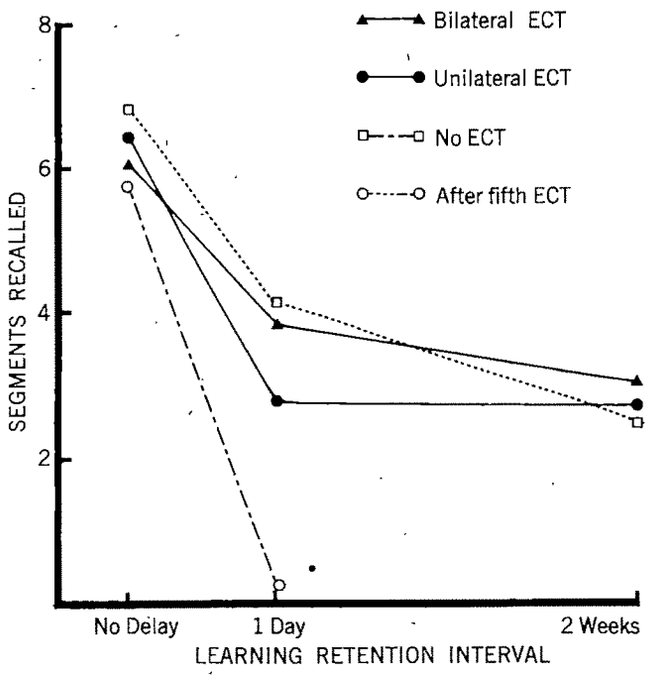


other on any of the memory tests. However, the group tested a few hours after the fifth bilateral ECT was impaired on all of the memory tests. Figure 3 shows the results for one of the memory tests. As might be expected, the inpatients had more memory impairment than the other groups. There was no significant difference between the retention scores of the three follow-up groups ($p>.3$ by analysis of variance).

Although no objective evidence was obtained in this study for persisting memory impairment long after ECT, we found in this relatively small sample ($N=31$) that subjects who had received bilateral ECT reported frequently that their memory was not as good as it used to be (31). Further study of memory complaints involving a larger sample of subjects who had received bilateral ECT or right unilateral ECT 6–9 months before ($N=70$) indicated that of the 55 subjects who had received bilateral ECT (mean number of treatments=9.9), 37 (67%) indicated that their memory was not as good as it used to be. By contrast, of 15 subjects who had received right unilateral ECT (mean number of treatments=9.4), 4 (27%) felt that their memory was impaired. (Individuals with memory complaints related only to the period of hospitalization were not scored as having perceived memory impairment.)

Such asymmetry in the distribution of memory complaints of bilateral and right unilateral groups could have occurred by chance less than 1 in 50 times (by chi square test). Most of the subjects with complaints felt that ECT was the cause of their memory problems.

FIGURE 3
Memory Test Scores for Patients Tested 6–9 Months After Bilateral ECT (N=16), Right Unilateral ECT (N=10), and No ECT (N=12) and for Inpatients Tested 6–10 Hours After Their Fifth Bilateral ECT (N=15)



Eleven of the 37 subjects who had complaints after bilateral ECT selected from four statements the one they felt best described their circumstances. None felt that they had "severe memory problems that interfere with almost everything I do"; 2 felt that they had "many memory problems that are disturbing and that occur frequently"; 6 felt that they had "minor memory problems that occur frequently"; and 3 indicated that they had "only an occasional minor problem."

Unfortunately, the discrepancy between subjective and objective measures of memory function cannot be conclusively resolved at this time. For example, the possibility cannot be ruled out that failures of recall persist after ECT that are not detected by conventional memory tests. We also cannot rule out the possibility that patients receiving bilateral ECT were different from patients receiving unilateral ECT in some way that favored the development of memory complaints. For example, patients receiving bilateral ECT might have initially been more depressed than patients receiving unilateral ECT, or they might initially have had different expectations about memory impairment. Thus it should not be concluded that bilateral ECT causes persistent memory complaints in any depressed patient. However, it seems quite clear that individuals judged clinically appropriate for bilateral ECT do have memory complaints long after ECT.

One possibility that must be considered is that bilateral ECT itself might lead to a lingering sense of memory impairment. Thus the marked impairment of recent and remote memory initially associated with bilateral ECT might cause some individuals to be more sensitive to subsequent failures in recall, even if they occur at a normal frequency. According to this hypothesis, unilateral ECT, which causes less memory impairment than bilateral ECT, would not be expected to lead to memory complaints. In its strongest form, this explanation of memory complaints supposes that bilateral ECT might lead many individuals (with or without psychiatric illness) to have a persistent illusion of memory impairment. It is too early to accept or reject any of the hypotheses outlined here; further work is needed before it will be possible to choose among them.

CONCLUSIONS

The findings reviewed above lead to the following general conclusions about ECT and memory loss: 1) bilateral ECT is associated with greater anterograde amnesia than right unilateral ECT, even when memory is assessed with tests known to be particularly sensitive to dysfunction of the right cerebral hemisphere; 2) bilateral ECT also produces more extensive retrograde amnesia for remote events than right unilateral ECT; 3) the activation just before ECT of previously learned material does not cause amnesia for that material; 4) memory substantially recovers 6–9 months after the completion of bilateral or right unilateral ECT, but persisting memory complaints are common in individuals who receive bilateral treatment.

Many different opinions have been expressed about ECT and about the memory loss that accompanies it. In view of the facts presented here, together with the considerable body of information available about ECT, opinions and clinical impressions no longer seem sufficient in most discussions about memory loss and ECT. The experimental findings reported here summarize what is now known about the risks of ECT to memory functions. All other things being equal, right unilateral ECT is preferable to bilateral ECT because the risks to memory associated with unilateral treatment are smaller than the risks associated with bilateral treatment. These risks should be considered together with the benefits of ECT to provide a basis for clinical judgment.

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Efficacy of ECT in Affective and Schizophrenic Illness

BY MILTON GREENBLATT, M.D.

The author reports on studies of the comparative efficacy of ECT, the newer psychotropic drugs, and combinations of both in the treatment of depression and schizophrenia. He concludes that ECT is indicated for acutely suicidal and other severely impaired depressive patients but not necessarily for schizophrenic patients, although ECT has been successful with some schizophrenic patients for whom drugs were ineffective.

THE THERAPEUTIC VALUE of convulsive therapy has been recognized for 40 years. My personal recollections go back to 1939, shortly after the introduction of pentylenetetrazol (Metrazol), when as a medical student I was allowed to inject the drug into chronically ill patients at Worcester State Hospital in Massachusetts against their terrified and frightened resistance, which, I might add, was overpowered by several burly attendants. In those days we required only the approval of the patient's next of kin for this procedure; we had few qualms about proceeding against the patient's physical

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resistance. Those who now regard ECT as barbaric will be interested to learn that when ECT replaced pentylenetetrazol, it was hailed as a great humanistic triumph because the patient no longer suffered the awful preseizure sensations that accompanied the pentylenetetrazol injection; furthermore, patients given ECT were fortunate to have a period of amnesia after the treatment.

Such gratifying modifications in technique as the reduction, rectification, and commutation of current soon followed the introduction of ECT. These modifications made it possible to induce a seizure by the least jolt, so to speak. Preconvulsive barbiturate anesthesia, suppression of motor response by succinylcholine chloride (Anectine), and positive pressure oxygenation to ease the return to respiratory equilibrium were also introduced. More recently, unilateral nondominant ECT has been touted as greatly mitigating the organic features of the post-shock period (1, 2), allowing more treatments to be given in a shorter period of time.

We always regarded it as unfortunate that the terms "shock treatment" and "convulsive treatment" have hung on because they are still frightening to patients and their families. Even the term "electrotherapy" conjures up bad images in anxious patients. Nevertheless, ECT has dominated the treatment scene for many years; indeed, for a long time it was the only dramatically effective therapy in the psychiatric armamentarium. Thousands of patients thought to be doomed to long months of suffering from depression, involutional psychosis, or schizo-affective disorders were granted a new lease on life through ECT. The treatment not only helped patients but also was an unquestioned boon to psychiatry as a profession; psychiatry suddenly began to earn a little respect in medical circles and from the general public.

The efficacy of ECT has been the subject of two reports of the Group for the Advancement of Psychiatry, one in 1947 (3) and one in 1950 (4). These reports stated that although no adequate theory explained the mode of action of ECT, the treatment materially shortened the duration of psychotic depression, involutional depression, and depression associated with organic brain disease and was beneficial for some patients with neurotic depression. ECT was recognized as effective in the manic phase of manic-depressive illness, in schizophrenia with affective disorder, and in organic psychosis with excitement (e.g., catatonia), but it was contraindicated for patients with psychosomatic disease, psychopathic personality, behavior disorders of childhood, and neuroses other than neurotic depression. The Frankel Task Force reaffirmed this in 1973 (5).

Studies in the literature suggest that best results from ECT occur in patients with a family history of depression who manifest psychomotor retardation, early morning awakening, weight loss, and guilt. In other words, the treatment is effective for patients with classical clinical features of endogenous depression (1, 6, 7). When ECT is used in schizophrenic patients, more favorable results occur if the illness is of short duration, features major affective elements, has an identified precipitating event, and involves a well-adjusted premorbid personality (5).

TABLE 1
Minimum and Maximum Dosages of Various Drugs and Number of ECT Treatments Used In 8 Weeks

Item	Minimum Dose or Number of Treatments	Maximum Dose or Number of Treatments
Isocarboxazid	40 mg	60 mg
Imipramine	200 mg	250 mg
Phenelzine	15 mg	60 mg
Amitriptyline	50 mg	150 mg
ECT	9 treatments (3 per week)	Left to discretion of project physician

COMPARATIVE EFFECTIVENESS OF ECT AND DRUGS IN DEPRESSION

With the advent of the newer psychotropic drugs came the question of the comparative effectiveness of ECT versus the tricyclics and monoamine oxidase inhibitors. This has been dealt with extensively in two major studies, one by my associates and me at the laboratory of the Massachusetts Mental Health Center in 1959–1966 (8–10) and one from a collaborative study in England in 1965 (11). Taken together, these studies investigated the comparative efficacy of somatic therapies for more than 600 hospitalized depressed patients and obtained essentially the same findings.

The American Study

Our study involved 352 depressed patients admitted to three state hospitals in the Commonwealth of Massachusetts. The patients had the following diagnoses: 27.0% had manic-depressive depression (N=95); 19.3% had involutional depression (N=68); 8.2% had psychotic depressive reaction (N=29); 18.2% had psychoneurotic depressive reaction (N=64); 7.7% had depression with other psychopathology (N=27); and 19.6% had schizo-affective depression (N=69). These patients were randomly treated with either ECT, imipramine (Tofranil), amitriptyline (Elavil), phenelzine (Nardil), isocarboxazid (Marplan), or placebo. Imipramine and amitriptyline are tricyclic compounds; phenelzine and isocarboxazid are monoamine oxidase inhibitors.

The dosages used over an 8-week treatment period are shown in table 1. The schedule of observational evaluations of patients is shown in table 2. After the 8-week treatment period our results showed considerable differences in improvement in patients given the various treatments. Table 3 shows the percent of patients who showed marked or moderate improvement in each group, and table 4 shows the results of

TABLE 2
Schedule of Observational Evaluations of Patients

Measure	Time of Observation	Staff Member Who Collected Data
Depression Rating Scale (12)	Before treatment and weekly	Hospital physician and team physicians
Ward observation inventory	Before treatment and weekly	Ward nurses
Mental status symptom checklist	Before treatment and at discharge	Hospital physician and team physicians
Family and social background inventory	Completed by third week of hospital stay	Project social worker
Clinical side effects protocol	Before treatment and weekly	Hospital physician
Assessment of recovery	At discharge	Hospital physician
Follow-up interview	Annually	Project social worker

TABLE 3
Patients Who Improved Markedly or Moderately with Various Treatments (N=352), in Percents

Treatment	Patients Who Improved
ECT	92.9
Phenelzine	75.0
Imipramine	74.4
Amitriptyline	71.9
Placebo	64.2
Isocarboxazid	55.8

TABLE 4
Statistical Analyses of Differences Between Patients with Marked Improvement and Those with Moderate or No Improvement

Comparison	Chi square	Significance
ECT versus isocarboxazid	35.55	$p<.001$
ECT versus placebo	20.87	$p<.001$
ECT versus imipramine	13.00	$p<.001$
ECT versus amitriptyline	10.60	$p<.01$
ECT versus phenelzine	9.15	$p<.01$
Imipramine versus isocarboxazid	7.38	$p<.01$
Phenelzine versus isocarboxazid	6.97	$p<.01$

statistical analyses of the differences between patients who showed marked improvement and those who showed only moderate improvement or no improvement at all. These analyses clearly reveal that ECT was significantly superior to isocarboxazid, placebo, imipramine, amitriptyline, and phenelzine.

In following the status of patients over time we noted that ECT produced the fastest amelioration of symptoms; imipramine was next. The other treatments—isocarboxazid, phenelzine, and placebo—seemed to show less satisfactory results on a time scale. The greater efficacy of ECT was clearly obvious at 3 weeks (10, p. 585).

Other findings of our study as well as their implications for somatic therapy include the following:

1. ECT is indicated for acutely suicidal patients; improvement can be expected after 3–6 treatments.
2. Imipramine or phenelzine is indicated for patients who are not acutely suicidal; if no improvement occurs after 3 weeks, ECT is indicated.
3. Imipramine or a monoamine oxidase inhibitor may be effective for minimally impaired patients.
4. All of the medications, placebo, and ECT produced side effects.
5. If the patient shows no response to medication in 2–3 weeks, increasing the dose is usually unavailing.
6. Antidepressant medication may increase nondepressed schizoid symptoms in schizo-affective patients.
7. The fact that some patients responded positively to placebo points to the value of a supportive milieu.
8. ECT produced the most marked improvement in severely impaired patients. Next in effectiveness were imipramine and amitriptyline. Phenelzine and isocarboxazid were the least effective.

The British Study

The results of a short-term (4-week) British study of 250 depressed patients comparing ECT with imipramine, phenelzine, and placebo (11) were quite similar to our findings in the United States. They confirm that for the most severely ill depressed patients ECT was outstanding, that improvement occurred faster with ECT than with any other treatment, and that ECT doubled the rate of improvement over placebo, especially in women. However, as in our study, the results with placebo alone were substantial.

To assess long-term results the same researchers followed discharged patients for up to 6 months, taking note of crossover or supplementary treatment prescribed by the clinicians who followed the patients. The authors' summary noted that ECT and imipramine were the most satisfactory treatments overall. About one-third or less of the patients were satisfactorily improved with either placebo or phenelzine, and about one-half were satisfactorily improved with either ECT or imipramine. ECT was used as a supplementary treatment more often than any other, and it was used more often with phenelzine or placebo than with imipramine. As indicated above, women responded better to ECT, and men appeared to respond better to imipramine. Women who were initially given drugs eventually required more ECT than did men.

COMPARATIVE EFFECTIVENESS OF ECT AND DRUGS IN SCHIZOPHRENIA

May's impressive 1968 study comparing ECT with ataractic drugs alone, drugs plus psychotherapy, psychotherapy alone, and milieu therapy alone in 228 schizophrenic patients (13) showed that drugs plus psychotherapy and drugs alone provided the best results. Psychotherapy and milieu treatment provided the least improvement, and ECT showed improvement in approximately the middle of the range. The schizophrenic patients in this study were considered as having "middle" prognoses, i.e., they were neither on the way to spontaneous recovery nor likely to require more than 2 years of continuous hospital care under the conditions at Camarillo State Hospital in California.

Gilligan's exhaustive 1965 review of the treatment of schizophrenia (14) showed that there was substantial agreement at that time that chlorpromazine was more effective in the treatment of chronic schizophrenia than ECT.

In his 1975 review of ECT treatment of schizophrenia (15) May tried to make sense of the many conflicting reports on the subject. A fair conclusion would seem to be that drugs are generally superior to ECT in the treatment of schizophrenia, but ECT has been successful in some cases in which drugs have failed. On the other hand, May pointed out, a number of studies have claimed that ECT is no better than placebo. Studies of the relative efficacy of ECT and psychotherapy, insulin, or psychosurgery are inadequate or lacking; there are no controlled studies of the comparative usefulness of ECT in schizo-affective disorders.

COMBINATIONS OF ECT AND DRUGS

Systematic consideration of combinations of ECT with drugs in the treatment of depression and schizophrenia has been the subject of some recent reports. The question is, can something be gained through such combinations that would benefit patients more than using ECT alone? We are indebted to Abrams for his thorough review of this problem as of 1975 (16).

Depression

Abrams cited six studies involving a total of more than 500 depressed patients using combinations of ECT with various drugs according to several experimental designs. For example, Abrams described Sager and Bird's study of 40 hospitalized patients given either 150 mg of imipramine a day or placebo and then given ECT 1 week later. They found that the ECT and imipramine combination resulted in the same number of ECT treatments and the same length of hospital stay but fewer relapses in the 6-month follow-up period.

Abrams also cited a study by Wilson of 36 hospitalized depressed women assigned to either ECT plus placebo, ECT plus 150–220 mg of imipramine a day, anesthesia plus imipramine, or anesthesia plus placebo. This study found no difference between ECT plus placebo versus ECT plus imipramine. However, ECT plus imipramine was superior to anesthesia plus imipramine and anesthesia plus placebo.

Another of Abrams' citations was Imlah's study of 150 patients divided into three groups: those given ECT plus 45 mg of phenelzine a day; those given ECT plus 75 mg of imipramine a day; and those given ECT plus placebo. This study noted no short-term differences between groups, but at 6 months the relapse rate was greater in the ECT plus placebo group. Abrams also cited two studies that found either diazepam or chlorpromazine in combination with ECT to have a possible depressing effect on ECT plus amitriptyline or ECT plus placebo, respectively. Abrams concluded that in depression, antidepressant drugs combined with ECT do not "reduce the number of treatments required, accelerate the treatment course, or enhance the final treatment outcome."

There appears to be no evidence of either good or bad synergistic effect in ECT plus the drugs mentioned above. However, the use of ECT with reserpine should be avoided because fatalities have been reported with this combination. Since tardive dyskinesia has been reported with some neuroleptics after relatively short periods of administration, their routine use should also be avoided. Combinations of tricyclics with ECT entail the risk of inducing a manic state; nevertheless, tricyclics may be used after successful ECT for maintenance of improved status. There may also be numerous instances in which drugs are required to manage agitation before or during the early stage of depression. If so, the medication should be withdrawn as soon as clinically feasible.

Schizophrenia

Studies of the use of drugs plus ECT in schizophrenia are too few to draw firm conclusions, but there is some evidence that the combination of ECT and chlorpromazine may be better than chlorpromazine alone, at least for a short period (3–4 weeks).

SUMMARY

The therapeutic value of ECT has been recognized for 40 years. Many technical improvements have been achieved during this time, including the reduction and modification of current, suppression of motor components of the convulsion, and use of unilateral nondominant ECT to mitigate the organic features of the post-shock period. Although no adequate theory exists to explain its mode of action, ECT has been of benefit to patients with psychotic depression, involutional melancholia, depression associated with brain disease, mania, schizo-affective disorders, and organic psychosis with excitement as well as to some patients with neurotic depression. It is contraindicated in psychosomatic disease, psychopathic personality, behavior

disorders of childhood, and the neuroses other than neurotic depression.

Studies of the comparative efficacy of ECT and drugs in depression suggest that ECT produced the fastest amelioration of symptoms; imipramine was next best, followed by amitriptyline, phenelzine, and isocarboxazid. ECT would be indicated for acutely suicidal patients and those who are most impaired by their illness. Results with placebo alone, it should be noted, have been substantial.

In schizophrenia, ataractic drugs plus psychotherapy and drugs alone gave results superior to those of either psychotherapy alone or milieu treatment; ECT-treated patients showed only moderate improvement. ECT has been successful in some cases in which drugs have failed.

Regarding combinations of ECT with drugs in depression, it would appear that, in general, antidepressives combined with ECT do not enhance the final outcome of treatment. The use of ECT with reserpine should be avoided because fatalities have been reported with this combination. Tricyclics may be used after successful ECT for maintenance of improved status.

Regarding combinations of drugs with ECT in schizophrenia, there are too few studies to draw firm conclusions; however, there is suggestive evidence that the combination of ECT and chlorpromazine may be better than chlorpromazine alone.

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ECT and Ethical Psychiatry

BY CARL SALZMAN, M.D.

The author discusses the right to receive ECT and describes voluntary and involuntary patients for whom the treatment seemed both ethical and humane. The right to refuse ECT must be respected in competent patients. However, problems arise when patients' decision-making ability is compromised by their illness. Informed consent questions include how much and what type of information patients should receive and how much those with disordered cognitive functioning can be expected to understand. The author believes that ECT should be fully voluntary whenever possible and that a court-appointed guardian should make such treatment decisions for incompetent patients.

A GROWING controversy is crystallizing around the subject of ECT as an ethical mode of psychiatric treatment. Many physicians view ECT as accepted clinical practice, without ethical ambiguities (1), but others (2), supported by some patients'-rights advocates (3), would call for its abolition.

Questions regarding the ethics of ECT arise from a number of sources, including: 1) the physically invasive nature of the procedures, 2) the bad reputation ECT has acquired as a result of earlier, unmodified treatment methods, and 3) the potential for severe neurological side effects. For these reasons, there is justified concern about the potential abuse of ECT. Such problems, however, must be separated from ethical concerns that attend the legitimate use of ECT. These ethical problems are 1) the right to receive ECT, 2) the right to refuse ECT, and 3) the nature of informed consent about ECT and its potential hazards. These concerns, which are shared with many other areas of psychiatric treatment, are often difficult to resolve because ECT is used primarily for severely ill patients, who may not be able to assist in the decision-making process regarding their treatment.

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THE RIGHT TO RECEIVE ECT

The most radical critics of ECT reject the notion that it has any therapeutic benefits. Others believe that whatever small benefit it might have is far outweighed by the attendant hazards. However, if one accepts the sizable body of evidence suggesting that ECT is a rapid and relatively safe form of treatment for severe affective illness, then ethical issues are cast in a different perspective. One must consider whether it is a physician's ethical right to *refuse* an effective treatment. Consider the following clinical vignette.[1]

> A 49-year-old woman was voluntarily hospitalized with her third severe depression in 7 years. Her depressive symptoms included severe early morning insomnia, anorexia and weight loss, and an inability to concentrate. The latter symptom was especially troublesome since the patient was a concert violinist who had become unable to perform because she could not concentrate on her music. Previous treatment with ECT at other institutions had offered rapid relief and a return of her ability to perform. The patient refused antidepressant medication, which had made her more agitated in the past, and requested ECT. She denied memory loss from her previous treatments.

This patient responded to eight treatments without memory loss or confusion and returned to an active teaching and performing career. Would it have been ethical to withhold this therapy from a consenting patient or to use it only as a last resort when we had evidence that her condition would probably improve rapidly following ECT administration?

The ethics of withholding ECT are of particular concern for the patient who has not responded to other forms of therapy or who, because of medical illness, cannot safely take therapeutic doses of standard antidepressants. Consider also the patient who, for professional or personal reasons, cannot financially afford the 4–6 weeks that may elapse before therapeutic blood levels of chemical antidepressants are reached and who must be returned to his/her job or family as soon as possible. Should not such patients have the voluntary right to ECT? Is it not more ethical for the psychiatrist to consider the most rapid and safe form of antidepressant therapy for such patients, i.e., ECT? The following case vignette is illustrative.

> An 82-year old man with a history of two heart attacks, active diabetes mellitus, and a fluctuating cardiac ar-

[1] This clinical vignette, like the others in this paper, describes a real patient whose identity is markedly disguised.

rythmia was admitted on a voluntary basis because of severe depression accompanied by insomnia, an 11.4-kg weight loss, constipation, and dehydration that had resulted in serious electrolyte imbalance and decreased renal function. Because of the severe medical complications, the patient was not medically cleared for chemical antidepressant therapy but was cleared for ECT and anesthesia. An internal medicine consultant expressed the opinion that the patient was in danger of another heart attack or potentially fatal cardiac arrythmia if the symptoms of his depression (particularly the anorexia, as well as the insomnia) were not rapidly alleviated.

This patient responded after only six treatments and "seemed like a new person," according to his family. If one accepts the available data that ECT is a useful treatment for certain forms of affective illness and that it is sometimes less hazardous than doing nothing or using antidepressant drugs or long-term hospitalizations (or psychotherapy), then it would seem an ethical necessity to offer ECT to the consenting voluntary patient.

The right to treatment is most often associated with involuntary hospitalization. In the case of *Wyatt v. Stickney* the courts ruled that a patient cannot be confined involuntarily if he/she is not offered treatment (4–6). What if the patient is involuntarily hospitalized with a severe depression? The ethical problems of considering ECT for such a patient are illustrated by the following example.

The patient, a 67-year-old man, had a 5-month history of progressive weight loss, insomnia, and increasing suicidal preoccupation. He lived alone and prepared his own meals, and a neighbor had become concerned when she had not seen him for several days. With police assistance, he was discovered lying in his own waste; his body was covered with bed sores from excoriation and immobility. He was nearly mute, delusional, and clearly unable to care for himself. After medical treatment he was involuntarily hospitalized for treatment of a psychotic depression. His medical condition prohibited chemical antidepressants. He was unable to get out of bed and barely able to talk, so he could not participate in the therapeutic life of the ward community or in any type of psychotherapy. He was considered to be a psychiatric emergency.

Although few would disagree that this patient was severely ill and in need of care, he clearly had a right to be released from involuntary hospitalization if he could be offered appropriate treatment. The decision to offer ECT treatment was ethical, humane, and well within his rights as a patient. Beresford (7) has suggested that "involuntary patients who are not offered ECT for their psychotic depression may later contend that the hospital was negligent in omitting this medically indicated and highly effective treatment."

THE RIGHT TO REFUSE ECT

The ethical problems associated with ECT grow more complex when one considers a patient's right to refuse ECT. This right to refuse treatment, i.e., to decide one's own destiny, was well expressed by the 19th century British philosopher John Stuart Mill (8):

Considerations to aid his judgment, exhortations to strengthen his will, may be offered to him, even obtruded on him by others; but he, himself, is the final judge. All errors which he is likely to commit against advice and warning, are far outweighed by the evil of allowing others to constrain him to what they deem his good. . . . But neither one person, nor any number of persons, is warranted in saying to another human creature of ripe years, that he shall not do with his life for his own benefit what he chooses to do with it. He is the person most interested in his own well-being: the interest which any other person except in cases of strong personal attachment, can have in it, is trifling. . . . (8, p. 273)

More recently, Szasz (9) reminded us that patients have a right to self-determination, including a right to refuse treatment. The right to refuse ECT, like the right to refuse psychosurgery or other treatments, is a civil liberty that must be protected.

The ethics of considering ECT for nonconsenting patients involve the concept of *parens patriae* or psychiatric paternalism, i.e., the belief that the psychiatrist knows better than the patient what is good for the patient's health. Here we must consider the peculiar dilemma that confronts all psychiatrists: the patient's ability to make a rational decision about his or her own well-being is sometimes compromised by the nature of the disorder for which the treatment is indicated. This may be the case when ECT is being considered for treatment of psychotic depressive disorders. The following case vignette raises such an issue:

A 69-year-old man with a history of depressions was referred for hospitalization because of severe depression. The patient was agitated, pacing, wringing his hands, and crying "Oh, my God" over and over. He refused voluntary hospitalization, stating that his case was hopeless because his intestines and brain were rotted with cancer. This was untrue. When confronted with evidence of his normal physical functioning, the patient groaned that the cancers which were rotting his body were punishment for past sexual sins; the doctors' failure to perceive the cancers was dismissed as incompetence. For the next three weeks of hospitalization his delusions became even more fixed, and he refused food as well as any form of treatment.

This patient was given ECT after a court hearing at which he was judged incompetent to care for himself. A legal guardian was appointed to sign permission for the treatment. The depression lifted after 10 ECT treatments, and the patient left the hospital quite grateful for the help he had received. He stated he was glad that he had been treated against his will because "That wasn't the real me talking when I was sick."

What if the patient has an illness that is known to respond to ECT but is competent and continues to refuse the offer of treatment despite personal suffering as

well as the anguish of a concerned family and community? This situation, which is one of the most ethically precarious in psychiatry, is illustrated by the following brief case report:

> A 58-year-old divorced woman had several voluntary admissions to the hospital during a 6-month period. Each hospitalization was preceded by a brief stay in a medical hospital where she was treated for inanition, multiple self-inflicted skin excoriations, severe insomnia, and agitation. She would regularly visit many of the better staffed university hospital emergency rooms in Boston with multiple medical symptoms; all physical examinations were within normal limits. A diagnosis of recurrent unipolar depression had been made during a previous admission, and the patient was started on tricyclic antidepressant medication. During the ensuing months, several different tricyclic medications as well as a trial of monoamine oxidase inhibitors and lithium carbonate failed to alter her symptoms or suffering. She consistently refused ECT. She was judged to be nonpsychotic and not commitable by virtue of her ability to feed, clothe, house, and protect herself.

This patient did *not* receive ECT, even though the hospital staff tried to help her understand that without the treatment her suffering might worsen. Is the cost to the patient of declining health, as well as possible cost to a family or community, also part of the ethical decision to be made by the psychiatrist? I believe that in such cases the patient's wish not to be treated must be respected, despite pressures from family, friends, or community. Although it is painful for us to watch a patient suffer or even grow more symptomatic, we cannot force treatment on a competent nonconsenting person. We must, in these cases, relinquish the role of *parens patriae* in order fully to protect patients' rights.

Stone (10) has suggested that at a full judicial hearing a third-party decision maker in the person of a judge be required before ECT is administered to a protesting patient. He adds that "other third parties such as guardians, relatives, patient rights committees or other physicians not on the hospital staff will not insure the patient's civil liberties." He suggests a two-step procedure: a preliminary judicial hearing at which it is decided whether or not the observation, diagnosis, and preliminary treatment have been appropriate, and a second hearing, with the patient represented by counsel, in which parts of the treatment plan to which the patient objects are dealt with. Although such a policy may help safeguard patient rights, the essential ethical dilemmas are still unanswered. Does a psychiatrist have the right to do more than offer treatment? What is the psychiatrist's role with a patient whose ability to make health care decisions may be compromised by the symptoms for which the treatment is indicated?

INFORMED CONSENT FOR ECT

The last of the ethical dilemmas concerns the issue of how much information is necessary for a patient to make an informed decision about having ECT. Clearly, informed consent is an essential prerequisite for ECT. The dilemmas arise in 1) determining how much information must be given to ensure that consent is "informed," without scaring away the patient, and 2) establishing how much can be understood by patients with disordered cognitive functioning. In recent years, for example, the question of permanent disabling memory loss following ECT has become a concern. A thoughtful article in *The New Yorker* (11) recounts the case of a highly intelligent professional woman who, after being treated with a standard course of unilateral, nondominant temperoparietal ECT for a mid-life depression, became unable to resume her job as a result of alleged permanent memory loss (objective memory testing apparently was not performed). Although post-ECT amnesia has generally been demonstrated to be transient, this report raises the possibility that in rare or occasional instances the patient may experience memory loss as permanent. Is there sufficient evidence about such permanent memory loss to require psychiatrists to inform their patients of this possible consequence of ECT? At what level of statistical frequency does it become necessary to mention such a risk to a patient? Does the sharing of such information constitute a risk in itself?

It is hard to imagine that any patient who has been fully informed of the possibility of permanent, near-total memory loss would consent to such a procedure. If patients who might benefit from ECT are frightened away, have we done them a service? Ledensohn (12) notes that in discussions of the possible risks of ECT the patient and family must also be cautioned about the possible risks of not receiving ECT, such as suicide. Stone (10) noted that very large doses of potentially toxic medication, restraints, or maximum security measures may be necessary to protect the life of an acutely suicidal patient who does not receive ECT. Krouner (13) has reviewed and commented on the great variability among the different state laws regarding informed consent and ECT. State courts have had difficulty deciding how much information is helpful to a patient facing ECT, and decisions have ranged from no informed consent to the requirement of reasonable and recognized risks.

An even more difficult question involves the capacity of a severely ill patient to be informed at all. Consider the following case.

> A 52-year-old fully employed woman with a long history of recurrent depressions was voluntarily hospitalized with a psychotic depressive illness. She agreed to ECT without seeming to pay attention to the explanations of the treatment. After reading and signing the informed consent form, the patient remarked, "It doesn't make any difference anyway because it is all over for me; I am dead." This voluntary patient would probably have consented to any procedure. Did she truly understand the treatment—its effects and side effects—for which she gave permission?

To varying degrees, psychotically depressed patients may be incapable of fully appreciating what they are being told; they may be equally incapable of making an appropriate decision on the basis of such information. May we ethically treat them on the basis of such consent?

COMMENT

On the basis of available evidence, ECT has a legitimate clinical use for certain patients. It is neither a panacea nor a sadistic intrusion on personal liberty. Because patients for whom ECT is indicated often are severely ill, ethical dilemmas can arise regarding its use. The following guidelines are offered to help ensure that patients' rights, as well as concerns related to health, are respected and protected:

1. ECT should be given whenever possible as a fully voluntary procedure.
2. In cases of legal incompetence or when patients are unable to give informed consent, a court hearing should be convened to appoint a legal guardian for the patient. This guardian must act on behalf of the patient, not the psychiatrist or hospital, and decide about ECT as the patient would if he or she were competent to decide about health care as well as about civil rights. The guidelines suggested by Stone are excellent (10).
3. Information given to obtain informed consent should include the possibility of increased risks when there is concurrent medical pathology or dementia. The potential for memory loss should be discussed with the patient in advance, although it is reasonable to reassure the patient that on the basis of available evidence and clinical experience, memory loss is usually transient. The risks of not having ECT should also be explained, i.e., the risks of no treatment or alternative somatic therapy. Informed consent for ECT must be obtained independently from other hospital consent forms *before* the first treatment is given.

Although a patient's right to receive or not receive ECT can be protected, ultimately the public and the psychiatric profession must decide whether they believe ECT to be therapeutic or harmful. With increased cooperation among psychiatrists, lawyers, and civil libertarians, research can continue to evaluate the future role of ECT in psychiatry. Such cooperation rather than antagonism can help to establish a better set of ethical guidelines for the treatment as well as protection of patients' health and civil rights.

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Shock Treatment, Brain Damage, and Memory Loss: A Neurological Perspective

BY JOHN FRIEDBERG, M.D.

The author reviews reports of neuropathology resulting from electroconvulsive therapy in experimental animals and humans. Although findings of petechial hemorrhage, gliosis, and neuronal loss were well established in the decade following the introduction of ECT, they have been generally ignored since then. ECT produces characteristic EEG changes and severe retrograde amnesia, as well as other more subtle effects on memory and learning. The author concludes that ECT results in brain disease and questions whether doctors should offer brain damage to their patients.

A 32-YEAR-OLD WOMAN who had received 21 ECT treatments stated 5 years later,

> One of the results of the whole thing is that I have no memory of what happened in the year to year and a half prior to my shock treatments. The doctor assured me that it was going to come back and it never has. I don't remember a bloody thing. I couldn't even find my way around the town I lived in for three years. If I walked into a building I didn't even know where I was. I could barely find my way around my own house. I could sew and knit before, but afterward I could no more comprehend a pattern to sew than the man in the moon. (1, p. 22)

By 1928, 10 years before the introduction of electroconvulsive therapy, it was known that accidental death by cardiac arrest could result from as little as 70 to 80 milliamperes in the human (2). It was also known in this early period that voltage applied to the head, as in legal electrocution, produced hemorrhage and rupture of cranial contents. Ugo Cerletti (3) demonstrated that electricity in the range of 100 volts and 200 milliamperes is rarely fatal when the current path is confined to the head, but does evoke a grand mal seizure marked by a stereotyped succession of events. A tetanic muscular contraction, the "electric spasm," is followed after a latency of seconds by unconsciousness, a high voltage paroxysmal spike and sharp-wave discharge, and a clonic convulsion. Upon recovery of consciousness the subject is left with a transient acute brain syndrome, a high likelihood of permanent brain damage, and greater retrograde amnesia than is seen in any other form of head injury.

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BRAIN DAMAGE IN EXPERIMENTAL ANIMALS

Before examining the premise that ECT damages human brains, a brief discussion of the lesions produced in animals by electrically induced convulsions is worthwhile. The many reports on this subject indicate that petechial hemorrhages scattered throughout both white and gray matter and concentrated in the path of the current are the most consistent finding. If animals are sacrificed after a delay of days or weeks following a convulsive series, hemosiderin pigment in phagocytes remains as evidence of vascular insult. Proliferation of glial cells, neuronal changes, and dropout are also commonly reported.

In 1938, the year of the first use of ECT on a human being, Lucio Bini, Cerletti's collaborator, reported "widespread and severe" brain damage in dogs with mouth to rectum electrode placement (4). At least seven subsequent animal studies employing conventional cranial electrodes supported his findings (5–11). These culminated in the exhaustive controlled experiment by Hans Hartelius in 1952 (12). This researcher found discernible vascular, glial, and neuronal changes in cats subjected to a maximum of 16 shocks. The animals were not paralyzed but were protected from physical injury during the seizure. Damage was slight but consistent, and the author concluded: "The question of whether or not irreversible damage to the nerve cells may occur in association with ECT must therefore be answered in the affirmative." Furthermore, by examination of unlabeled slides alone Hartelius was able to correctly recognize 8 of 8 slides from shocked animals as well as 8 of 8 controls. Although he considered many of the vascular and glial changes to be reversible, there was no mistaking the brain of a shocked animal for that of a control.

Since that time, ECT in humans has been modified through the use of oxygen and muscle paralysis to reduce the incidence of bone fractures. Although it is believed that these modifications also reduce brain

damage, there are no animal studies to support this idea. On the contrary, recent work in England by Meldrum and associates (13, 14) on status epilepticus in primates suggests that the overexcited neuron by itself may be an important factor in seizure damage, especially in the hippocampus.

HUMAN BRAIN DAMAGE

Let us turn now to the neuropathological findings in humans who died during or shortly after ECT. As in lower animals, bleeding is the most frequent nonspecific tissue response to injury and the one seen most often after electric shock. The first autopsy study in this country revealed brain damage identical to that seen in experimental animals. Alpers and Hughes (15) described the brains of 2 women who had received 62 and 6 shocks, respectively. The first woman's seizures had been suppressed by curare. Both brains showed hemorrhagic lesions around small blood vessels, rarefaction of tissue, and gliosis.

Throughout the 1940s similar reports continued to call attention to brain changes after ECT, including cases in which oxygen and curare had been administered (16). In 1948 Riese (17) added 2 more autopsy studies to the growing list and commented, "In all observations of sudden death after electric shock reported so far, petechial hemorrhages, cellular changes and some glial proliferation stand out prominently, as an almost constant whole."

Pathologists were especially interested in cases that discriminated between the direct effect of electricity and the mechanical and hypoxic effects secondary to convulsive motor activity. In 1953 Larsen reported on a 45-year-old man who had been given 4 electroshocks in the course of 5 days. The ECT did not induce any convulsions. The subject died from pneumonia 36 hours after the fourth electroshock. At autopsy fresh subarachnoid hemorrhage was found in the upper part of the left motor region—"at the site where an electrode had been applied" (18).

In 1957 Impastato summarized 254 electroshock fatalities. Brain damage was the leading cause of death in persons under 40 years of age, and nearly one-fifth of all cerebral deaths were hemorrhagic (19).

Some physicians were alarmed by the evidence of human brain damage. In 1959 Allen reported 18 cases in which he had found signs and symptoms of neurological sequelae following ECT. He concluded, "It is probable that some damage, which may be reversible but is often irreversible, is inseparable from this form of treatment," and called for "more serious consideration" of the entire procedure (20).

In 1963 McKegney and associates (21) reported the case of a 23-year-old man who became comatose 15 minutes after a single shock. The significance of this case was twofold: first, a complete physical and neurological examination was reportedly normal prior to ECT, and second, the ECT technique was contemporary and impeccable. The patient had received .6 mg of atropine, 16 mg of succinylcholine (Anectine), and forced oxygenation pre- and post-shock. ECT parameters were conventional, i.e., 130 volts for .3 seconds. Four days later a brain biopsy showed diffuse degeneration of neurons with hyperplasia of astrocytes. The young man never regained consciousness and at autopsy 2 months later evidence of old hemorrhage was found in the brain. This was the last detailed report in the English-language literature.

The damaging effects of ECT on the brain are thoroughly documented. All told, there have been 21 reports of neuropathology in humans (22–36). It is interesting that, despite the importance of a negative finding, there has not been a single detailed report of a normal human brain after shock.

ELECTROENCEPHALOGRAPHIC EFFECTS OF ECT

Like other insults to the brain, ECT produces EEG abnormalities. Diffuse slowing in the delta and theta range, increased voltage, and dysrhythmic activity are seen in all patients immediately following a series of bilateral ECT and, according to Blaurock and associates (37), may persist more than 6 months in 30% of the cases. Such slowing suggests damage to the thalamus.

Sutherland and associates (38) showed that the side of the brain shocked with unilateral ECT could be predicted by double-blind assessment of EEG tracings.

The seizure thresholds of the hippocampus and other temporal lobe structures are the lowest in the brain; considerable interest has centered recently around "kindling," or seizure induction by subthreshold stimulation of these areas in animals (39). The induction of a permanent epileptic disorder following ECT in humans was first reported in 1942 and other reports followed (40).

MEMORY LOSS

ECT is a common cause of severe retrograde amnesia, i.e., destruction of memories of events prior to an injury. The potency of ECT as an amnestic exceeds that of severe closed head injury with coma. It is surpassed only by prolonged deficiency of thiamine pyrophosphate, bilateral temporal lobectomy, and the accelerated dementias, such as Alzheimer's.

After ECT it takes 5 to 10 minutes just to remember who you are, where you are, and what day it is. In the first weeks after a full course, retrograde and, to a lesser extent, anterograde amnesia are evident to the casual observer. But as time passes compensation occurs. As in other forms of brain injury, the subject is often oblivious to the residual deficit. Unless specific memories essential to daily living are discovered to be unavailable the victim may never know for sure the extent of memory loss. Unless sensitive tests for spon-

taneous recall of personal preshock data are employed, no one else will know either.

The memory loss following ECT generally follows Ribot's law for all pathological amnesias: the new dies before the old. This, of course, is the opposite of normal forgetting. Squire, however, has shown that the loss may extend to items learned more than 30 years before (41).

The effect of ECT on memory was common knowledge within a few years of its introduction. There were reports of persons who forgot they had children (42, 43), although most amnesias involved humbler matters, such as the woman who forgot how to cook familiar dishes (44) and another who couldn't remember her own clothing and demanded to know who had put the unfamiliar dresses in her closet (45). Some doctors dismissed these sequelae as trivial or transient, although one psychiatrist remarked that psychotherapy was useless in patients undergoing ECT because they couldn't remember "either the analyst or the content of the analytic sessions from one day to the next" (46).

Numerous such case reports finally led to a definitive study of the effects of ECT on memory by Irving Janis in 1950 (47). He found that all 19 subjects in a controlled prospective investigation had significant memory loss 4 weeks after ECT, compared to negligible losses among control subjects. He also noted that these losses may involve events of early childhood dating back 20 to 40 years, with the more recently encoded memories being the most vulnerable. Patient E, for example, a 38-year-old woman, had told Janis in an interview prior to ECT that thyroid medication had caused heart palpitations and panic which led to her admission to the psychiatric hospital. When asked after a course of 10 shocks if she had ever taken thyroid she responded, "I don't think so."

In the late 1940s, when the enthusiasm for ECT seemed to have passed its peak (48), Lancaster and associates (49) advocated the use of unilateral nondominant ECT in treating patients who earn their livelihood with retained knowledge. In this variant the current path and most of the damage is confined to the nonverbal side of the brain, usually the right hemisphere. This exploits the well-known neurological phenomenon of anosognosia, or denial, that is associated with right-hemisphere lesions—victims can't verbalize their difficulties. They complain less. Cohen and associates (50), however, using design-completion tests, proved that shock to the right hemisphere produces its own kind of memory loss—visual and spatial. Inglis found in 1970 (51) that the effects of unilateral ECT were comparable to those of right and left temporal lobectomy, with identical impairment of memory and learning.

Recently there has been a good deal of human experimentation in a futile effort to find electrode placements that eliminate amnesia. As the use of ECT has shifted from state hospitals to private practice, the literature has focused more and more on memory loss. Although some studies have purported to show improvement of learning ability after ECT, not one used sham ECT as a control and few used any controls at all.[1]

In regard to more general intellectual ability, a study in 1973 (54) showed that the performance on the Bender Gestalt perceptual motor test of 20 institutionalized subjects who had received 50 or more ECT treatments 10 to 15 years before testing was significantly impaired compared to the performance of 20 carefully matched control subjects who had not received ECT. The authors inferred that ECT had caused permanent brain damage.

MECHANISM OF ACTION OF ECT

The mechanism of action of ECT can now be summarized on the basis of evidence accumulated since its introduction. Penfield and Perot showed in the 1950s that memory traces may be evoked by direct electrical stimulation of the temporal lobe cortex, and nowhere else (55). Scoville and Milner (56) discovered that bilateral hippocampal resection utterly abolished the ability to remember any new material, resulting in a catastrophic inability to learn. From numerous studies of the neuropathology of the amnestic-confabulatory syndrome of Korsakoff it is known that the mammillary bodies, the dorsal median nuclei of the thalamus, and the gray matter surrounding the third ventricle and aqueduct are essential to the general memory process. All of these critical brain structures are just beneath the thin squamous plate of the temporal bone, within seven centimeters of the electrodes, in the direct path and highest density of the current during ECT.

CONCLUSIONS

From a neurological point of view ECT is a method of producing amnesia by selectively damaging the temporal lobes and the structures within them. When it was first introduced it was only one of several methods of producing brain damage employed in psychiatry, including insulin coma (1927), camphor and pentylenetetrazol (Metrazol) injections (1933), and prefrontal lobotomy (1935). It is the only such method from that era still used on a large scale. It is highly unlikely that ECT, if critically examined, would be found acceptable by today's standards of safety.

From a neurological point of view ECT produces a form of brain disease, with an estimated incidence of new cases in the range of 100,000 per year (57). Many psychiatrists are unaware that ECT causes brain damage and memory loss because numerous authorities and a leading psychiatric textbook (58) deny these

[1] Sham ECT, an essential control technique, has been employed in only two studies, which were tests of efficacy, not tests of memory loss. Neither study showed any superiority of ECT over the control treatment (52, 53).

facts. Others, who know of its effects, argue that the interruption of unpleasant states of mind is worth the damage. Some are beginning to give the client a truly informed choice, although most state laws still allow ECT to be imposed if the doctor feels that "good cause" exists.

Assuming free and fully informed consent, it is well to reaffirm the individual's right to pursue happiness through brain damage if he or she so chooses. But we might ask outselves whether we, as doctors sworn to the Hippocratic Oath, should be offering it.

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Current Perspectives on ECT: A Discussion

BY FRED H. FRANKEL, M.B.CH.B., D.P.M.

MY COMMENTS about each of the preceding papers on ECT reflect my personal views and not those of the former Massachusetts Task Force on ECT (1) or the American Psychiatric Association's Task Force on ECT. Space limitations preclude a complete discussion of each of the papers, but I will attempt to address at least some of their important or controversial points.

ECT: A NEUROLOGICAL PERSPECTIVE

In my view, Dr. Friedberg has weakened his position by the manner in which he has gathered the evidence to support it. The questions he raises are relevant; they have been asked by many others. However, he has attempted to answer them with data that have been carelessly culled from the literature and frequently reported inaccurately.

Dr. Friedberg's evidence against ECT is arranged in four main sections. He reports on the neuropathological findings in experimental animals subjected to electrically induced convulsions, and in humans who have died during a course of ECT or within weeks or months afterward. He then reports studies and subjective accounts of memory loss and, finally, EEG changes. He concludes that "from a neurological point of view ECT is a method of producing amnesia by selectively damaging the temporal lobes and the structures within them." He states that "ECT produces a form of brain disease" of epidemic proportions and asks why doctors, who are sworn to the Hippocratic Oath, are offering it.

Dr. Friedberg has listed 58 references in his article. I have examined 25 of these and selected those which seemed especially relevant to his argument.

In presenting his evidence for brain damage in animals exposed to electrical currents, Dr. Friedberg states: "In 1938, the year of the first use of ECT on a human being, Lucio Bini, Cerletti's collaborator, reported 'widespread and severe' brain damage in dogs. At least seven subsequent animal studies confirmed his findings." The following quotation is from the Bini reference:

> We have so far employed exclusively the method of Viale, which consists of passing the street current (120 volts) for a very short time (1/15 to 1/20 second) through the entire body of the animal with one of two electrodes (carbons from a voltaic arc) in the mouth and the other in the rectum.
>
> With this method we succeeded in producing constantly typical epileptic attacks in dogs. During the passage of the current the animal howls and has a violent tonic spasm with opisthotonus which lasts several seconds after the circuit is opened. Then there appear frequent and violent generalized tonic and clonic convulsions with foaming at the mouth, biting of the tongue, and incontinence of urine and feces. The duration of this second phase varies from 1–2 minutes. There follows a comatose state with complete muscular relaxation, absence of corneal and pupillary reflexes and stertorous breathing. In a short time the

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> animal returns to its normal state, so that after a few minutes new seizures may be induced.
>
> The alterations found by us in the nervous systems of these dogs were widespread and severe. (2, p. 173)

Bini did not state how many seizures were induced, but clearly they were multiple, and the current was applied along the course of the entire body, with one electrode in the mouth and the other in the rectum. I cannot seriously believe that Friedberg considers this to be the customary method of administering ECT, and yet he has quoted Bini's report as if the procedures described were analogous to those used today. Furthermore, there is no mention of the method of sacrificing the animals and no consideration of the fact that anoxia or trauma could have contributed to Bini's findings in the central nervous system.

Dr. Friedberg then claims that seven subsequent animal studies confirmed Bini's findings. In doing so, he has revived the controversy that waxed during the 1940s. I have examined five of these references. Far from confirming the point, two (3, 4) unequivocally disagree with the idea that widespread and severe damage results from electrically induced convulsions in animals, *unless* the current is abnormally prolonged or administered repeatedly or in excess of that used in ECT.

The three other studies I examined are the well-publicized reports by Alpers and Hughes (5), Heilbrunn and Weil (6), and Neuberger and associates (7). They all reported damage to the central nervous system. However, a detailed study of their comments revealed less than the well-established evidence that Friedberg claims supports his argument. Alpers and Hughes (5) made no mention of the voltage used. Heilbrunn and Weil (6) reported an electrically produced convulsion in 28 rabbits, 16 of which were paralyzed in the lower limbs, bladder, and rectum after the first few shocks. Nonetheless, they were then still shocked daily for several days before being sacrificed and examined. The size of the electrodes and the current used were equivalent to those employed in ECT and were totally disproportionate to the size of the rabbit's brain. In no way can that study be considered relevant to the current administration of ECT. Neuberger and associates reported that although they found pathological changes in the brains of the animals studied, these changes "were not to be regarded as serious."

Space limitations preclude a detailed examination of the other contradictions and inconsistencies that unfold during an examination of Dr. Friedberg's bibliography, but it is necessary to study a few of them in some detail if we are to place his paper in perspective. He has published widely and has worked hard at trying to influence public opinion. He has revived the controversy of the 1940s and 1950s and maintains that the introduction of anesthesia, oxygenation, and muscle paralysis since that time has in no way diminished the risks of ECT. He appears to base his position on the following two principal assertions: 1) he argues as if the damage caused by a single seizure must be as great as that caused by large numbers of seizures concentrated within a brief period of time, and 2) he states emphatically, "Although it is believed that these modifications [of ECT, namely, the use of anesthesia, oxygenation, and muscle paralysis] also reduce brain damage, there are no animal studies to support this idea."

Dr. Friedberg's next sentence is perhaps the most misleading. "On the contrary, recent work in England by Meldrum and associates [8, 9] on status epilepticus in primates suggests that the overexcited neuron by itself may be an important factor in seizure damage, especially in the hippocampus."

The recent reports by Meldrum and associates deal with the effects of prolonged seizure activity in baboons in an attempt to understand the effects of status epilepticus in humans. In the first study (8), seizures were precipitated in 8 anesthetized adolescent baboons by injections of the convulsant bicuculline. The animals were artificially paralyzed and oxygenated, and the seizure activity was recorded on EEG. Physiological factors were monitored. The activity lasted from 3¼ to 7½ hours in 7 of the 8 animals. The eighth animal experienced a period of 13 minutes of seizure activity and then, after a second injection of the convulsant almost 3 hours later, experienced an additional hour and 49 minutes of seizure activity. The animals were sacrificed during or shortly after the seizures and the brains examined. There were ischaemic cell changes in the brains of the first 7 animals, but not in the brain of the eighth animal, which had experienced only a little over 2 hours of seizure activity. Meldrum and associates (8) noted that in comparison with their previous work, the presence of peripheral motor paralysis and artificial respiration greatly reduced the severity of the systemic changes and modified the pattern of epileptic brain damage.

How do these comments fit in with Dr. Friedberg's assertion that there are no animal studies to support the idea that modifications of ECT reduce brain damage? In support of his position he reported, out of context, a suggestion by Meldrum and associates that the overexcited neuron by itself may be an important factor in seizure damage. The overexcited neuron alluded to in the original paper refers to the neuron after excessive discharge and seizure activity lasting from 3¼ to 7½ hours, which is scarcely comparable to the neuronal activity associated with a single session of ECT.

In a subsequent study by Meldrum and associates (9), 13 baboons were submitted to similar experiments using the convulsant allylglycine. Five animals developed status epilepticus, and in the other 8 animals, 6–63 seizures occurred in a period of 2–11 hours. Progressive hypoglycemia and hyperpyrexia occurred with the recurrent seizures. The animals were sacrificed at varying stages up to 6 weeks after the experience, and the brains examined. The animals that had had 6–26 seizures over a period of several hours showed *no* pathological sequelae. The animals that had had more than 26 seizures, or status epilepticus,

showed varying degrees of ischaemic cell change and neuronal loss with gliosis in the neocortex and lesions in the hippocampus. Meldrum and associates felt that the relative importance of the various physiological changes associated with the repeated seizures could not be definitively established, and they concluded that the effects of hypoglycemia, hyperpyrexia, reduced oxygenation, and impaired cerebral perfusion were probably additive in causing the damage.

Dr. Friedberg's discussion of brain damage found at autopsy on patients who died during or after ECT contains an equally unconvincing body of evidence. It leaves the reader wondering, in many instances, about the true nature of the disease for which ECT had been given, how carefully neurological disease had been excluded prior to administration of ECT, and what role cardiovascular and renal disease had played in causing the pathology found at autopsy. McKegney and Panzetta's report in 1963 (10) of a 23-year-old man who became comatose 15 minutes after his first course of ECT and died 2 months later without having regained consciousness illustrates the point very well. The following material is from the original article:

> The patient, a 23-year-old, single, white male . . . was initially seen in the emergency department of a general hospital on Apr. 5, 1962 because of withdrawn and bizarre behavior. He had a long history of "emotional difficulty". . . .
>
> The medical house officer who first saw the patient . . . was unable to perform a complete physical examination since the patient seemed purposively to resist, giggling inappropriately at times, curling himself up and lying with his hands between his thighs. Psychiatric consultation at that time suggested the diagnosis of schizophrenic reaction, catatonic type, and the patient was admitted to the psychiatric unit.
>
> The patient remained essentially unchanged throughout the rest of the evening and the following day, lying in bed with his eyes closed. When called by name he would start and half open his eyes, but then close them quickly and turn his back on the examiner. . . . There were considerable posturing and unusual movements. A complete physical and neurological examination, performed on the second hospital day, was negative in all respects other than the behavior noted above. The axial temperature was 37° C; blood pressure 120/72; pulse 58 per minute and regular; respiration 16–20 per minute and regular.
>
> In order that the patient could be fed and more readily mobilized, he was given his first ECT on the morning of Apr. 7 (atropine .6 mgm., as premedication; anectine 16 mgm. intravenously; 130 volts for 0.3 seconds; forced breathing with oxygen pre and post ECT). (10, pp. 398–399)

It was soon apparent that the patient's post-ECT recovery was not normal. The patient died June 11, 1962, more than two months later, without ever deviating from his progressively deteriorating course. Brain biopsy showed diffuse degeneration of neurons with hyperplasia of astrocytes. At autopsy, old hemorrhage was found in the brain.

There are several interesting and challenging details of this case report not recounted fully here. I would nonetheless have hoped that Dr. Friedberg, as a neurologist, would have been sensitive to the neurologically salient points in the case. It is most unlikely that the subtle measurements that are part of a complete neurological examination were possible in a patient as uncooperative as this one; and the EEG and cerebrospinal fluid analysis and culture were carried out *after* the ECT, not before it. The diagnosis of catatonic schizophrenia is poorly supported by the information in the case history, and in my opinion the presence of organic brain disease had not been ruled out before the hasty use of ECT to control the patient's symptoms. In the absence of a clear diagnosis, the use of ECT to control symptoms is ill-advised.

Allusions in Dr. Friedberg's paper to several other articles fail to reflect the balanced view that the articles actually convey when read in the original. Interested readers should examine for themselves some of the studies to which he refers. The literature of that period is rich in reports expressing differing views. Dr. Friedberg either ignores them or, as I have indicated, quotes them erroneously.

His comments regarding memory loss after ECT, and EEG changes, are made with the same bold, sweeping strokes. There are occasional complaints of memory loss after ECT, but the results of unilateral electrode placement would not be regarded as a "futile effort" to remedy the problem. Dr. Friedberg would have us believe that because of damage to the right cerebral hemisphere after unilateral ECT, the improvements reported by patients are attributable to their denial of their illness and their inability to verbalize their difficulties. No one who has ever talked with patients and shared their relief and delight at feeling better and capable of a normal life after ECT can take Dr. Friedberg's assertion seriously, especially when 6–12 months later these patients are still functioning and feeling well.

None of my statements should be construed to mean that there are no unanswered questions regarding ECT. I believe there are many unsolved problems, as the other articles in this special section have attested. There is no reason to assume that we can induce large numbers of seizures with inpunity because less than 26 seizures in Meldrum and associates' baboons did not seem to be associated with cerebral damage. In addition, it is clear that there are insufficient data at this time from which to understand the occasional complaints of persistent memory disturbance after ECT. The APA Task Force on ECT, on which I serve, was established to weigh the evidence as carefully as possible and to assess realistically the cost/benefit ratio of ECT. There is no simple answer to the questions, and Dr. Friedberg's determination to present one has involved him in a report of the literature that is not only inaccurate and careless, but also somewhat indiscriminate. In addition, his reports of memory impairment have been gathered from people who answered his newspaper advertisement soliciting complaints. He

headed the advertisement, "Shock treatment is not good for your brain." He is, therefore, basing his conclusions regarding memory loss on a biased sample; in addition, he could have harmed the very people he claims to be helping by reinforcing the secondary gain provided by their amnesia and their overwhelming yet poorly understood anger.

It seems to me that Dr. Friedberg's personal opinions have been presented as scholarly decisions. He has directed them at stirring up emotions in an area already sorely in need of reasoned discourse and a serious effort to understand why, when ECT has brought relief to so many thousands of patients in the past, in some cases it has resulted in dissatisfaction and angry complaints. I hope that he will consider joining the ranks of those who aim to grapple with the issues by carefully and painstakingly sifting the evidence and concentrating on the facts.

ECT AND MEMORY LOSS

Dr. Squire presents a methodology for the understanding of ECT and memory loss that explores areas which have been especially challenging. By a system of comparisons among patients who received bilateral ECT, right unilateral ECT, or no ECT, and using tests that are quite ingenious in design, he found that memory recovers substantially by six to nine months after the completion of bilateral or right unilateral ECT, but that persisting memory complaints are not uncommon in individuals who have received bilateral treatment.

The risks to memory associated with unilateral treatment are markedly less than those associated with bilateral treatment, but the fact remains that some risk does exist. Clinical situations must be evaluated carefully. The benefits to be derived from the procedure must be weighed against its risks and the risks weighed against the discomfort or anguish of no treatment, the discomfort and even danger—especially in the older patient—of the side effects of antidepressant drugs, and the risk of suicide.

I would hope that Dr. Squire's studies will be replicated by others. Confirmation of his findings by other investigators will be reassuring and is, in fact, necessary. I find especially interesting his suggestion that bilateral ECT might itself lead to a lingering sense of memory impairment due to the fact that some individuals are more sensitive to subsequent failure to remember than others. Studies of differences in personality and cognitive style among those who are sensitive and those who are not might be helpful.

EFFICACY OF ECT IN AFFECTIVE AND SCHIZOPHRENIC ILLNESS

Dr. Greenblatt's paper conveyed the feeling of relief and dramatic uplift that attends the successful administration of ECT—not only for the patient who experiences the mood change, but also for the relatives and the psychiatrist. His statements remind us that ECT is and often has been prescribed by sincere people who are motivated to help their patients get better. The motivation to use ECT would have been dampened long ago had the treatment never promised, nor produced, successful results. The studies reported by Dr. Greenblatt bear testimony to the fact that for some forms of depression ECT is by far the most useful treatment. What is less obvious is why it does not always work when we expect it to and why it occasionally works with conditions for which it is considered to be only minimally effective.

Dr. Greenblatt proposes a protocol that the cautious may already be following, namely: "Imipramine or phenelzine is indicated for patients who are not acutely suicidal; if no improvement occurs after 3 weeks, ECT is indicated." While there are probably many physicians who would want to wait longer than three weeks, and others who would wait indefinitely rather than give ECT, there are those who believe that under some circumstances the use of ECT at the very beginning of the treatment procedure is justified. Would we agree that there are situations in which that might be the wisest clinical course?

In the treatment of atypical and reactive or neurotic depressions, the comparative value of ECT on the one hand, and medication plus psychotherapy on the other, has not been adequately studied. Nevertheless, we know that ECT is often administered in the presence of neurotic or reactive depression, with little documentation of the outcome or the cost. That practice has raised serious questions in some quarters. We need to bear in mind that definitive answers are not yet available regarding the value of ECT in depressive reactions that are often easily treated by skilled psychotherapy and/or medication.

MYTHS OF ECT

In Dr. Fink's rich commentary on ECT he points out that we do, indeed, know enough about ECT to state that the electric shock and convulsion per se are not essential for the treatment to be successful and that the length of the cerebral seizure has been shown to be related to the effectiveness of the treatment. Neither electric current nor a convulsion is central to the therapeutic process. Dr. Fink emphasizes, too, that we now also know that memory impairment is not essential for improvement and that ECT is not merely palliative in the treatment of depression.

He refers to the unknown biochemical changes that accompany and persist after ECT and offers the interesting conjecture that the central action of ECT is to elicit prolonged diencephalic stimulation. He points out that hypothalamic products affect behaviors related to the syndrome of depression. He also makes the interesting suggestion that in schizophrenia, ECT appears to have a related but different focus of action.

The clinical efficacy of ECT in schizophrenia appears to be less, and the number of seizures needed to effect a change, greater. He challenges the notion that the ECT process is unitary for all conditions, suggesting that it varies from one diagnostic entity to another.

These assertions and hypotheses are interesting, and they carry two important practical implications. The first is the need to examine the cost/benefit ratio in the consideration of a proposed treatment, and the second is the need for further studies on the outcome of treatment where the cost/benefit ratio is unknown because of insufficient data. We know that for selected populations, ECT has a cost/benefit ratio at least as good as that of other treatments. Those selected groups include patients with psychotic depressive states and those with acute schizophrenic symptoms in need of control. In all other conditions, including the use of ECT with children and adolescents, in maintenance treatment, in neurotic depressive states, and in chronic schizophrenia, the data are either minimal or insufficient. There is a cost to memory, reversible in the great majority of patients but apparently persistent, at least subjectively, in some. The data also show that subsequent seizures might be kindled in a small percentage of patients receiving ECT.

Under these circumstances, decisions to administer ECT cannot be taken lightly, even when no alternative treatment is available. When the effectiveness of ECT is less certain, or when it is used experimentally, it should be acknowledged as such and no effort spared to study the outcome.

Now, how can we communicate the complexity of these concepts to patients and relatives as we struggle with the subtleties of informed consent?

ECT AND ETHICAL PSYCHIATRY

Dr. Salzman has captured the absurdity of a system that demands informed consent from those who, although not disturbed enough to warrant involuntary admission to a hospital, are not well enough to understand the full implications of the procedure they are consenting to. Under these circumstances, fully informed consent is impossible. One could dwell for hours on the pros and cons of the concept of informed consent, but our task takes us directly to Dr. Salzman's two questions: 1) Does a psychiatrist have the right to do more than offer treatment? 2) What is the psychiatrist's role with a patient whose ability to make health care decisions may be compromised by the symptoms for which the treatment is indicated?

Our Hippocratic Oath demands, on the one hand, that any regimen we adopt shall be for the benefit of those who consult us; but the law, on the other hand, has been increasingly vocal in prescribing limits to our authority to do so. I think our responsibilities are nevertheless still clear. Regarding Dr. Salzman's questions, I believe we must do more than merely offer or suggest the treatment we believe to be the most effective. If we have any serious question about the patient's ability to understand the complexities of the situation, whether he be voluntarily or involuntarily admitted to a hospital, we have a duty to move the matter into the courtroom for the decision to be made there. I agree that the courts must decide on the competence of the patient to understand the issues. However, I am not as sanguine as Stone (11) and Salzman that the courts should be central to clinical decisions regarding diagnosis and treatment.

In discussing what he considers to be abuses of ECT, Dr. Salzman reports that several states have been prompted to enact regulations to control the practice of ECT. He believes such regulations to be useful and proper. I believe that while regulations are appropriate and, at times, indicated, legislative statutes have no place in the practice of medicine. They are subject to the whims and bargaining that are part of the political process and readily become rigid, resisting the modifications that are demanded by the discovery of new knowledge. However, local regulations are in a different category; government or semigovernment bodies that grant licenses to treatment facilities may occasionally have to help maintain standards of clinical practice, and at times it appears necessary that they do so. The Massachusetts Department of Mental Health, as a result of a task force report requested by then Commissioner Milton Greenblatt, developed a set of regulations that placed a ceiling on the number of sessions of ECT that could be administered to a patient within one year. Patients considered to be in need of additional sessions had to be examined by consultants and reported to the commissioner's office. Statistics attest to the fact that those regulations were capable of influencing clinical practice in the desired direction (12).

Perhaps we should be relieved that lawyers, legislators, and administrators are so eager to lighten our burden by making decisions for us. Would they were the wise ones to whom we could defer. However, their record over the past decade persuades me that systems developed to deal with patients not capable of informed consent are likely to fall wide of the mark if the sifting of clinical evidence is in the hands of nonclinicians. In my view, the clinical interests of the mentally ill will suffer irreparably unless the input of experienced and well-informed clinicians remains the pivotal point of the clinical decision.

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Medical Histories of Delinquent and Nondelinquent Children: An Epidemiological Study

BY DOROTHY OTNOW LEWIS, M.D., AND SHELLEY S. SHANOK, M.P.H.

The authors studied the hospital records of 109 delinquent children and a matched comparison group of 109 nondelinquent children. The number, reasons for, and timing of hospital contacts significantly differentiated the delinquent from the nondelinquent children; perinatal difficulties did not. The authors hypothesize that an interaction of developmental factors and parental inadequacies accounts for the clustering of hospital contacts before age 4 and between ages 14 and 16 in the delinquent sample. They discuss the implications of their findings for prevention.

THE PURPOSE of this paper is to report the striking differences we found between delinquent and nondelinquent children with respect to the number, quality, and timing of medical problems. We will look at the relationship of accidents, injuries, and illnesses throughout childhood to social maladjustment. We will examine the emphasis in the literature on perinatal factors in the etiology of deviance and explore the importance of later medical events.

This study sprang from the clinical observation that many of the children referred to a juvenile court clinic had experienced multiple accidents, injuries, and illnesses. They had been hit by cars, fallen from roofs, and suffered serious physical trauma at the hands of relatives and peers. From a clinical standpoint, some of these injuries and illnesses seemed to have contributed to the children's inability to form appropriate judgments, assess reality, and control their behavior.

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They had made frequent visits to the emergency room and clinics of one of the general hospitals in the community. On the other hand, relatively few of these children, according to their parents, had been the products of difficult pregnancies or deliveries, and almost all were reported to have had normal developmental milestones. Because we recognized that histories based on parental recollection alone were possibly unreliable we saw the need to study the question of medical histories more systematically.

THE LITERATURE

There is evidence that perinatal factors may have long-term effects on the adjustment of children (1–4). Whatever the limitations and questions raised by studies of perinatal effects, together they comprise an impressive body of knowledge. Although some authors have reported an association between neurological disease and delinquency (1, 5), those who have looked at the later medical status of delinquent children have reported few immediately obvious serious medical disorders (6, 7).

A few investigators, although they recognized that only a small percentage of delinquent children show flamboyant evidence of physical disorders, have speculated on the possible behavioral effects of what may appear to be minor physical disorders (8, 9). Gibbens reported that, although obviously serious head injuries such as encephalitis or skull fracture had occurred in only 1%–2% of the delinquents he studied, many delinquents did have histories of having sustained what seemed at the time of accident to be minor head injuries. Unwilling to dismiss these apparently trivial injuries, Gibbens speculated that "the effects of these minor head injuries and of special handicaps are very difficult to assess" (9, p. 1,087).

In studies of hospital records the psychological effects of separation have often been emphasized rather

TABLE 1
Hospital Use by Delinquent and Nondelinquent Children Through Age 16

Hospital Service Used	Contacts of Delinquent Children			Contacts of Nondelinquent Children			Analysis*	
	Mean	Median	Range	Mean	Median	Range	t	Significance
Emergency room	4.029	2	0–33	2.398	1	0–29	2.987	p=.004
Clinic	5.733	2	0–68	3.096	0	0–43	2.411	p=.017
Ward	0.476	0	0–9	0.223	0	0–3	2.295	p=.023
Total	10.286	5	0–87	5.204	1	0–48	2.987	p=.004

*By two-tailed t test.

TABLE 2
Specific Medical Problems of Delinquent and Nondelinquent Children*

Specific Medical Problem	Delinquent Children		Nondelinquent Children		Analysis	
	Number	Percent	Number	Percent	Chi Square	Significance
Accident or injury	66	61.0	45	41.7	6.928	p=.009
Head or face trauma	39	35.9	24	21.6	4.475	p=.035
Perinatal difficulty	12	10.9	16	14.3	0.238	p>.500
Psychiatric symptom	19	17.1	9	8.0	3.085	p=.080
Child abuse	9	8.6	1	1.0	6.328	p=.012

*A number of children had more than one medical problem.

than the other possible meanings of hospital contacts (10, 11). The present study looks beyond questions of separation and uses hospital data to examine a possible interaction of medical, developmental, and familial factors contributing to delinquent behavior.

METHOD

Two groups of children were chosen for study: 1) a random sample of 109 delinquent children referred to the juvenile court but not to the court clinic and 2) a sample of 109 nondelinquent children from the New Haven area with demographic characteristics similar to those of the delinquent population. We decided to use a random sample of delinquent children instead of those referred to the court clinic (on whom our initial clinical observations were made) because of the likelihood that a clinic-referred sample would be biased toward physical pathology as well as psychopathology and therefore would not be typical of the general court population (12).

Although the juvenile court records did not always allow for accurate socioeconomic class assignment, it was generally recognized by court and clinic staffs that the great majority of delinquents were from the lower socioeconomic classes in the area. Thus the nondelinquent sample was chosen from a population of which 75% would have been classified as coming from classes IV and V of the general population according to the Hollingshead and Redlich scale (13).

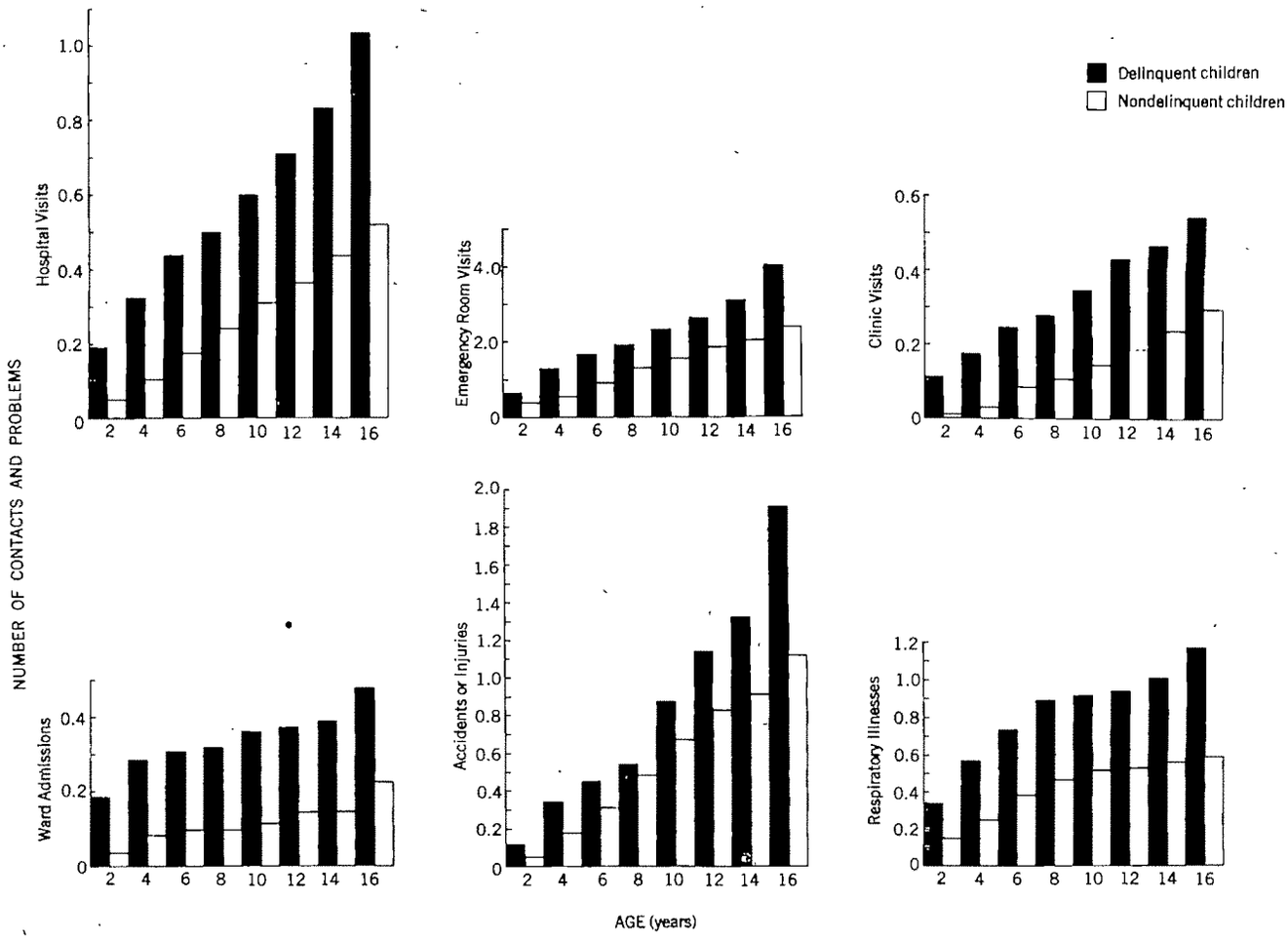
Each of the 109 delinquent children was matched with a comparison child for age, sex, and race. Two-thirds of each sample were white; the other third were black. Two-thirds of each sample were boys; the other third were girls. At the time of the study the average age of both groups was 19 years. Because of the legal and ethical limitations on access to data about siblings of the index patients, it was not possible to include a comparison group of nondelinquent siblings.

ASSESSMENT OF MEDICAL STATUS

An evaluation of the medical history of each child in the study was made on the basis of an extensive review of the child's hospital record. Our choice to limit the study to one large general hospital raises the issue of case loss. Although it would have been desirable to interview parents and children regarding medical histories, such personal contact was prohibited by the court. However, our own experience as well as that of other investigators (14) suggests that, although personal histories can be valuable clinically, they are notoriously unreliable regarding numbers of illnesses and dates of medical events, information essential to this study.

Data from other public and private medical facilities were unavailable because of issues of confidentiality; however, as far as we could determine, our two populations had equal access to other community resources, making it unlikely that one group made greater use of such clinics than the other. Furthermore, serious accidents and illnesses would have most likely been referred to the hospital. The choice of private medical care was relatively rare in both groups be-

FIGURE 1
Cumulative Hospital Contacts and Medical Problems of Delinquent and Nondelinquent Children Throughout Childhood



cause of financial considerations. At the time of study, the hospital selected tended to be used by the less affluent in the community as a source of primary care. In fact, the hospital subsequently developed a primary care center to meet the needs of the many patients who used the emergency room and clinics in this way. Finally, case loss would create underestimates of the actual prevalence of disorders and use of medical facilities in both groups.

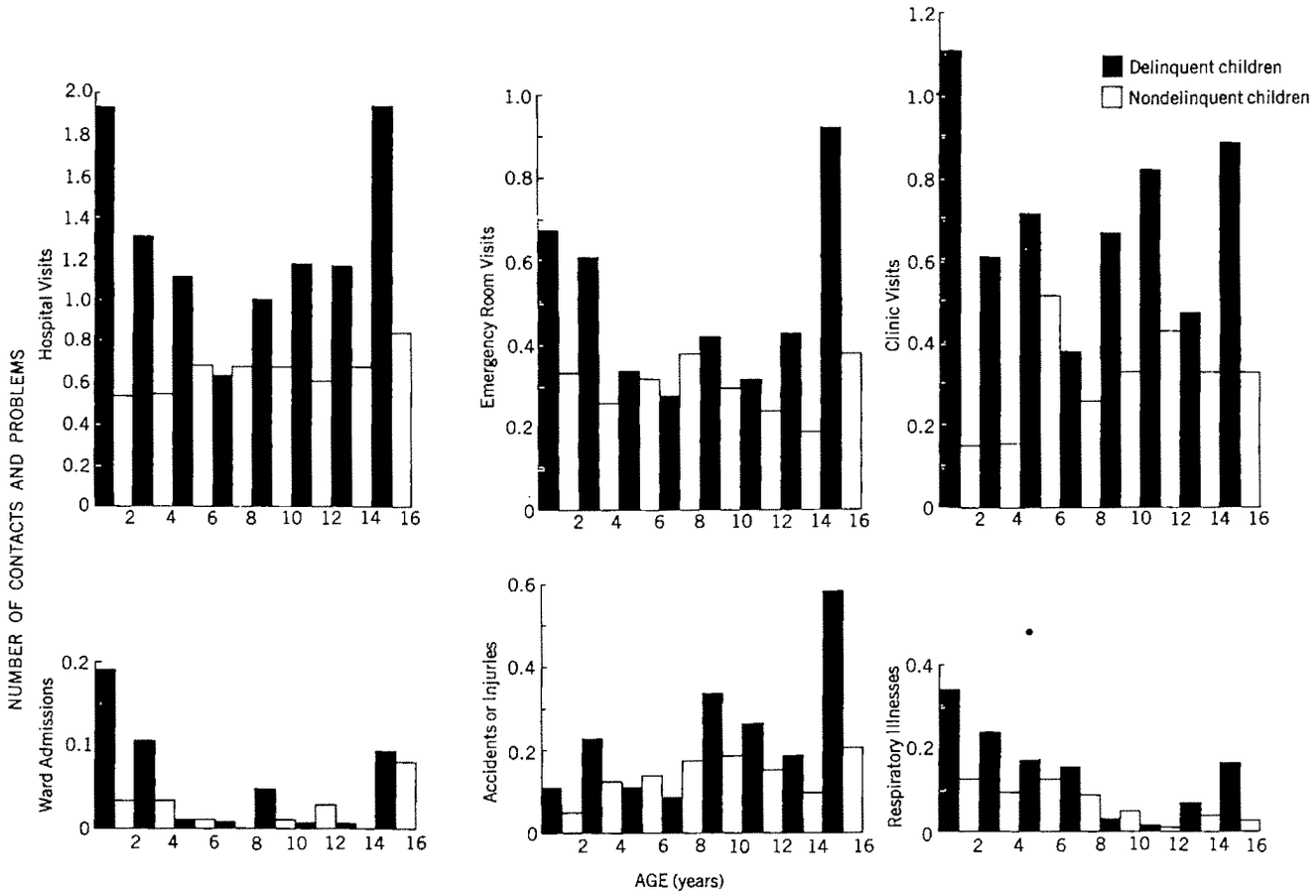
Medical records were assessed in terms of numbers of visits to the hospital, timing of visits, use made of different hospital services (e.g., emergency room, clinics, and wards), and reasons for visits (e.g., accidents, head or face trauma, respiratory illnesses, child abuse, and psychiatric problems).

FINDINGS

The use of hospital facilities, the kinds of disorders treated, and the longitudinal picture of medical contacts are shown in tables 1 and 2 and in figures 1 and 2. As shown in table 1, delinquent children made significantly greater use of all hospital facilities than did their nondelinquent counterparts. Table 2 indicates that although delinquent children were far more likely than nondelinquent children to be seen for accidents (particularly head or face trauma) and to demonstrate psychiatric disturbance, they were no more likely than nondelinquent children to have suffered from perinatal difficulties. The timing of hospital contacts was of particular interest. As shown in figure 1 and table 3, delinquent children made significantly more hospital visits than did nondelinquent children. Differences between the two groups were most striking before age 4 and between ages 14 and 16.

When the above data were further analyzed in terms of medical events occurring within two-year intervals (see figure 2 and table 4), the years 0–2, 2–4, and 14–16 were the age intervals in which delinquent children differed most significantly from their nondelinquent peers in terms of overall hospital use. Figure 2 shows clearly the increased hospital use by delinquent children in early childhood and in adolescence in contrast to the nondelinquent group, which had relatively constant use that seemed unaffected by age.

FIGURE 2
Two-Year Interval Data on Hospital Contacts and Medical Problems of Delinquent and Nondelinquent Children



DISCUSSION

The results of this study suggest that delinquent children have significantly more hospital contacts and significantly more accidents and injuries throughout childhood than do nondelinquent children. However, to our surprise we found that delinquent children were no more likely to have experienced perinatal trauma. It was subsequent medical events that clearly distinguished one group from the other. It would seem, therefore, that medical problems throughout childhood rather than perinatal problems are associated with juvenile delinquency.

The particular clustering of medical problems during the early years (0–2 and 2–4 years) and at the onset of adolescence (14–16 years) suggests that a particular developmental factor may be operating. Both periods have in common increased physical (motor) capacities and an imbalance between inner behavioral controls and heightened impulses. In normal families the parents of very young children usually provide appropriate external controls, protecting and supporting the child until the child develops his/her own internal controls. If the parents, for any reason, are unable to provide this structure, there is a greater likelihood that immature impulsive behavior will result in frequent accidents and injuries. The parents of delinquent children are often unable to provide adequate support and protection because of their own psychopathology and the adverse social situation in which they live. The evidence for psychopathology in parents of delinquent children has been documented (7, 15, 16) and is made evident in this study by the high prevalence of child abuse in the delinquent sample.

Early accidents and injuries, possibly a consequence of parental inadequacies, may contribute to CNS dysfunction, which, in turn, further aggravates the child's difficulties with impulse control and social adaptation. The disappearance of striking differences in hospital contacts between the ages of 4 and 14 may be partially accounted for by the development of better internal controls during this period and the support offered by a school setting. Between the ages of 5 and 14 most children are in a school setting that offers protection and structure several hours each day.

With the onset of adolescence the school becomes a less effective force. Many adolescent delinquent children who have been held back over the years attend school only sporadically or drop out entirely. The burden of guidance, control, support, and protection once more falls on the shoulders of the parents. Unfortunately, the continuing inadequacy of the parents again

TABLE 3
Results of Two-Tailed t Tests of Cumulative Hospital Contacts and Medical Problems of Delinquent Versus Nondelinquent Children

Item	Age of Child (years)							
	2	4	6	8	10	12	14	16
Hospital visits								
t	2.462	3.001	2.610	2.296	2.345	2.615	2.518	2.787
Significance	p=.015	p=.004	p=.010	p=.023	p=.020	p=.010	p=.013	p=.004
Emergency room visits								
t	1.757	2.366	2.023	1.548	1.588	1.578	1.787	2.411
Significance	p=.081	p=.019	p=.045	p=.124	p=.114	p=.117	p=.076	p=.017
Clinic visits								
t	2.067	2.354	2.102	2.039	2.237	2.253	2.170	2.913
Significance	p=.041	p=.020	p=.037	p=.043	p=.027	p=.026	p=.032	p=.053
Ward admissions								
t	2.625	2.675	2.355	2.429	2.636	2.215	2.370	2.295
Significance	p=.010	p=.009	p=.020	p=.017	p=.010	p=.028	p=.019	p=.023
Accidents or injuries								
t	1.545	1.718	1.136	0.370	0.972	1.290	1.513	2.449
Significance	p=.124	p=.088	p=.258	p>.500	p=.332	p=.199	p=.132	p=.016
Respiratory illnesses								
t	1.458	1.632	1.222	1.238	1.161	1.184	1.273	1.620
Significance	p=.147	p=.105	p=.224	p=.217	p=.248	p=.238	p=.205	p=.107

TABLE 4
Results of Two-Tailed t Tests of Two-Year Interval Data on Hospital Contacts and Medical Problems of Delinquent Versus Nondelinquent Children

Item	Age of Child (Two-Year Intervals)							
	0–2	2–4	4–6	6–8	8–10	10–12	12–14	14–16
Hospital visits								
t	2.462	2.266	0.913	0.191	1.405	0.908	1.035	2.685
Significance	p=.015	p=.025	p=.363	p>.500	p=.162	p=.365	p=.302	p=.008
Emergency room visits								
t	1.757	2.209	0.431	0.899	1.070	0.489	1.165	2.754
Significance	p=.081	p=.029	p>.500	p=.370	p=.286	p>.500	p=.246	p=.007
Clinic visits								
t	2.067	2.206	0.656	0.789	1.819	0.747	0.632	2.158
Significance	p=.041	p=.029	p>.500	p=.431	p=.071	p=.456	p>.500	p=.033
Ward admissions								
t	2.626	1.402	0.009	0.990	1.125	0.722	0.226	0.330
Significance	p=.010	p=.163	p>.500	p=.324	p=.262	p=.472	p>.500	p>.500
Accidents or injuries								
t	1.545	1.267	0.287	1.474	1.617	1.397	1.080	2.717
Significance	p=.124	p=.207	p>.500	p=.142	p=.108	p=.164	p=.282	p=.008
Respiratory illnesses								
t	1.458	1.611	0.367	0.917	0.665	0.434	0.930	2.545
Significance	p=.147	p=.109	p>.500	p=.360	p>.500	p>.500	p=.354	p=.012

renders them ineffective when left to cope with their adolescent children. At the same time the adolescent's heightened impulses and increased physical strength, along with access to weapons, cars, alcohol, and drugs, make his/her impulsive behaviors most injurious to himself/herself and to others. This may partially account for the increased use of the hospital between the ages of 14 and 16 compared with the earlier years.

These findings have certain implications for the prevention of serious delinquency. A history of frequent hospital contacts for a variety of different reasons and in the absence of a single medical disorder (e.g., asthma) should alert the physician to the possibility of associated delinquent behavior and severe intrafamilial disturbance. Rather than treat each hospital contact as an isolated medical event, a frequent practice in busy emergency rooms and clinics, physicians should recognize the pattern of repeated hospital contacts for many different kinds of disorders as a possible concomitant of delinquency and as an indication for a thorough medicopsychosocial evaluation in the interest of preventing or at least curtailing maladaptive antisocial behavior.

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Coexisting Childhood Schizophrenia and Myasthenia Gravis Treated Successfully with Neostigmine Bromide

BY ROBERT C. SCHNACKENBERG, M.D., AND GEORGE HOLMES, PH.D.

A 12-year-old boy who had suffered from severe schizophrenia for 8 years developed myasthenia gravis. Both disorders were treated simultaneously and successfully with neostigmine bromide. The authors suggest that these two disorders may in some cases be part of the same disease process and urge that physicians consider this possibility in patients with severe emotional problems and muscle weakness.

BIOLOGICAL factors have long been felt to contribute to the etiology of childhood schizophrenia (1–3). A child who had suffered from the symptoms of childhood schizophrenia for eight years and then developed myasthenia gravis improved dramatically after he was placed on a regimen of 15 mg of neostigmine bromide, administered orally four times a day.

Heath and Guerrero-Figueroa (4) found that the introduction of acetylcholine into the septal region of psychotic patients resulted in pleasurable feelings and alertness. However, when certain anticholinergic agents, especially atropine, were introduced into the septal region, they induced focal spiking or slow waves or both at this site. The EEGs resembled those of psychotic patients and rhesus monkeys that have received injections of taraxein fractions.

Currently, the basic abnormality in myasthenia gravis is thought to be a reduction of acetylcholine receptors at neuromuscular junctions (5). This reduction is thought to result from an autoimmune attack directed against receptors (6). Extracts of thymic tissue have been shown to contain acetylcholine receptors (7) similar to those in skeletal muscle (8). This suggests that an autoimmune reaction against acetylcholine receptors might be initiated within the thymus gland itself.

Apparently, faulty acetylcholine metabolism is involved in both of these disorders. This raises the interesting question as to the possible relationship between childhood schizophrenia and myasthenia gravis and

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whether neostigmine bromide should be tried with other children who suffer from this disorder and have been refractory to other forms of treatment.

The odds against childhood schizophrenia and myasthenia gravis occurring in a 12-year-old boy are approximately two-hundred million to one. The concurrence may be greater. Several symptoms of these disorders overlap. When concurrence occurs, psychiatrists may diagnose the disorder as childhood schizophrenia and pediatricians as juvenile myasthenia gravis.

In the case reported in this paper, informed consent was obtained from the patient, his guardian, and both parents.

CASE REPORT

A 12-year-old boy was admitted to a residential treatment center for emotionally disturbed children in September 1969. His referring psychiatrist stated that he was "involved in a dream world and detached from reality." He did not relate to peers, had many compulsive rituals, talked to himself, had an imaginary playmate, made inappropriate facial patterns, had screaming temper tantrums when he did not get his way, had difficulty in expressing himself verbally, and needed to touch people when he talked to them. He was generally withdrawn, deficient in eye-to-eye contact, and spoke in a low tone of voice.

The patient's birth weight was 3.5 kg following a normal gestation period. However, respiratory difficulty was encountered after birth and required caffeine treatment. He was in an oxygen tent for his first 24 hours. Early development was delayed; the patient began saying simple words at age 3 and sentences at 6½. His first steps were at age 2, but he could not walk well until he was 5 years old. In contrast to these developmental delays, he could count to 10 when he was 4 years old. At age 4, he was evaluated by mental health and speech and hearing professionals, who felt he had both aphasia and childhood schizophrenia.

His parents, who had divorced in 1968, have both had multiple psychiatric hospitalizations. His mother, who is 40, has been given the diagnosis of schizophrenic reaction, simple type, and his father, schizophrenic reaction, paranoid type. Both parents were hospitalized for the first time when the patient was aged 4–6. The father was discharged from the Air Force with a 100% psychiatric disability after hospitalizations in 1962, 1963, and 1964. Both parents were cared for by their own parents, although intellectually they appear to be at least average.

The patient is the second of four siblings. An older brother died in an automobile accident in 1966, a younger brother was living in a home for orphans, and his sister was living with his mother and maternal grandmother.

In 1965 the mental health agency considered trying to move the patient and his brother away from the parents' pathological influence. In 1968 his younger brother was placed in the home for orphans because of the pathological family environment, but the patient was felt to be "almost hopeless and in extreme need of residential treatment."

Psychological testing from 1963–1967 revealed an IQ fluctuating between 82 and 91 (based on the Stanford-Binet Intelligence Scale and the Wechsler Intelligence Scale for Children [WISC]).

In his first month of residential treatment, he compulsively loved to keep score for the other boys and would not participate in their activities. His school performance was commensurate with his age, but his approach to learning was idiosyncratic and he would not take instruction from the special education teacher.

His therapeutic program consisted of special education; individual and group psychotherapy; 2 grains of thyroid extract per day for his chronic hypothyroidism (serum cholesterol and T_3 and T_4 determinations have been normal); and specific work on his sense of body image, motor coordination, and time and spatial concepts. In March 1970 an inappropriate posturing of his left arm was noted, and he explained that "bad things would happen if he moved his left arm normally." In June 1970 a progress note stated that he had made "minute progress." He was noted to be more assertive and less fearful of his peers and performed better in the group situations, with less withdrawal to sleep during activities. At school he still displayed a rigid approach to learning and was poor in oral and written language expression and abstract thinking. His mother and maternal grandmother, who had been worked with throughout the year, seemed to be less overprotective than in the past.

After the July vacation month the patient returned to the residential treatment center in August 1970 with some shifting of symptoms. His posture was more slumped, with his chin resting on his chest. His speech was slurred and barely audible, and he complained of difficulty in swallowing solid foods. In October he was noted to tire easily, and his extraocular muscles also became affected. At this time the diagnosis of myasthenia gravis was made, and he was placed on 15 mg of neostigmine bromide four times a day. His posture normalized, his speech became more articulate, he became more alert, eye muscle function normalized, and swallowing improved. However, the most noteworthy improvement was his increased ability to communicate and relate to people, his new-found assertiveness, and the subjective feeling of several people who examined him that he had become more emotionally related to other people. (Neostigmine ordinarily penetrates the blood-brain barrier poorly [9]. However, the usual penetration of 15% may be sufficient or may increase under pathophysiological conditions.)

The patient continued to make excellent progress, and he was discharged from the residential treatment center in 1972. He has resided in a children's home since that time because his parents are too emotionally

ill to care for him. He has shown no signs of schizophrenia since 1970 and would best be described as an overly perfectionistic or obsessive-compulsive personality, a frequent residual of recovered childhood schizophrenia as noted by Bender (10, p. 215).

Psychological assessment preceding the patient's successful treatment with neostigmine bromide revealed a

> 12-year-old male who behaviorally and from psychological testing manifests both organic and emotional problems. The organic manifestations are in the area of generalized restlessness, poor concentration-attention ability and perseveration. Emotional features, probably developed as a means of compensating for the organic problems, are compulsive rituals, anxieties, and at times lability and expansiveness of his affect.

The most recent intellectual assessment, in 1973, revealed a full-scale IQ of 96 (based on the WISC).

The patient has been followed at monthly outpatient intervals since 1972. During the 1972–1973 school year he was voted the "best-liked boy in his class" and obtained a B average. He was able to compete in sports with other boys his age, with his major difficulty being some residual social awkwardness. In April 1974 it was decided after neurological consultation to withdraw him from the neostigmine bromide; he was placed in the hospital where this was accomplished without difficulty. He continued to be followed at monthly intervals with no recurrence of childhood schizophrenia and myasthenia gravis. His only medication was 2 grains of thyroid extract per day. (It should be noted that hypothyroidism frequently accompanies myasthenia gravis.)

We offer the following speculations as to why the symptoms did not recur with the removal of neostigmine.

1. Both disorders occasionally remit spontaneously during adolescence (11, p. 668; 12).
2. The predisposition to allergic reactivity changes as a function of age (13, p. 13).

DISCUSSION

Strauss and associates (14), using fluorescent antibody techniques, demonstrated the presence of globulin on the A-band striations of affected muscles of patients with myasthenia gravis. Lindstrom and associates (6), Aharonov and associates (7), and Kao and Drachman (8) have shown that skeletal muscle cells in the thymus may initiate an autoimmune attack, reducing available acetylcholine receptors at neuromuscular junctions.

The studies of myasthenia gravis by Strauss and associates (14) led Heath and Krupp (15) to their immunological hypothesis of schizophrenia. Heath and associates (15,16) postulated that taraxein might represent an antibody capable of binding itself to specific antigen sites in septal tissues of the brain to cause an anticholinergic effect similar to that on the muscles of patients with myasthenia gravis. Heath and Guerrero-Figueroa (4) noted a schizophrenic-like psychosis caused by impairment of acetylcholine activity in the septal region. In addition, Heath and Krupp (15) found that control sera from a patient with myasthenia gravis induced psychosis in a monkey.

Inherited factors may be significant, since Pirskanen (17) found a genetic association between myasthenia gravis and the human leukocyte antigen system. HLA-8, coded on autosome number 6, is associated with neurovegetative lability as well as autoimmune and endocrine disorders (17).

This supports the hypothesis that a subgroup of schizophrenia may be a central autoimmune disorder, which in some cases may be related to myasthenia gravis, a peripheral autoimmune disorder.

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Pituitary Thyrotropin Response to Thyrotropin-Releasing Hormone in Affective Illness: Relationship to Spinal Fluid Amine Metabolites

BY PHILIP W. GOLD, M.D., FREDERICK K. GOODWIN, M.D., THOMAS WEHR, M.D., AND ROBERT REBAR, M.D.

The authors studied pituitary thyrotropin, i.e., thyroid-stimulating hormone (TSH), response to thyrotropin-releasing hormone (TRH) in patients with primary affective disorder. There were no overall differences between either depressed or manic patients and normal controls; however, the TSH response was significantly lower in the unipolar depressed patients than in either bipolar depressed patients or normal subjects. Bipolar patients in the manic phase tended to have a lower response than bipolar depressed patients. In the unipolar group, the TSH response showed a significant negative correlation with the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA) in the CSF. These neuroendocrine responses may constitute markers of specific monoamine dysfunction in subgroups of patients with affective illness.

SEVERAL ASPECTS of the symptom complex of affective illness, such as disturbances in sleep, altered energy levels, changes in appetite and libido, diurnal variation in symptoms, and amenorrhea, suggest hypothalamic dysfunction. The abnormalities of neuroendocrine response seen in affective illness have been thought to be biological representations of this hypothalamic dysfunction. Interest in the hypothalamic-pituitary axis has been further stimulated by recent findings that the monoamine neurotransmitters thought to play an important role in the pathogenesis of affective illness modulate the synthesis and release of a number of hypothalamic peptides and pituitary hormones (1). Thus, examination of pituitary hormones in plasma can shed light on the functional activity of biogenic amine systems. Moreover, these peptides and hormones may have behavioral effects or influence the metabolism of biogenic amines or other central nervous system regulators (2). The recognition of this crucial relationship between central nervous system function and neuroendocrine secretory systems provides the basis for new methods for investigating the biological basis of affective illness.

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METHOD

Our subjects were 23 patients with primary affective disorder, diagnosed independently by two psychiatrists according to the criteria of Spitzer and associates (3), and 10 normal volunteers, all hospitalized on clinical research units at the National Institutes of Health. The depressed patients were subdivided into unipolar and bipolar groups based on the absence or presence, respectively, of a history of mania and/or hypomania (3). All patients adhered to a controlled monoamine diet and received no medications for 3 weeks before the study. After an overnight fast, a 19-gauge scalp vein was inserted into an antecubital vein and kept open with a slow drip of normal saline. Baseline thyroid-stimulating hormone (TSH) levels were determined 15 minutes before and just before the infusion of 500 μg of synthetic thyrotropin-releasing hormone (TRH), given as an I.V. bolus over 30 seconds. Following the TRH infusion, blood was drawn every 15 minutes for at least 1 hour and analyzed for TSH. The TSH response to TRH represented the difference between the maximum TSH increment following TRH infusion and the average of the baseline TSH values. TRH was assayed by the double antibody technique. The intra- and interassay coefficients of variation were 6.0% and 14.0%, respectively. The limit of assay sensitivity was 2.0 μU/ml.

The major metabolites of serotonin, norepinephrine, and dopamine in the central nervous system (5-hydroxyindoleacetic acid [5-HIAA], 3-methoxy-4-hydroxyphenyl glycol, and homovanillic acid [HVA], respectively) were analyzed in cerebrospinal fluid (CSF) after 18 hours' pretreatment with probenecid (100 mg/kg), a drug that blocks the egress of both 5-HIAA and HVA out of the central nervous system, resulting in their accumulation in the CSF. CSF was collected, stored, and analyzed as previously described (4). The lumbar punctures and the TRH infusions were performed on separate days but under similar drug-free conditions and during the same clinical state, i.e.

mania or depression. When some of the patients were given lithium and then retested, the lithium level was maintained between 0.8 and 1.2 mEq/liter. Cycling premenopausal women were tested only during the low estrogen, early follicular phase of the cycle (5).

RESULTS

There was no significant difference in baseline plasma T_3, T_4, or TSH between any of the subgroups tested. When the TSH response to TRH was examined, no relationship was found to age, sex, baseline plasma T_3, T_4, or TSH, either in the overall groups of patients or within any subgroup.

No significant difference was noted when the TSH response in the overall group of depressed patients was compared with the TSH response in control subjects. However, the unipolar patients showed a significantly lower TSH response compared with bipolar patients ($p<.01$) and controls ($p<.01$) (see figure 1). The 9 bipolar patients in the manic phase tended to have a lower response than the 7 in the depressed phase (8.82 ± 1.44 μU/ml versus 12.31 ± 2.21 μU/ml; $p<.10$, Student's t test). Chronic lithium treatment significantly increased the TSH response ($p<.01$, paired t test); this change was accompanied by a significant decrease in T_4 ($p<.05$, paired t test).

As illustrated in figure 2, a significant negative correlation was found in the 6 unipolar depressed patients on whom lumbar punctures had been performed between the probenecid-induced accumulation of 5-HIAA in the CSF and the TSH response ($r=.89$, $p<.05$). There were no other significant correlations between amine metabolites in CSF and the TSH response, either overall or in the various subgroups tested.

Two unipolar patients with blunted TSH responses in depression were studied in the drug-free state following recovery; in each the TSH response returned to normal. In one of these patients, CSF 5-HIAA was also obtained during depression and following recovery; in the recovered state, the 5-HIAA fell approximately 50% in apparent association with the normalization of the TSH response.

FIGURE 1
TSH Response to TRH in Depressed Patients and Controls

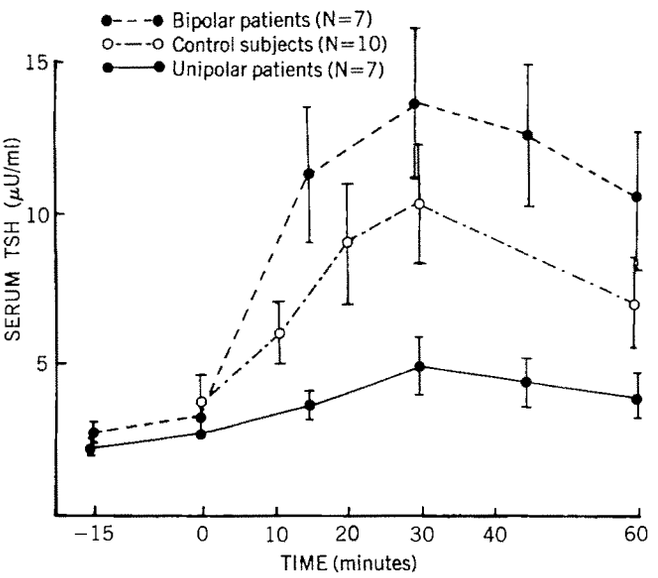
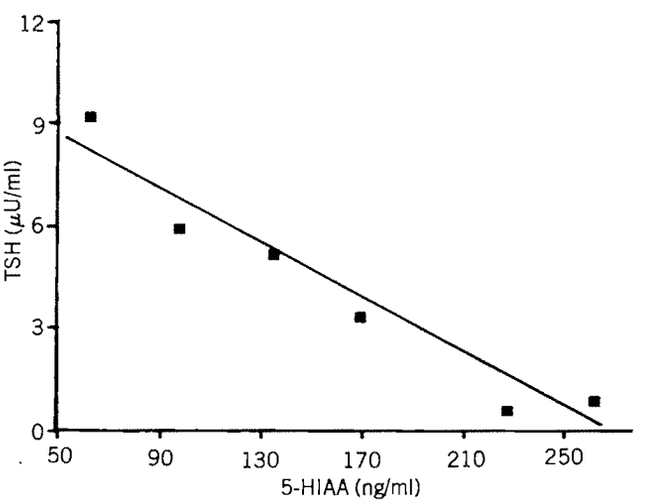


FIGURE 2
Correlation Between Age, Corrected CSF 5-HIAA, and Difference in TSH Values in 6 Unipolar Patients*



*r=.89; p<.05.

DISCUSSION

Factors that might be associated with a blunted TSH response include increased T_3 and T_4 (6), drugs such as aspirin (7), prolonged fasting (8), and elevated glucocorticoids (9). On the other hand, elevated TSH response can be associated with decreases in T_3 or T_4 (10) and elevated estrogen of either endogenous or exogenous origin (4); thus, studies in normal subjects indicate that the TSH response is slightly higher in women than in men and is somewhat higher in women during the midmenstrual cycle compared with the early follicular phase (4). In addition, alterations in the sleep-wake cycle or in the level of endogenous TRH could also affect the TSH response.

A number of these factors can be excluded from influencing the findings reported here. Patients received no drugs during the study, and menstruating women were tested only during the low estrogen phase of the cycle. Since there are more women in the unipolar group than in the bipolar group, unequal sex distribution cannot account for the lower responses in the unipolar patients.

The sleep disturbances seen in manic and depressed patients could also affect the TSH response, since TSH levels tend to be high during waking hours and to fall rapidly after the onset of sleep (11). However,

there was no relationship between TSH response and hours of sleep. Evaluation of the role of endogenous TRH levels on the TSH response awaits a clinically applicable TRH assay.

Glucocorticoids have been reported to be elevated in unipolar depressed patients (12), a factor that could account for the lower TSH responses in this group. However, it has also been recently reported that there is no significant difference in glucocorticoid levels between unipolar and bipolar depressed patients (12, 13), so elevated glucocorticoids may not be the governing factor in the lower TSH response of unipolar depressed patients. Alternatively, elevated glucocorticoids and the lower TSH response could each be markers for an underlying abnormality in neurotransmitter function in unipolar depressed patients.

Because the TSH response can be considerably magnified by even small, nonsignificant decrements in T_3 and T_4 (10), the fact that there were no significant differences in the level of circulating thyroid hormones among the various groups of subjects does not exclude thyroid hormone levels as a factor in this study. Thus, the antithyroid effects of lithium, which might persist for several weeks after discontinuation of the drug, could result in a greater TSH response in patients who had received previous lithium therapy during the course of their illness. Although none of the subjects received any drugs for at least 3 weeks prior to study, 5 of 7 bipolar and only 1 of 7 unipolar patients had received lithium therapy earlier in the course of their illness, a factor that could contribute to the difference in TSH response between these groups. The possible contribution of previous lithium therapy to TRH studies in affective illness requires further evaluation.

Although some bipolar patients have been included in previous reports of blunted TSH responses in depression (14–19), the question of differences in the mean response between unipolar and bipolar groups was not addressed. The factor of previous lithium therapy may play a role in our results; however, the unipolar-bipolar difference noted here is also consistent with a variety of clinical, genetic, pharmacological, and biological data (20) which suggest that these subgroups may represent different illnesses.

It is generally accepted that the hypothalamic-pituitary-thyroid (HPT) axis is regulated by TRH, which is produced within the central nervous system in the area of the hypothalamus. The cells that synthesize and release TRH possess the ability to respond to activation and inactivation by monoaminergic neurotransmitters (1). The TSH response to TRH is also modulated by alterations in the functional activity of monoaminergic neurotransmitter systems, presumably at the level of the pituitary (1). Thus, it is at the level of both the hypothalamus and the pituitary that monoaminergic neurotransmitters regulate HPT function and affect the TSH response to TRH. Although it has been assumed that CSF 5-HIAA primarily originates in neurons with a paraventricular origin, the TSH to CSF 5-HIAA correlation suggests that CSF 5-HIAA might reflect the activity of serotonergic systems in the hypothalamus or pituitary; recent anatomic studies suggest substantial interconnectedness between the hypothalamic-pituitary axis and paraventricular and other brain regions via axonal and portal transport systems and the CSF communication pathway (21).

The monoaminergic control of the synthesis and release of TRH itself is unclear. It is known, however, that in experimental animals (22, 23) and in man (24–26), both dopaminergic and serotonergic stimulation inhibit the TSH release and response to a variety of stimuli. Thus, the blunted TSH response in unipolar depressed patients may be associated with increased serotonergic or dopaminergic activity, perhaps particularly the former, in light of the significant negative correlation between the TSH response and CSF 5-HIAA. Viewed in this way, the TSH response data would be consistent with a hypothesis of increased functional serotonergic activity in unipolar depression. The previously reported effect of lithium treatment on TSH response (27) was confirmed in this study. Although it has been assumed that this effect is secondary to the antithyroid effect of lithium, an additional factor might involve direct effects of lithium on the serotonergic system (28).

Other neuroendocrine work from this laboratory is also compatible with a hypothesis of increased serotonergic activity in some severely depressed unipolar patients. We recently reported that the 24-hour urinary free cortisol production was significantly elevated in a group of unipolar depressed patients compared with control subjects (13) and that the degree of elevation correlated positively with the CSF 5-HIAA. This finding is in agreement with data from both experimental animals and man, indicating that serotonin may be a neurotransmitter that drives the hypothalamic-pituitary-adrenal axis (29, 30). Elevated glucocorticoids and a blunted TSH response may thus each reflect increased functional serotonergic activity in some unipolar depressed patients.

The tendency for a greater TSH response in bipolar depressed patients compared with bipolar patients in the manic phase is also of interest and could reflect alterations in the functional activity of dopaminergic systems in bipolar depressed patients. We have recently reformulated a "dopamine hypothesis of affective illness," integrating these neuroendocrine data with relevant metabolite and pharmacological response findings (31).

The concept of biochemical heterogeneity in the affective disorders has recently been given support by reports of apparently bimodal distributions of amine metabolites (32, 33) and by evidence that separable amine metabolite subgroups may show differential responses to different antidepressant drugs (33). Work is now under way to determine if TSH response and other neuroendocrine measures may also be useful in further delineating the question of biochemical heterogeneity and its relationship to diagnostic subgroups and pharmacological response.

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Tardive Dyskinesia in Young Adults

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The authors describe two cases of tardive dyskinesia in which severe axial dystonia and intense facial grimacing produced marked discomfort as well as social and physical disability. Both patients experienced the onset of psychiatric symptoms as young adults, showed a prompt response to antipsychotic drug therapy, and were subsequently left on maintenance treatment for indefinite periods. The severity of this frequently irreversible and disabling condition warrants careful consideration in the use of long-term antipsychotic drug treatment in the young psychiatric outpatient population.

IT IS well known that the chronic use of antipsychotic drugs may lead to the appearance of a persistent, involuntary movement disorder known as tardive dyskinesia (1, 2). This syndrome has been reported in patients exposed to virtually all phenothiazines and occasionally a thioxanthene (3) or butyrophenone (4). Although many cases occur in institutionalized patients with chronic psychiatric disorders, the syndrome also appears in psychiatric outpatients and nonpsychotic patients treated with phenothiazines for psychoneurosis, chronic gastrointestinal disturbances, chronic pain, and personality disorders (5). Although a relationship with organic brain disease was emphasized in early reports, subsequent studies have demonstrated no significant association with previous brain damage, ECT, or frontal leukotomy (2). Although there appears to be an increased risk for tardive dyskinesia in patients older than age 50 (6), the condition is by no means restricted to the elderly; it occurs in middle-aged and young adults as well. The purpose of this communication is to report the appearance of a remarkably severe and disabling form of tardive dyskinesia in two young men and to thereby emphasize the risks inherent in the long-term treatment of young individuals with antipsychotic drugs.

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CASE REPORTS

Case 1. A 23-year-old enlisted man was admitted to a military hospital in August 1972 for depression, delusions, flight of ideas, and inappropriate affect. There was no family history of neurological or psychiatric disorder. Neurological examination, laboratory studies, skull X rays, brain scan, and EEG were normal. A diagnosis of acute undifferentiated schizophrenia was made, and he was treated with 20–30 mg of trifluoperazine daily, with rapid improvement. Within 2 months he was free of evident thought disorder and was discharged from military service. He was followed in a Veterans Administration hospital psychiatric outpatient facility where he remained free of psychiatric symptoms and was maintained on 30 mg of trifluoperazine daily. In October 1973, 300 mg of thioridazine daily was substituted for the trifluoperazine. In March 1974 it was noted for the first time that the patient had choreoathetosis and dystonia involving the left arm, shoulder, and mouth. Thioridazine was discontinued and 8 mg of trihexyphenidyl daily was administered. The dyskinesias worsened and became associated with marked motor restlessness, diaphoresis, excessive thirst, and a 40-pound weight loss. Trihexyphenidyl was discontinued and the patient was referred to the neurology service of the Boston VA Hospital in May 1974.

The findings from a general examination were normal except for diaphoresis and a pulse rate of 120–140 during periods of physical hyperactivity. There was generalized motor restlessness; continuous facial grimacing; intermittent blepharospasm; spasmodic contractions of the tongue, jaw, and facial muscles; torticollis and retrocollis; athetotic movements of the hands; and abnormal posturing of the arms in abduction and extension (more severe on the left than the right side). The patient frequently assumed bizarre positions in order to abort the dyskinesias, such as crouching in bed on all fours with the top of his head pushed against a pillow or standing with his forehead pressed forcibly against a wall. The other findings from the neurological examination were normal. Findings from routine laboratory studies, serum copper and ceruloplasmin, urinary copper, serological studies, calcium, and thyroid function studies were normal. Slit-lamp examination showed no Kayser-Fleischer rings. Skull X rays, EEG, brain scan, and cerebrospinal fluid were normal. A psychiatric consultant found no evidence of active thought or affective disorder.

Eight mg of haloperidol daily produced prompt and complete abolition of all dyskinesias except for mild posturing of the left arm while the patient was walking. Because of concern over the long-term effect of continued exposure to anti-

psychotic drugs, haloperidol was discontinued after several weeks, with a prompt recurrence of all dyskinesias. Intravenous diphenhydramine, benztropine, and physostigmine and oral diphenhydramine, dantrolene, propanolol, and deanol had no effect. Severe depression ensued, and a regimen of 8 mg of haloperidol daily was resumed, with a virtual disappearance of all dyskinesias within several days. During the next 3 months the patient exhibited only mild posturing of the left arm and pursing movements of the lips. However, mild drug-induced parkinsonism appeared for the first time, and the patient became increasingly depressed and paranoid. In February 1975 the patient committed suicide. There was no postmortem examination.

Case 2. A 19-year-old musician began exhibiting severe obsessive-compulsive, ritualistic behavior requiring psychiatric hospitalization in 1966. An underlying schizophrenic process was suspected, and the patient was treated with 20–40 mg of trifluoperazine daily, with resolution of symptoms within 4 weeks. He continued this medication and led a normally active professional and social life until May 1974. At that time he reported that involuntary eye blinking had been present for 2 years and arching of the torso and restless movements of the arms and legs for 2 months. While sitting in his car he would repeatedly arch his torso and forcibly press the back of his head into the seat. Trifluoperazine was discontinued, and he was referred to the psychiatric outpatient department of Massachusetts General Hospital. Examination disclosed continuous, large amplitude dystonic movements and postures of the neck and torso. His gait was characterized by an exaggerated lordotic posture of the torso and forward thrusting movements of the abdomen and hips. There was frequent eye blinking, blepharospasm, choreoathetotic hand movements, vermicular tongue movements, and repeated stereotyped caressing movements of his hands to his face and moustache. He denied any subjective compulsion to perform the movements. The other findings from the neurological examination were normal.

Family history included schizophrenia in one sister and obsessive behavior in the mother; there was no family history of neurological disorder. Findings from routine laboratory studies, serum copper and ceruloplasmin, thyroid function studies, calcium, skull X rays, EEG, brain scan, liver scan, and slit-lamp examination were normal.

Treatment with trihexyphenidyl, diphenhydramine, diazepam, 2 mg of haloperidol daily, and 4 g of L-dopa daily had no significant effect. Three grams of deanol daily appeared to reduce the involuntary movements, but because of a marked increase in obsessive symptoms deanol was discontinued; there was no significant increase in dyskinesia. When the patient was hospitalized in February 1975 he displayed obsessive-compulsive signs and symptoms, auditory hallucinations, paranoid delusions, a thought disorder, and the persistent dyskinesia. A regimen of 60 mg of haloperidol daily was begun, with improvement in the patient's psychiatric symptoms and a significant reduction in the dyskinesia. He is currently followed as an outpatient; both the dyskinesia and psychiatric symptoms persist.

DISCUSSION

These two individuals display a number of features in common. Both manifested the onset of psychiatric symptoms as young adults, showed a prompt and satisfactory response to trifluoperazine, were left on maintenance treatment, and subsequently enjoyed an interval free of psychiatric symptoms. The subsequent insidious appearance of involuntary movements during treatment with phenothiazines, exacerbation of dyskinesia following drug withdrawal, and subsequent suppression with haloperidol are all quite typical of tardive dyskinesia (2). Insofar as possible, other neurological disorders have been excluded. The motor behavior of both individuals was dominated by large amplitude, bizarre, often grotesque dystonic postures of the torso and extremities. These postures were extremely uncomfortable, caused intense embarrassment, and virtually precluded satisfactory social relationships and useful employment.

Tardive dyskinesia is virtually unique in the remarkable variety of hyperkinetic involuntary movements that may appear, including orofacial dyskinesia, facial tics, grimacing, chorea, athetosis, dystonia, torticollis, and motor restlessness (2). Although orofacial dyskinesia is probably the best known feature of the syndrome and often appears first in older patients, involuntary movements of the extremities and torso are usually apparent as well.

In younger individuals involvement of the extremities and torso is often more prominent and may include unusual postures of hyperextension and abduction of the arms, ballistic movements, exaggerated and lordotic postures of the trunk, shoulder shrugging, and rotatory pelvic movements. A number of studies (7–10) have reported several striking examples of tardive dyskinesia in patients below age 50, with marked axial involvement including retrocollis and opisthotonic arching of the back; some of the cases strongly resembled our own. A syndrome resembling tardive dyskinesia has also been reported in children following the withdrawal of antipsychotic drugs; the involuntary movements primarily involve the extremities, torso, and, only rarely, the mouth (11). The effect of age on the topographic distribution of tardive dyskinesia therefore bears some resemblance to phenothiazine-induced acute dystonic reactions and idiopathic torsion dystonia (dystonia musculorum deformans), in which children and adolescents tend to suffer generalized axial involvement while adults often manifest more localized face and neck involvement (12, 13).

It is sometimes stated that patients with tardive dyskinesia are relatively unaware of and undisturbed by their movements. In our experience, however, this is more characteristic of patients with coexisting chronic psychosis or dementia. As we have emphasized in this report, young individuals, who often manifest generalized dyskinesias, suffer obvious discomfort, embarrassment, and social and physical disability as a result of the disorder.

The management and treatment of tardive dyskinesia remain particularly difficult problems and have been reviewed in detail elsewhere (2). Prevention must be considered of paramount importance. The use of long-term antipsychotic drug therapy in the treat-

ment of conditions responsive to other modes of therapy should be discouraged. In addition, the decision to maintain patients on long-term drug therapy following a single acute schizophrenic reaction must be weighed with particular care. Patients should be examined at regular intervals for early features of dyskinesia such as vermicular tongue movements, facial tics, particularly frequent eye blinking, and subtle athetotic finger movements. Drug "holidays" have also been recommended in order to reduce drug exposure, unmask latent dyskinesia, and assess the need for continued medication (1, 2). Unfortunately, the patients described in this report are not unique and, in fact, are only two of several young individuals with extensive and disabling tardive dyskinesia whom we have seen in recent years. We have presented detailed descriptions of their disorder with the aim of reemphasizing the gravity of the condition, particularly when it appears in the young psychiatric outpatient population.

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CLINICAL AND RESEARCH REPORTS

This section contains reports of new clinical or laboratory observations, single cases of unusual interest or importance, descriptions of pilot studies, suggestions for future research, etc. Reports should be submitted in duplicate, typed double spaced throughout, and may contain 1,000 words, no more than 1 table, and a maximum of 10 pertinent references. Figures cannot be used. Reports will be reviewed by at least one referee and the Editor; the author will receive a postcard informing him/her of the disposition of the submission. Reports will be edited for clarity and conformance with Journal *style.*

Juvenile Manic-Depressive Illness

BY JAMES H. WHITE, M.D., AND GREG O'SHANICK, M.D.

Although manic-depressive illness is described as a disease of adulthood, Kraepelin (1) observed that 0.4% of his manic-depressive patients had evidence of sporadic mania before the age of 10. In 1960, Anthony and Scott (2) formulated the following criteria for diagnosis of childhood manic-depressive illness: 1) an abnormal psychiatric state similar to the classical description; 2) a family history of manic-depressive disorders; 3) an early tendency for cyclothymic mood with increasing severity and length of mood swings, and delirious manic or depressive outbursts occurring during febrile illness; 4) recurrent or periodic mood disorder (at least two episodes); 5) diphasic moods showing swings of pathological dimension; 6) minimal influence of environment on symptoms; 7) severe symptoms requiring inpatient treatment; 8) an extroverted personality; and 9) absence of features of schizophrenic or organic states.

Psychogenic etiologies have not been proved, and psychogenic features are less apparent after the affective disorder is controlled with lithium (3–6). Weinberg and Brumback (7) noted a positive correlation with hyperactivity in three documented cases. An X-linked genetic factor has also been proposed (8).

Case Report

The patient was a 15-year-old boy who was hospitalized in an acutely agitated state with incoherent speech.

On admission he displayed markedly grandiose delusions, flight of ideas, loose associations, and general agitation. He claimed to have taken amphetamines, barbiturates, LSD, and marijuana. He was very disoriented; he claimed that it was January 1961, that he was in "God's world," that he had nothing to worry about, and that he, in fact, was God's son. He talked to God constantly and claimed to be able to see him.

The physical and neurological examinations were normal, as were laboratory studies done at admission. Drug screens were negative. His initial EEG displayed one brief paroxysm of epileptiform activity, but a repeat tracing was normal.

The patient was an identical twin; both the other twin and the mother showed cyclothymic personalities. The father was subject to recurring psychotic depressions. Affective disorders were also present in the paternal grandfather, the paternal grandmother, and several paternal aunts who were diagnosed as schizo-affective. His early development was mildly delayed, and he was diagnosed as being hyperactive at age 6 and treated with stimulant drugs without effect. On two occasions he was hospitalized for behavior control, with no change in his extreme hyperactivity and impulsiveness. His IQ was borderline. Throughout his childhood, he was noted to have marked mood swings unrelated to environmental events. Behavior problems at home and school continued, culminating in a series of runaway episodes that led to this admission.

Phenothiazines were tried, but the patient developed erythema multiforme. Haloperidol was substituted to control the patient's agitation. Lithium carbonate was started at an initial dose of 1800 mg/day until a serum lithium level of 1.0 mEq/liter was reached. The dosage was then decreased to 1200 mg/day. A marked improvement was noted in the patient's behavior after 4 days on lithium: his psychomotor agitation decreased, delusions of grandeur disappeared,

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thought processes were more coherent, and orientation was appropriate. Supportive psychotherapy was initiated at this time.

The patient developed a second episode of erythema multiforme, which resolved when the haloperidol was stopped. Four weeks after his initial improvement with lithium treatment, he became markedly depressed and showed psychomotor retardation. The lithium was discontinued, but 1 week later manic symptoms reappeared, with gross flight of ideas, psychomotor agitation, and grandiose auditory and visual hallucinations. Lithium was started again; oral fluphenazine was added to control his agitation while his serum lithium level was increasing to therapeutic levels. He had no evidence of allergic reactions to fluphenazine. Over a period of 3–5 days, his agitation, affect, and thought processes improved. He was discharged 2 weeks later to outpatient follow-up. Two weeks after discharge, the fluphenazine was stopped without a recurrence of symptoms. The patient has reentered a special school program and lives with his mother and stepfather. He continues to take lithium carbonate, 1200 mg/day, and is seen in supportive outpatient psychotherapy. There has been no further evidence of manic or depressive symptoms.

Discussion

Previous reports of children and young adolescents eventually diagnosed as manic-depressive indicate that they were often originally misdiagnosed as hyperactive. However, these children fail to respond to stimulant drug therapy in the way the hyperactive child usually does (3, 7).

This case is presented to emphasize the fact that manic-depressive illness can be seen very early in life. It often resembles the hyperactive child syndrome initially, but closer investigation and a more thorough history reveal significant differences. As the child enters adolescence, the symptoms more closely resemble the adult form of manic-depressive illness. Early recognition is important because these patients respond to lithium but not to other treatments.

In the child or young adolescent with increased psychomotor activity, marked mood swings, and a positive family history of affective disorders, manic-depressive illness should be considered. A favorable response to lithium does not remove the need for psychotherapy to help the patient understand and cope with his illness. Psychotherapy is also necessary to help the individual deal with the interpersonal problems associated with a life-long illness.

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Transcultural Attitudes Toward Antisocial Behavior: Opinions of Mental Health Professionals

BY JAMES M.A. WEISS, M.D., AND MARGARET E. PERRY, M.S.

Antisocial and criminal behaviors are complex phenomena often associated with psychiatric disorders (1). However, labeling theory, a major new direction in crime research, supports the view that deviant characteristics reflect the obverse of the norms and values of the larger community, which defines the rules by which certain behaviors come to be considered aberrant (2). There is evidence that mental health professionals may be influenced by cultural definitions of aberrant behavior (3, 4). However, these public norms, values, and attitudes are generally implicit, and little qualitative research has been done in this area.

For these reasons, we have developed a series of continuing projects concerned with public attitudes toward crime and antisocial behavior in various cultures and subcultures. In the first three studies (5–7), we found that people in urban areas scattered throughout the world are most concerned about crimes of violence (especially homicide), theft, exploitation, and public dishonesty. Other kinds of antisocial behavior apparently are seen as important crimes by only relatively small groups within the various populations studied. More people endorse a traditional, moralistic, punitive approach to social control of crime than a preventive-rehabilitative approach, although a majority wants an organized, problem-solving approach. Significant differences among respondents in perceptions of what

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constitute the most serious crimes are related primarily to geographic location, but differences in defining strategies for control are related to social class, educational, and experience variables: upper-class individuals and experts tend to endorse preventive and rehabilitative strategies. The present study will describe such variances in opinion among mental health professionals.

Method

Most studies of public opinion involve quantitative rather than qualitative approaches. However, by using Thompson's schema for the measurement of qualitative attributes (8), Stephenson (9) developed formal models for investigation in different cultural settings of public opinion regarding complex problems to determine in-depth, authentic patterns of opinion and to test the invariance of the factors.

We modified Stephenson's design to obtain a balanced sample of empirically derived representative opinions by using small-sampling techniques. This design involved a stratified quasi-balanced factorial sampling of 30–35 subjects in each of eight large cities (Athens, Bombay, Dublin, Istanbul, London, Paris, St. Louis, and Tokyo). The procedure allows us to classify responses by age, sex, socioeconomic status, and degree of expertise and/or involvement of the respondents in each city, yielding a universe of 2,603 different statements of opinion.

These statements then were subjected to content analysis and categorized into various subsets. The data were examined by an analysis of variance approximation and a centroid factor analysis. The requirement for statistically significant differences was a p value of .05 or less; p values of .50 or more were considered as indications of a high probability of agreement among the various demographic subgroups. Further details of method and category definition can be found in previous papers (6, 7, 10).

Results

Among the 258 respondents in our sample, there were 41 mental health professionals (23 psychiatrists, 7 psychologists, 6 nurses, and 5 social workers) distributed randomly throughout the eight sample cities. Approximately two-thirds of this group were in the middle age range (30–59); two-thirds were male. All of the mental health professionals (except 1 nurse in this sample) were upper class by our definition. Most psychiatrists and psychologists were categorized as "experts," while most nurses and social workers were classified as "informed."

The first noteworthy finding was the high level of agreement among the subjects in these eight very different kinds of cities. The commonality of attitudes toward the "most serious crimes" was high among all respondents, but among the various subgroups of mental health professionals there was substantial agreement ($p>.50$) in 39 out of 48 possible rating categories—there was agreement between mental health professionals and other respondents in 32 out of 48 categories. Mental health professionals, however, ranked the offense of "perceived sexual misbehavior *not* involving violence" significantly lower in terms of seriousness than did other respondents. Mental health professionals in Tokyo ranked nonlethal, nonsexual violence as significantly higher in terms of seriousness than did other respondents, as did those in Athens public dishonesty. Mental health professionals in Paris ranked theft significantly lower as a "worst crime" than did other respondents.

In terms of "what to do about crime," there were greater differences between mental health professionals and other respondents. Almost two-thirds of the total group of respondents endorsed an organized, problem-solving strategy; all mental health professionals except 1 psychologist in Bombay strongly endorsed this orientation. Holding the demographic variables of geographic location, age, sex, socioeconomic status, and degree of expertise constant, mental health professionals showed significantly higher loadings on what might be called the "public health" factor, i.e., an approach involving general social programs to prevent or reduce antisocial behavior (directly or indirectly), more information and research, and (especially) treatment, rehabilitation, and/or reeducation processes. Judgmental, pessimistic, and distorted responses were significantly less common among mental health professionals. Psychiatrists and psychologists showed significantly lower adjusted loadings on the "traditional-moralistic-punitive" factor, although nurses as a group significantly overendorsed traditional values and a concern for retributive justice. Mental health professionals also significantly overendorsed the otherwise low-ranked concern for humaneness per se. General social programs of prevention were favored significantly more often by mental health professionals in St. Louis and significantly less often by those in Bombay and Tokyo.

Discussion

In terms of ranking the "most serious crimes," mental health professionals throughout the world tend to have attitudes similar to those of other persons in their geographic location: like the majority of respondents, they are most concerned about crimes of violence, theft, exploitation, and public dishonesty. Perceived strategies for social control of crime, however, involve variables that are related primarily to education, occupation, and degree of involvement and/or expertise. But even more than other respondents of the upper socioeconomic and expert classes, mental health professionals tend toward a strong emphasis on an organized, problem-solving approach, including research, programs of prevention and defense, and treatment and rehabilitation. Data from previous studies indicate that persons from lower socioeconomic classes are most likely to hold negative attitudes toward rehabilitation and treatment procedures, although they have the highest rates of conviction for crime. Since mental

health professionals are most likely to endorse nonpunitive preventive and therapeutic strategies, they must expect antagonism, then, not only from the offenders themselves but from the larger body of lower and middle-class, uninformed, and uninvolved persons who endorse more traditional, moralistic approaches. A majority of people, however, especially mental health professionals, wants an organized, step-by-step problem-solving strategy. Our previous data clearly show that those people closest to the action most want more facts, more careful analysis, and more knowledge before proceeding with action programs. To help provide such information and research data on which to base a problem-solving strategy, perhaps more psychiatrists must join the group most emphasizing that need—those who are involved.

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Anticholinergic Psychosis in a Bilingual: A Case Study

BY EDWARD LASKI, M.D., PH.D., AND ELISABETH TALEPOROS, PH.D.

There has been an increasing number of reports concerning "atropine psychosis" or "anticholinergic delirium" following either intentional or accidental ingestion of psychotropic medications with marked parasympatholytic properties (1). The physician must always be aware of this possibility in patients being treated with tricyclic antidepressants, neuroleptics, antiparkinsonian agents, and especially the combinations of these drugs that are common in psychiatric treatment. The signs and symptoms of this toxic state may vary dramatically from obvious peripheral and autonomic features (mydriasis, xerostomia, hyperpyrexia, flushing, and tachycardia) to a florid anticholinergic syndrome with hallucinations, psychotic behavior, impaired sensorium with marked confusion, and, ultimately, coma and death. Proper treatment with a cholinesterase inhibitor such as physostigmine salicylate, which is capable of reversing both peripheral and central anticholinergic effects, has often dramatic and sometimes lifesaving results.

We would like to describe the case of a bilingual schizophrenic patient who presented with clear signs of such a delirious psychosis. His symptoms in the toxic state included the sudden development of auditory hallucinations in his native language. Although it has been noted that stress or disease can be related to language regression, the aspect of psychopathological bilingualism discussed here has rarely been commented on in the literature, and only a few recent case reports have emphasized its importance (2–4).

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Case Report

The patient, a 13-year-old Hispanic male, was admitted to the adolescent psychiatric ward of Bellevue Hospital on petition by the New York City Bureau of Child Welfare. Charges of physical and sexual abuse had been brought against the patient's 45-year-old father, an unemployed alcoholic. The patient, the second oldest of seven siblings, had come to New York City from Puerto Rico 4 years previously.

Before this hospitalization, the patient had been referred to a psychiatric clinic at another hospital because of constant "acting out" in school (truancy and fighting with peers and teachers). He was in psychotherapy and diazepam was prescribed. However, he became more anxious and guarded, and during the week before his admission to Bellevue he threatened to take all of his pills. He admitted that his father, while intoxicated, had forced him to perform "certain sexual acts" one night. The treating psychiatrist

thought the boy was frankly psychotic and insisted on hospitalizing him.

On admission to our service, the patient's appearance was that of a handsome, frightened young man who made minimal eye contact and kept picking at his fingernails. His command of English was good, but his speech was extremely guarded. His affect was constricted in range and depth but appropriate to the situation. His mood was anxious and depressed. The patient admitted having auditory hallucinations in English: he said that a devil was calling him "faggot" and saying "I'm going to get you." He thought he heard people around him calling his name. His sensorium was clear and there was no history of alcohol or drug abuse. Results of the medical examination were within normal limits.

The patient was diagnosed as having a schizophrenic reaction, paranoid type. Treatment was begun with chlorpromazine, and after 2 weeks he was on a maintenance dose of 600 mg p.o. h.s. His hallucinations had subsided entirely. Because of moderate cogwheel rigidity, benztropine mesylate, 2 mg b.i.d. and h.s., was added during the third week. After 2 days, the night nursing staff noticed that the patient was restless, had difficulty sleeping, and mumbled to himself. The next day he was wandering deliriously around the ward. He was disoriented, hallucinating, and spoke only in Spanish; he described hearing a devil telling him, "Yo te voy a coger" (I'm going to get you).

Physical examination revealed that his pupils were mydriatic and poorly reactive to light; he had dry skin, a normal temperature, mild hyperreflexia, diminished bowel sounds, and tachycardia (120 beats/minute). An ECG was started and 0.5 mg of physostigmine salicylate was administered subcutaneously three times in 30 minutes. His sinus rate returned to 80 per minute, and his bowel sounds increased. The patient dramatically reverted to English, describing his hallucinations vividly. He complained of the urge to defecate and of mild nausea. His delirium subsided. He was kept drug-free for the next 24 hours and was then maintained on chlorpromazine, 300 mg h.s., without any recurrence of his symptoms.

Discussion

Bilingualism and its relation to psychopathology have received little attention in the general research or clinical literature. Psychological studies of bilingualism have been concerned primarily with implications for cognitive functioning and learning (5). Most psychiatric studies of language in pathology have been limited to discussions of the content of schizophrenic language and its relation to thought disorders (6). The case we have described highlights the potential importance of combined efforts in these relatively unallied areas of investigation.

In one of the few attempts to combine psychiatric observation with research on bilingualism, Hemphill (7) described auditory hallucinations in South African polyglots. True polyglots, who are equally proficient in at least two languages, reported auditory hallucinations in the first language learned (the primary or mother tongue). However, those polyglots whose hallucinations appeared to be drug induced reported auditory hallucinations in all languages known to them. Hemphill contrasts the hallucinations of true polyglots with those of "refugee" polyglots who emigrated and were forced to learn a new language. Their hallucinations (generally of a persecutory nature) were reported only in the second language, and often were denied by the patients when they were questioned in their primary tongue.

Marcos and associates (8) compared clinical judgments of the severity of psychopathology when patients were interviewed in Spanish, their primary tongue, and when they were interviewed in English, their second language. Although no specific differences were found for hallucinations per se, the estimates of total psychopathology were greater when patients were interviewed in English. This finding suggests that these patients were probably refugee polyglots.

The case we have reported presents a somewhat different problem for interpretation. The patient behaved differently at different times; the language he used to describe his hallucinations depended on circumstance. Like refugee polyglots, this patient initially had persecutory hallucinations in his second language. As he improved, his hallucinations subsided and he seemed relatively free of schizophrenic symptoms. Shortly before his discharge he developed a toxic reaction to medication, and while the content of the hallucinations remained the same, they appeared and were reported only in Spanish.

It is possible that this patient lost some of his adaptive coping mechanisms as a result of the drug-induced psychotic state. His delirium is consistent with a picture of temporary loss of recently learned material, including perceptions of his immediate environment and ability to communicate in his relatively new language. Once the toxic reaction had abated, the patient's general adaptive mechanisms were restored, and he returned to his normal level of functioning in his second language.

An alternative explanation is that the patient experienced two different states of arousal. Less vigilance and a lowered level of arousal in the toxic state may account for his reversion to his mother tongue.

The findings presented indicate the need for further investigation and additional documentation of this kind of clinical observation. It is not possible to design controlled experiments, but clinical observations of this type may provide researchers in the fields of bilingualism and psychopathology with directions for answering basic questions in their individual areas, as well as with potential links between them. This clinical phenomenon has implications for those studying the relation of bilingualism to brain function, memory storage, and information processing, as well as to

those interested in linguistic and paralinguistic aspects of psychiatric phenomena.

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Do Tricyclic Antidepressants Enhance Adrenergic Transmission?

BY ALAN FRAZER, PH.D., AND JOE MENDELS, M.D.

It has been suggested repeatedly that the tricyclic antidepressants exert their effects by blocking the neuronal reuptake of biogenic amines (norepinephrine and/or serotonin), thereby increasing the amount of neurotransmitter available to act at postsynaptic receptors and overcoming a hypothesized amine deficiency. Most of the evidence for this theory has been derived from studies of the acute pharmacological actions of these drugs, either after a single injection or by examination of in vitro effects. Since it is widely believed that the tricyclic compounds do not exert a significant therapeutic effect until they have been administered for 10–14 days or longer, it is surprising that relatively little attention has been directed toward their pharmacological actions after chronic administration. The available information suggests that tricyclics initiate effects that are different from, and perhaps opposite to, their acute effects. As is discussed below, such effects may be compensatory changes to the acute pharmacological actions of tricyclics.

In the course of a continuing series of studies dealing with this question (1), we have noted a late-developing pharmacological action of desmethylimipramine (DMI) that is relevant to the functional activity of the noradrenergic system.

Male Sprague-Dawley rats (200–300 g) were given intraperitoneal injections of DMI (5 mg/kg) once daily for either 4, 11, or 18 days. Control animals received intraperitoneal injections of isotonic saline solution for corresponding time periods. Twenty-four hours after the last injection of the series, the rats were decapitated. Procedures used for preparation of cerebral cortical slices, labeling with ^{3}H-adenine (10μCi/ml), and determination of ^{3}H-adenosine 3′,5′-monophosphate (cAMP) in the slices have been reported previously (1). Data from cortical slices of the three groups of saline-injected rats were not significantly different and have therefore been combined to form a single control group.

From table 1, it can be seen that DMI treatment did not significantly alter the basal accumulation of cAMP in cerebral cortical slices. In contrast, DMI treatment significantly reduced the net synthesis of cAMP stimulated by norepinephrine (NE) when the treatment lasted for more than 4 days.

As noted above, enhancement of adrenergic or serotonergic neuronal activity, as a consequence of tricyclic-induced blockade of amine reuptake (2), is the pharmacological effect to which the therapeutic effect of tricyclics is most often ascribed. Based on this idea, we explored initially the effect of treatment with imipramine given for 5 days on NE-stimulated cAMP net synthesis in cerebral cortical slices (1). The expectation was that blocking amine reuptake would cause an exaggerated response (i.e., stimulation of cAMP) to NE. However, the response to NE was reduced. Similar observations have been reported by others (3, 4), and it is apparent the administration of tricyclics does, over time, produce a diminished responsiveness to NE. This diminished response produced by exogenous NE after *chronic* tricyclic treatment is the opposite of the effect that would be predicted on the basis of blockade of amine reuptake.

There are several other pharmacological effects of chronic tricyclic treatment that might attenuate the postulated enhancement of adrenergic transmission produced by these drugs. Segal and associates (5) reported that in rats treated with DMI for 8 days and killed 24 hours after the final injection, the activity of

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TABLE 1
Influence of DMI on Accumulation of cAMP in Cerebral Cortical Slices*

Drug Treatment	N	In Vitro Drug Addition: None Mean	None SE	None p**	Norepinephrine ($2x10^{-6}$ M) Mean	Norepinephrine ($2x10^{-6}$ M) SE	Norepinephrine ($2x10^{-6}$ M) p**
0.9% NaCl	15	0.58	0.04		2.96	0.20	
DMI, 4 days	5	0.65	0.09	> .4	2.73	0.34	> .5
DMI, 11 days	5	0.45	0.03	> .05	2.00	0.28	< .025
DMI, 18 days	6	0.49	0.04	> .2	2.10	0.17	< .025

*Results are expressed as the percent conversion of radioactivity into cAMP.
**Student's t test (two-tailed) for comparisons with results from saline-injected group.

brain tyrosine hydroxylase, the rate-limiting enzyme in catecholamine biosynthesis, was reduced significantly. This may account for the reports that chronic treatment with tricyclic drugs lowers brain concentrations of NE, whereas acute treatment has no such effect (6).

It would be incorrect to assume that the lowered concentration of brain NE observed after chronic treatment with tricyclic drugs necessarily indicates diminished activity of NE-containing neuronal systems, as there need not be a correlation between amine concentrations and functional effects. Therefore, studies of the effect of tricyclic treatment on the "turnover" of NE may provide a better correlation with activity in NE neurons. Acute administration of tricyclic drugs reduces amine turnover, but chronic treatment with imipramine in rats accelerated the disappearance of radioactive NE from brain (6). Schildkraut and associates (6) speculated that enhanced turnover of NE after chronic treatment with imipramine might result in greater availability of NE to adrenergic receptors despite reduced brain NE concentration. In other words, chronic tricyclic treatment would still result in enhanced adrenergic transmission.

An alternative interpretation of data on NE turnover arises from our studies of the effects of chronic tricyclic treatment on NE-induced responses in brain tissue. The diminished effects produced by NE in rats given tricyclic drugs chronically may be due to the development of adrenergic receptor subsensitivity. The development of receptor subsensitivity may be viewed as a compensatory change to the presence of sustained elevated concentrations of NE in the synaptic cleft—a consequence of DMI blocking amine reuptake. It is possible decreases in adrenergic receptor sensitivity lead to a second compensatory change, namely, an increase in amine turnover, in much the same way that drug-induced blockade of adrenergic receptors produces an enhancement of turnover in adrenergic neurons (7). If this interpretation is correct, then tricyclic-induced alterations in presynaptic neurotransmitter metabolism would be related inversely to activity at the postsynaptic receptor site. The point to be emphasized is how difficult it may be to distinguish primary pharmacological effects from secondary compensatory ones.

It is clear that the tricyclic drugs produce multiple effects at the adrenergic synapse, and it seems premature to assume that any one of these interrelated responses is in itself the crucial factor in mediating the therapeutic action of these drugs. At this stage we still do not know the net effect of chronic treatment with tricyclic drugs on adrenergic transmission. There are short-term effects that appear to enhance this activity, while some long-term effects may diminish it. The net effect remains unknown and must be resolved in future studies. It is intriguing to speculate that there may be an association between the long-term tricyclic-induced effects on adrenergic responses and the apparent delay in development of therapeutic response. The fact that both require time to develop may be coincidental or it may suggest an important relationship. Perhaps the "tolerance" and "rebound" effects on REM sleep seen with the tricyclic drugs (8) may reflect long-term changes in receptor sensitivity induced by these agents. Alternatively, the long-term changes in adrenergic responsiveness may be deleterious, serving to attenuate the results produced by the acute (therapeutic?) effect of the tricyclics on amine reuptake.

These crucial questions remain unanswered. However, it is clear that it is necessary to completely reevaluate the relationship between the clinical effects of the tricyclics and their actions on biogenic amine systems.

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Coincidental Improvement in Asthma During Lithium Treatment

BY SUHAYL J. NASR, M.D., AND ROBERT W. ATKINS, M.D.

The simultaneous occurrence of two illnesses in the same individual offers a unique opportunity to learn about both illnesses. It has long been suspected that emotional factors contribute to asthma, and in a recent review Knapp and associates (1) suggested a strong correlation between mood disturbances and asthmatic attacks. The two case reports below illustrate another facet of this correlation as well as unexpected improvement in asthma following administration of lithium carbonate for concurrent manic-depressive illness.

Case Reports

Case 1. The patient, a 39-year-old married woman with three children, began having psychotic episodes in 1969. She carried the diagnoses of chronic schizophrenia and schizo-affective schizophrenia and was treated with various phenothiazines and butyrophenones. She experienced several relapses on this regimen and was admitted to the Strong Memorial Hospital, Rochester, N.Y., in December 1975 and again in March 1976. At the time of her second admission, the staff diagnosed her as manic-depressive, manic phase, and started her on lithium carbonate. Since that time, the patient has experienced no recurrence of her psychotic symptoms and has resumed normal functioning. The patient spontaneously reported in November 1976 that she has not needed any antiasthmatic medication since she started on lithium carbonate. She also mentioned that her antiasthmatic medication, particularly the xanthines, previously precipitated those hypomanic episodes that led to her hospitalizations. The past year has been the first since 1969 that she has had no psychotic episodes and no asthmatic attacks. She has been on lithium carbonate, 600 mg b.i.d., and has received no other form of therapy. Her serum lithium level has ranged between 0.6 and 0.9 mEq/liter.

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The authors would like to acknowledge the assistance of Drs. Haroutun Babigian and Mark Proctor.

Case 2. The patient, a 65-year-old married man, began to have psychotic episodes in 1949. He has had four episodes since that time and has received varying diagnoses of acute psychotic episode, psychotic depressive reaction, manic-depressive, manic phase, and manic-depressive, depressive phase. His last admission was in March 1976, when he was started on lithium carbonate. The patient has also had asthma for several years and is severely allergic to iodine. He reports that since he began to take lithium he has not had any asthmatic attacks and has used his antiasthmatic medications only for his occasional episodes of wheezing. He is presently well maintained on lithium, 300 mg b.i.d., with serum levels ranging between 0.8 and 1.0 mEq/liter.

Discussion

These two cases illustrate the good response of concurrent manic-depressive illness and asthma to one pharmacological agent, lithium carbonate.

Patients with asthma experience acute recurrent exacerbations of their illness and are usually symptom-free in the intervening periods, which is also true of manic-depressive patients, although each episode is significantly longer than that in asthma. In both illnesses persistence of the symptoms over longer periods of time leads to significant reduction in the psychological, social, and medical functioning of the individual.

Tricyclic antidepressants have a well-established role in the relief of depression (2). They are also suspected of exerting a beneficial effect in asthma through their anticholinergic, antihistamine, and catecholamine-blocking properties (1). Amphetamines produce immediate and short-lasting relief of depression. Epinephrine plays a strikingly similar role in acute asthmatic attacks.

Xanthines, steroids, and epinephrine, the drugs of choice in asthma, act through increasing available cyclic adenosine monophosphate (cAMP) (3). Prostaglandins, particularly the balance between PGE_1 and $PGF_{2\alpha}$, have been suspected of playing an important role in the pathogenesis of asthma (4). Lithium has

been proven effective in the treatment and prophylaxis of manic-depressive illness (2). A variety of modes of interaction between lithium, prostaglandins, and cAMP have been described. Murphy and associates (5) described inhibition by lithium of the PGE_1-mediated increase in cAMP. Horrobin (6) suggested a modulation role for lithium in prolactin-induced prostaglandin synthesis. Lithium has also been shown to increase glucagon-stimulated cAMP excretion (7). We suggest that lithium modulates the balance between PGE_1 and $PGF_{2\alpha}$ and cAMP and cyclic guanosine 5′-monophosphate, thus aborting the clinical manifestations of asthma in our patients.

Conclusions

We have presented two unusual cases of concurrent asthma and manic-depressive illness responding well to a single agent, lithium. This report raises several questions about the interactions among lithium, prostaglandins, and cyclic nucleotides. Patients with asthma and manic-depressive illness provide a unique opportunity to study these interactions.

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Increased Palpebral Aperture in a Patient Receiving Amitriptyline

BY DAVID J. GOODE, M.D.

Exophthalmos has not been reported as a side effect of amitriptyline. This paper will report the case of a 57-year-old man being treated for depression with amitriptyline who had a markedly increased palpebral aperture that resembled exophthalmos, although there was no protrusion of the orbit. The phenomenon appeared 3 days after amitriptyline was started at a dose of 75 mg/day and disappeared within 2 days after the drug was discontinued. The increase in palpebral aperture was accompanied by a reported decrease in depressive symptoms. The similarity of this idiosyncratic side effect to inhibition of reserpine-induced ptosis in experimental animals, one of the primary catecholamine-related effects common to all antidepressant drugs, is striking.

Case Report

Mr. A, a 57-year-old divorced man, was seen at a local mental health center in April 1974, for somatic complaints related to a depressed state. Since November 1973, he had consulted numerous emergency rooms for chest and abdominal pain and had received several drugs (he did not remember what they were). During early 1974, he developed increased insomnia, loss of libido, frequent crying spells, feelings of sadness, and lost approximately 23 kg. He had no history of alcoholism, drug abuse, or psychiatric consultation, and there was no known history of depression or alcoholism among first-degree relatives. He was placed on thioridazine, 150 mg/day, at the time of initial consultation, but he experienced little relief. In June 1974, he was admitted to the inpatient psychiatry unit at the University of Chicago because of a suicidal gesture in which he inserted a knife into an electric socket but was not hurt.

During hospitalization he was observed to be moderately hypertensive and was treated successfully with a combination preparation of 50 mg of triamterene and 25 mg of hydrochlorothiazide twice a day. His depression gradually improved on perphenazine, 24 mg/day, and supportive and milieu therapy. The patient gained 3 kg during his hospitalization and his somatic complaints decreased. An episode of gastric bleeding during hospitalization was found to be secondary to atrophic gastritis. Neurological examination was normal except for a minor residual Bell's palsy on the left.

When this work was done, Dr. Goode was Assistant Professor of Psychiatry, University of Chicago, Pritzker School of Medicine, and Chief of Research, Ward 10-W, Laboratory of Biological Psychiatry, Illinois State Psychiatric Institute, Chicago, Ill. He is now Associate Professor, Department of Psychiatry, Bowman Gray School of Medicine, Winston-Salem, N.C. 27103.

On the patient's discharge, the above medications and simethicone tablets were prescribed.

After his discharge, he was treated at the local mental health center; the perphenazine was replaced by thioridazine, 200 mg/day. He did moderately well but continued to appear somewhat depressed. In January 1975, he was placed on amitriptyline, 75 mg/day in divided doses, and the thioridazine was discontinued at that time. The patient reported some subjective improvement in mood within 3–4 days. However, his family and friends noted that his eyes seemed to be bulging. The patient did not complain about his eyes and in fact did not seem to notice the apparent exophthalmos. On examination, his eyes were widely open at rest, with sclera visible above and below the irides. However, the eyes were not protuberant and looked normal when closed. There were no other symptoms or side effects reported and the patient said his depressive symptoms had improved. Pulse rate and rhythm were normal, and the thyroid was not palpable. The amitriptyline was discontinued and thioridazine was reinstituted immediately. The apparent exophthalmos completely disappeared within two days after discontinuation of the amitriptyline. The patient was on no other medication, having failed to renew his diuretic prescriptions several weeks before institution of amitriptyline.

Discussion

The apparent exophthalmos in this patient did not involve the protrusion of the orbit that is seen in true exophthalmos, but the palpebral aperture was dramatically widened. There was no clinical evidence of thyroid disease, palpebral pain or edema, or scleral injection, and the symptom disappeared within 2 days of discontinuation of amitriptyline.

The widening of the palpebral aperture produced by amitriptyline in this patient might be analogous to the capacity of antidepressant drugs to inhibit reserpine-induced ptosis in the rat (1–4). The action of these drugs in this animal model is based on blockade of catecholamine reuptake or metabolic deactivation, combined with the catecholamine-releasing effects of reserpine, which makes larger amounts of catecholamine accessible to receptor sites (4). Reduced catecholamine availability has been postulated as a primary component of depressive illness (5). In the case I have reported a depression-related reduction in available catecholamine might lead to secondarily increased catecholamine receptor sensitivity, which could result in an exaggerated catecholaminergic effect caused by amitriptyline through inhibition of catecholamine uptake combined with heightened receptor sensitivity. However, in the experimental situation the antidepressant agent is more effective in reversing ptosis when it is given before catecholamine depletion by reserpine. When antidepressants are given after administration of reserpine, which seems more analogous to the depressed state, they are less effective or ineffective in reversing ptosis. Moreover, the increase in the palpebral aperture observed in this patient was not a restoration of a narrowed palpebral aperture to normal as is seen in the rat, but rather was an obvious increase above normal limits. An idiosyncratic sensitivity of palpebral adrenergic receptors may have also played a role in the etiology of this apparent exophthalmos.

A second possible mechanism involves interaction of the effects of the antidepressant with those of the phenothiazine that the patient was taking immediately prior to institution of amitriptyline. Phenothiazines block peripheral receptor sites for norepinephrine and epinephrine, which may lead to compensatory increases in peripheral adrenergic activity (6). Decreasing levels of phenothiazine-induced adrenergic receptor blocking activity, combined with compensatory increased adrenergic activity and blockade of catecholamine reuptake by amitriptyline, could also result in such an exaggerated catecholaminergic effect by amitriptyline.

LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

An Unfair Portrayal of Psychiatry

SIR: I am sure that many of my colleagues recently witnessed the so-called news report on ABC television entitled "Medicine and Madness." By using the most esoteric and tangential evidence possible and including, it seemed, every medical dissenter in the current ranks of psychiatry who could be jumbled into an hour (an approach perhaps epitomized by the classification of E. Fuller Torrey as a typical "ward psychiatrist"), ABC managed to dazzle our eyes with erudite expatients waxing eloquently on the evils of Thorazine (although we were not shown any of these people prior to their "poisoning"), expatients treated with ECT whose lives were shattered by irreversible losses of memory, and probably the most outrageous case of tardive dyskinesia I have ever seen.

I would like to answer Howard K. Smith and the tiny group of producers in New York with a series of questions and problems that I face daily:

1. What would you have us do with a man who believes he is Napoleon, a girl who attacks her parents in the belief they are Jesus Christ's assassins, or an elderly woman who washes her hands 200 times a day, believing they are contaminated? Are these people simply exhibiting a rather odd type of individuality?
2. If you wish to allow such a depth of personal variance in what is considered normal, can you tell me how to get schools, police, lawyers, and almost all of my fellow nonpsychiatric physicians to stop sending me homosexuals, pedophiles, alcoholics, addicts, drifters, and anti-social characters, who are supposedly not "ill" in the classical sense?
3. Why have the populations of our institutions fallen so much since the inception of antipsychotics? How many millions did you not interview, who are willing to trade some physical side effects for the priceless gift of being released from the terrors of unseen voices, lost emotions, and untold millions of dollars in lost productivity?
4. Must I know how the human brain works in complete and cosmic detail to realize that the brain, consciousness, and subsequent emotions are somehow associated? Is a neurosurgeon to ask himself profound theological questions about what he is doing before he allows himself to operate on the brain? With untold misery and suffering in our institutions, I refuse to wait until my philosophical colleagues decide the relationship in its totality between the brain and personality before I take note of the facts that lithium seems to help mania and an extremely depressed person regains his appetite with the aid of antidepressants. However the medications work, I believe that any agent that keeps my patient from losing his family, his friends, his possessions, and his self-respect is worth the gamble.
5. How do you persuade someone not to be catatonic—by talking with them, by telling the family that this individual has his own way of doing things? Will the catatonic patient hire a "tutor" as Drs. Szasz and Torrey would have it? If I cannot find a specific neurological lesion, do I conclude the individual is not ill?
6. Because I use the label of schizophrenia, am I to blame for the ancient prejudices against the emotionally ill that reflect themselves in endless punitive ways? The term schizophrenia is no more pejorative in my mind than that which it denotes—a human being who hears voices that are not there, sees visions, and cannot seem to control his thinking.
7. Will you please feature on network television a story highlighting the research we are doing into the causes of emotional disorders?
8. Would you please remove the needless violence, sexual flamboyance, mindless comedy, and one-dimensional perfect parents from your nightly programs? These common nightly events are watched by countless millions of people who may be receiving faulty sensory input.

This program did the worst damage not to ourselves, but to the great number of unhappy people who may be contemplating some sort of therapy. By outlining extremes of current treatment and theory and depicting occasional excess, and presenting these as the norm, it leaves great doubt in the minds of all who may need help. This is the worst form of journalistic tyranny.

EVAN M. TORCH, M.D.
Augusta, Ga.

Editor's Note: Hundreds of letters and telegrams, including an official protest from the American Psychiatric Association, were sent to ABC authorities to protest this vicious attack upon psychiatry and its patients.

GHB and GABA

SIR: We were very interested in the article by Daniel P. van Kammen, M.D., on "γ-Aminobutyric Acid (GABA) and the Dopamine Hypothesis of Schizophrenia" (February 1977 issue). Certainly, GABA involvement in schizophrenia is a major issue.

We are currently involved in this problem, and we would therefore like to point out that a useful way to increase the functional activity of GABA in the brain that did not appear

in appendix 1 of Dr. van Kammen's paper is the administration of sodium γ-hydroxybutyrate (GHB), which is a precursor of GABA that crosses the blood-brain barrier.[1]

This drug, which is used as an anesthetic in France, was released by Laborit and colleagues (1). Curiously, its use in psychiatry has been quite limited. One possible use could be in the treatment of sexual dysfunction.

We think that Gamma OH deserves to be investigated by well-controlled studies.

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F. PLANTEY, M.D.
Nantes, France

[1]This drug, under the name Gamma OH, is marketed by EGIC, 19, Avenue Morane Saulnier, Meudon La Foret 92360, France.

Dr. van Kammen Replies

SIR: Dr. Plantey is quite correct in suggesting that the effects of γ-hydroxybutyrate (GHB) in schizophrenia should be examined in a well-controlled study. However, its effects are not mediated through GABA.

GHB, like its precursor γ-butyrolactone (GBL), is a minor GABA metabolite with a short half-life (1). GHB increases delta sleep and decreases REM latency (2). It has been reported that some schizophrenic patients have decreased delta sleep. GHB inhibits impulse flow in dopamine pathways, which decreases dopamine release (3); it also induces dopamine synthesis and decreases release without affecting monoamine oxidase or catechol-*O*-methyltransferase (4). Studies done by Gianutsos and associates (5) suggested specific presynaptic dopamine effects in vivo. GBL potentiates haloperidol-induced catalepsy, can affect tyrosine hydroxylase activity, and has some antiamphetamine effects. It slows the disappearance of dopamine but not that of norepinephrine in rats after α-methyl *p*-tyrosine administration. Dopamine agonists inhibit dopamine accumulation following GBL treatment.[1]

As early as 1961, Laborit's group (6) and Danon-Boileau and associates (7) reported that GHB had antianxiety and potential neuroleptic effects in psychotic patients. We are studying GHB for its alleged antidopamine and antipsychotic properties in our schizophrenic patients. One patient, who has had chronic paranoid schizophrenia for eight years and has a low tolerance for antipsychotic neuroleptics, developed akathisia that lasted up to one week after discontinuation of GHB. Another patient who had been psychotic for three years lost all symptoms. A third patient with catatonic schizophrenia of nine years' duration had an amphetamine-like response, with euphoria and increased sexual feelings (a behavior she had not exhibited previously), but became more psychotic during the GHB administration. Our preliminary data indicate that GHB may have some effects on psychotic behavior. The extent to which the effects of GHB and GBL in man are dopamine-specific is currently unknown.

[1]Additional references are available from the author on request.

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Motivations for Drug Treatment

SIR: Robert J. Harford, Ph.D., and associates are to be congratulated for their attention to a very significant and neglected area in treatment research in "Effects of Legal Pressure on Prognosis for Treatment of Drug Dependence" (December 1976 issue). However, I feel that they have stretched their interpretations more than the data allow.

The crux of my concern rests with their definition of legal pressure ("legal pressure was defined to exist if the applicant reported being on probation, on parole, or awaiting trial at the time of application"). Leaving aside the question of whether what the applicant reported was the truth, I suggest that being on probation or parole or, to a lesser extent, awaiting trial, may actually involve very little if any implicit coercion for many persons. The authors noted this possibility but seemingly ignored it when generalizing their conclusion that legal pressure does not facilitate treatment. For example, in Seattle it is not at all uncommon for a person on probation (when a condition of probation was that he enter into and successfully complete a treatment program) to leave the program early, knowing that when he agreed to enter the program charges would in effect be dropped if he quit, and that there would be no sequelae to this violation of his contract with the court.

Just being on probation, parole, or awaiting trial cannot be considered to be legal pressure. However, assuming that legal pressure did exist for some subjects, it would be useful to describe what the exact dimensions of the pressure were. Obviously, a reduced possession charge that may result in 90 days in jail is insignificant compared to an armed robbery charge associated with a drug heist and carrying a possible 20-year jail sentence.

In addition, I think that it is quite chancy to suggest that rehabilitation success may be usefully or solely measured by program retention. It is my clinical impression that a person can be an involuntary member of a treatment program, leave before the prescribed completion time, and still possibly benefit through an improvement in future social behavior. Admittedly, there are no hard data in this area.

Motivation is a ponderous word. Strictly speaking, I believe few if any people "volunteer" (without any coercion) for any treatment, and I am not aware of any definitive way to measure "intrinsic motivation." Motivation may be internally or externally derived, yet I believe that clients can be successfully treated in either case. The involuntary client or patient may have more difficult and tenacious problems and dyssocial character traits simply by virtue of the fact that he has already been charged with a transgression of social codes. If this is true he will probably be a more difficult person to work with and be more resistant to efforts to change his behavior.

I believe that most persons who are presently in discomfort (e.g., an addict with viral hepatitis, an alcoholic who is frightened that his wife will leave him if he doesn't stop drinking) are better candidates for treatment than those who have been convicted of an offense (trafficking in heroin, driving while intoxicated, etc.) and show no remorse about their behavior but "volunteer" for treatment because they are definitely concerned about the prospect of future incarceration. Consequently, given a choice between individuals who are still externalizing the source of their difficulty and have not fully adjusted to the fact that the problem is really their own and those who are truly interested and sufficiently pained by their positions to be open to behavioral intervention, I would choose the latter. However, if I had to work with the former, I would categorically prefer that treatment be preceded by a firm, well-understood contract between the client, the therapist, and the criminal justice system. Such an agreement actually begins the treatment process by holding both the client and the criminal justice system responsible for all of their behaviors, including their formalized intentions.

ROBERT B. FISHER, M.D.
Seattle, Wash.

Dr. Harford Replies

SIR: Dr. Fisher's point that some applicants who are on parole, on probation, or awaiting trial do not feel coerced into continuing treatment is well taken. Since legal pressure is the principal reason for their application, many choose to terminate treatment when their cases are resolved. On the other side of the ledger, some persons classified as voluntary admissions apply out of anticipated or imagined legal pressure that can be generated, for example, when a drug-using acquaintance is apprehended. If the expected legal consequences fail to materialize, these "volunteers" also are highly likely to terminate. There is no necessary one-to-one correspondence between the procedures used by the criminal justice system to generate legal pressure and the degree of compulsion perceived by individual addicts.

We endorse Dr. Fisher's suggestion concerning the usefulness of a quantitative measure of perceived legal pressure in clarifying the relationship between status in the criminal justice system and perceived legal pressure. However, lacking such a measure, we are not persuaded to abandon the assumption that applicants who are not involved with the criminal justice system experience less external pressure for treatment than those who are on probation, on parole, or awaiting trial. The links between criminal justice status, perceived legal pressure, and motivation in our report are *entirely* hypothetical. However, our inferences are buttressed by empirical research indicating 1) consistent differences in psychological characteristics between volunteers and nonvolunteers (1, 2) and 2) higher rates of illicit drug use during treatment by nonvolunteers than by volunteers (3, 4). These characteristics have been interpreted as indications of differences in motivation for seeking treatment.

We agree that in important respects retention is an inadequate measure of rehabilitative success. Several recent investigations have demonstrated that retention in drug abuse treatment is not correlated with any dimension of treatment outcome. In addition, time spent in treatment can produce negative effects as well as benefits, especially when adolescents with relatively minor criminal violations are placed in programs in which older criminal heroin addicts predominate. It was with these reservations in mind that we conducted our principal statistical analyses on the relationships between legal pressure and graduation rates. Graduation can be considered as a composite of the several indications of rehabilitative success as operationalized by clinical judgment. The assumptions underlying the subsequent retention analyses were stated clearly (p. 1400), and the inadequacies of the measure were acknowledged, at least implicitly, by our conclusion that "future research also should consider measures of rehabilitative success other than retention" (p. 1403).

Concerning Dr. Fisher's judgment that we overinterpreted the data, we can do no more than refer readers to the précis (p. 1399), quote the first paragraph of the discussion to the effect that our finding "*suggests the possibility* that legal pressure inhibits rather than facilitates treatment for addiction" (p. 1402, emphasis added), and cite the conclusion that "our findings *merely suggest* that legal pressure does not enhance the probability of successful rehabilitation" (p. 1403, emphasis added).

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ROBERT J. HARFORD, PH.D.
New Haven, Conn.

Memory Loss with Lithium Treatment

SIR: We would like to respond with some confirming data to the letter by George F. Bajor, M.D., entitled, "Memory Loss with Lithium?" (May 1977 issue). Our material comes

from follow-up interviews done with patients who were administered lithium under an experimental protocol during the years 1966–1975 (1, 2).

Of the 28 cases we have thus far reviewed, 7 patients, ranging in age from 21 to 56, who have all been on lithium for a period of years, have complained of memory loss that they associate with lithium treatment. This memory loss in most cases is described as an inability to recall details that interferes with daily functioning. All but 2 of these 7 patients had also received ECT at some point in the course of their illness; however, at least one patient who had received both treatments claimed to be able to distinguish the post-ECT memory loss from that associated with lithium.

We agree that this topic merits further investigation, perhaps using techniques such as those reported by Koh and associates (3–5) for the study of memory processes in schizophrenia.

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Tricyclic Choice for Ill Elderly Patients

SIR: The discussion by C. Gibson Dunn, M.D., and David Gross, M.D., of factors involved in the use of tricyclic antidepressants in medically ill geriatric patients (Clinical and Research Reports, April 1977 issue) was to the point, but I question their choice of amitriptyline to treat a 71-year-old depressed man who had serious cardiac disease and was in need of a quinidine antiarrhythmic.

The well-known cardiovascular effects of tricyclics—orthostatic hypotension, conduction disturbances, tachyarrhythmias, congestive heart failure, and ST-T wave ECG changes—appear to occur most frequently with amitriptyline (1). In a patient with preexisting angina pectoris, extraventricular arrhythmias, first-degree A-V block, and ST-T wave changes, the choice of this particular drug is especially hazardous and probably ill-advised (1).

Furthermore, recent studies suggest that concurrent therapy with certain tricyclics and quinidine may increase the risk of drug-induced cardiac disturbances. In therapeutic doses, nortriptyline, an amitriptyline metabolite, prolongs atrioventricular conduction by selectively slowing the H-V interval demonstrated on His bundle recordings (2). Quinidine has the same selective effect. Imipramine also appears to possess similar quinidine-like properties (3). Coadministration of these agents is, therefore, likely to increase the hazard of drug-induced heart block (3), particularly in a patient who already manifests a first-degree atrioventricular conduction delay.

A more rational choice for such a patient is doxepin. Although some doubt still exists as to its relative clinical potency, cardiovascular studies of patients taking doxepin confirm its superior safety, even in the event of overdosage (1, 2, 4). Orthostatic hypotension, supraventricular tachycardia, conduction disturbances, and ECG changes occur less frequently with this tricyclic. His bundlegrams in patients on therapeutic doses and in cases of overdose reveal minimal delay in H-V conduction (2), suggesting less potential for adverse interaction with quinidine-like antiarrhythmics.

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DANIEL R. WEINBERGER, M.D.
Boston, Mass.

Drs. Dunn and Gross Reply

SIR: We appreciate the opportunity to review the major points of our article:

1. It is important (and may be critical) to diagnose depression in the medically ill geriatric patient.
2. Active treatment of such depression is feasible.
3. Medication side effects must be considered and specific precautions taken to minimize morbidity. These precautions may include careful vital sign observation, serial ECGs, serial tests of pulmonary function and arterial blood gases, good pulmonary toilet, and others indicated by a patient's physical condition.

Although drug selection was not intended to be a focus of our article, we agree that doxepin would be a reasonable alternative to amitriptyline. The choice is not as simple, however, as Dr. Weinberger implies. Doxepin is recognized to be a much less potent antidepressant (1). Its effect on inhibiting the reuptake of biogenic amines at the synapse may be only one-third to one-half that of the other tricyclics. Snyder notes the following relative central anticholinergic strengths of the most frequently prescribed tricyclic antidepressants: doxepin, 1.0; imipramine, 1.9; amitriptyline, 3.7 (2). If we allow for the larger doxepin dosage to achieve a similar clinical effect in a severely depressed patient, we have lost much of the advantage claimed. This is not to argue against doxepin. Rather, we stress that repeated careful monitoring of vital functions, not excessive faith in any single drug, should

be the primary precaution when treating depression in geriatric patients.

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A Computer-Based Lithium Library

SIR: Lithium salts are being widely used for the treatment and prophylaxis of bipolar affective disorder (manic-depressive psychosis). In addition, the effectiveness of this drug in more than 30 other illnesses is under investigation. In recent years the body of literature covering this field has expanded explosively and will soon exceed 4,000 articles.

In order to deal with this voluminous body of information in a way that provides rapid and complete access to the data base, we have adapted the "Great Paper Search" computer program[1] to create a computer-based lithium library.

As recently described (1), the library is an international index that is continually updated to keep abreast of recent developments; it currently contains over 3,000 articles. It can be thoroughly searched in a matter of minutes according to author, title, journal, and subject (key word). Any combination of these four headings may be searched simultaneously using Boolean "ands," "ors," and "nots" to define the limits of the search. We have on file all articles listed on the computer so that once a search has been conducted copies of pertinent articles can be obtained.

The program is now directly available to persons or institutions with access to a compatible computer terminal; those without access to a terminal can contact the Lithium Information Center by phone or mail (address: Lithium Information Center, University of Wisconsin Center for Health Sciences, 427 Lorch St., Madison, Wis. 53706; telephone: (608) 262-1674 or 262-6314).

This service is readily accessible to psychiatrists and other interested professionals, and we hope they will avail themselves of it to obtain up-to-date, comprehensive and problem-specific information about lithium and its medical-psychiatric applications.

Finally, the "Lithium Librarian" should serve as a model that will allow development and implementation of similar systems covering other specialized subject areas of interest to clinicians and researchers.

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JAMES W. JEFFERSON, M.D.
JOHN H. GREIST, M.D.
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[1]Information is available from Howard Bleich, M.D., Beth Israel Hospital Computer Medicine Laboratory, 330 Brookline St., Boston, Mass. 02215.

Public and Private Psychiatric Care

SIR: "Problems in Analyzing the Comparative Costs of Private Versus Public Psychiatric Care," by Steven S. Sharfstein, M.D., and associates (January 1977 issue), highlights issues with which I have been groping.

My work with three community mental health centers and in private practice has convinced me that the two methods of care, while overlapping, really serve two different types of patients. In the CMHCs I can handle chronic schizophrenic patients, most alcoholics, and multi-problem families better than I can in a private setting because there are many people and treatment methods available to apply to these problems. However, the CMHCs fail miserably when they try to deal with neurotic patients because the centers have too few people trained to do insight psychotherapy and because funds are too limited to allow fully trained therapists enough time to help such patients. At times I feel forced to cheat clinic patients because I must listen to them for only half an hour a month instead of really treating them.

Perhaps one problem is that we are unwilling to state clearly what we can and cannot do. We can assist a team in keeping a chronic schizophrenic out of the hospital and in keeping an aggressive alcoholic from brutalizing his wife, but the best therapist in the world, even given unlimited visits, can cure neither one. We can see five neurotics ten times a year and really help none, or see one neurotic once a week for two years and save his marriage and help him get a better job. Perhaps the ultimate in double binds is the plan that allows 30 visits a year. It allows abuses of supportive treatment and castrates almost all insight therapy. It is a procrustean bed, which looks like a compromise but ultimately escalates costs and prevents benefits.

WILLIAM L. CLOVIS, M.D.
Philadelphia, Pa.

Dangers of Minimal Drinking

SIR: The letter by E. Wayne Massey, M.D. (March 1977 issue) on "Neurology of the Nonalcoholic" alcoholic was most interesting and highlighted the potential dangers of what might be considered by many to be only minimal drinking.

I am writing to point out a third serious danger, possibly more dangerous than the two mentioned by Dr. Massey, peripheral neuritis and cirrhosis of the liver. This third factor is organic brain damage; it is probably omitted from consideration because it is very difficult to detect in its early stages or if there is only a minor degree of damage. The common causative factor in all of these three potentially serious conditions is probably the aggregation of red blood cells (blood sludging), as described by Moskow and associates (1). As these authors pointed out, the aggregation of red blood cells blocks the circulation and creates anoxemia of the organs involved; the nervous system is particularly sensitive to this effect. Everyone working in the field of alcoholism ought to be aware of the phenomenon of blood sludging.

The concept of blood sludging has led to a logical form of therapy, i.e., use of anticoagulants to reduce the blood sludging. We have found this approach to be very helpful in patients with alcoholic brain damage (2) but do not know if it would be helpful in cirrhosis or in peripheral neuritis, although one patient with the latter condition did improve considerably.

A fourth potentially serious complication of alcohol intake is now becoming more publicized—damage to the fetus during the first trimester of pregnancy. Although the early studies of the effects of alcohol during pregnancy were done with chronic alcoholics, it is quite possible that even small amounts of alcohol may cause serious damage to the developing fetus if ingested at a critical developmental period.

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ARTHUR C. WALSH, M.D.
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Dr. Massey Replies

SIR: I appreciate Dr. Walsh's response to my letter in the *Journal*. He was quite correct to point out another factor that is a danger of minimal drinking. I omitted organic brain damage because minor degrees are extremely difficult to detect and it is already well known that alcohol abuse can lead to it. My emphasis in the letter was on neurological entities that are often forgotten.

Blood sludging is indeed a possible factor in organic brain damage, although absolute proof is lacking in the literature. Since the cerebral system is so different from the peripheral one, one could not automatically assume that sludging of red blood cells was the causative factor in peripheral nerve disease even if it were proved the cause of changes in cerebral circulation. Work on cerebral vasculature sludging has been done at the University of South Carolina for 30 years, but results are difficult to determine when dealing with cerebral blood flow techniques.

E. WAYNE MASSEY, M.D.
Bethesda, Md.

Problems in Liaison Psychiatry

SIR: I have enjoyed the writings of James J. Strain, M.D., on how liaison psychiatry should work (1), including his article entitled, "The Medical Setting: Is It Beyond the Psychiatrist?" (March 1977 issue). However, in my study of a new psychiatric unit and department in a general hospital, I have found that his hopefulness is not warranted (2).

I fully agree with Dr. Strain that liaison psychiatry offers a broad, exciting field, and an opportunity to work closely with the rest of medicine. However, my study indicates that in actual practice things are not this simple. In my five-year experience with one psychiatry department, it went from active involvement with the rest of the hospital to a self-serving, independent psychiatric hospital that happened to be housed in a general hospital.

It has been my experience that when new medical and surgical procedures (e.g., aortic graft, kidney transplant, stomach dialysis for kidney failure, breast augmentation, total hip replacement, and now kidney dialysis) are introduced and being perfected, psychiatrists, as Dr. Strain stated, "are not only welcomed, but in some settings beseiged to collaborate in [patient] management." After these procedures become old hat and the operators more confident, psychiatry is generally ignored. (How many aortic transplants have psychiatrists been called in for in the past five years? How often is a psychiatrist called in to see patients with delirium tremens in an established alcoholism treatment unit?)

Dr. Strain sees only the tip of the iceberg. Fifty percent of the time of American psychiatrists is spent in private practice. He should not extrapolate his impressions from a large, organized setting and say they work or should work in other settings—they don't.

Since my study, I have personally visited or obtained data from 17 other hospitals with 200–400-bed capacities that have psychiatric units and departments; I found that all but one had had experiences like the one I described. The lone exception was looked on by third-party carriers as having custodial-type care and had a high rate of retroactive denial of payments. Hospitals in the 450-bed and over range tended to move in Dr. Strain's recommended direction. They tended to have psychiatrists as full-time hospital employees. Dr. Zigmund Lebensohn (personal communication) was critical of my findings and recommendations, but his figures from Sibley Memorial Hospital (357 beds) in Washington, D.C., indicated even less involvement by the Department of Psychiatry in liaison psychiatry than I found in my experience.

No one has discussed financial resistance to liaison psychiatry. Bertram Brown, M.D., pointed out that the cost of general medical services is sharply reduced after introduction of psychiatric services (3). The corollary is that the nonpsychiatric physician in private practice makes less money if a psychiatrist is called in. I would like to find out from Dr. Strain if in the setting in which he works there is any financial incentive to calling or not calling a psychiatrist for consultation.

Although Dr. Strain's article and book are informative and thought provoking, I feel his ideas are generally unworkable except in large institutions with psychiatric and nonpsychiatric physician employees.

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ROBERT BENSON, M.D.
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Dr. Strain Replies

SIR: I appreciate Dr. Benson's interest in our work and his evaluation of the feasibility of the program described in our book (1–3). Although Dr. Benson has accurately identified the dilemmas that may impede the implementation of such a program, his pessimism with regard to the future of liaison psychiatry is unwarranted; it has been our experience that these dilemmas are not insurmountable. I think that Dr. Benson's lack of clarity with regard to the appropriate setting for a liaison psychiatry program and the distinction between liai-

son psychiatry and psychiatric consultation may account for many of the questions and doubts he raises.

The teaching of psychological concepts would be regarded as a legitimate activity in a teaching hospital but might not necessarily be viewed as such in a community hospital. We view liaison psychiatry as a pedagogic pursuit that attempts to teach medical caretakers at every level of training about core issues in medical psychology, e.g., depression in the medically ill, psychopharmacology, psychological reactions to illness and hospitalization, with the expectation that in time the interns, residents, and other health care workers will provide most of the psychological care for their patients themselves.

We regard consultation and liaison psychiatry as separate and distinct activities. Although consultation is the cornerstone of our work, the teaching efforts of the liaison psychiatrist should ultimately enable the medical staff to offer adequate psychological as well as medical care to aortic transplantation patients, patients with delirium tremens, etc., themselves, rather than routinely referring such patients for consultation. Since 50%–80% of medically ill patients have primary psychological dysfunction or psychological dysfunction secondary to physical illness, psychological care must reside with the medical caretakers and not the psychiatrist. Furthermore, these findings underscore the need for wide dissemination of knowledge of the psychology of the medically ill among medical caretakers.

Dr. Benson's observation that psychiatry departments in general hospitals tend to become independent and self-serving over time is an astute one. He has identified a major dilemma of the liaison psychiatrist but, once again, we do not feel this problem is insurmountable. He needs to convince departments of psychiatry of the purpose of his mission in order to avail himself of the resources of the department to implement his goals. In the last chapter of our book we have spelled out ways in which this problem has been resolved at Montefiore. Although we have not yet achieved the ideal relationship with our own department of psychiatry, we do feel that such an alliance lies within our grasp. However, without a considerable liaison effort directed to one's own department of psychiatry and the hospital administration, the situation that Dr. Benson describes as almost universally prevailing inevitably occurs.

To illustrate, the primary goal of the liaison psychiatrist is to persuade the department of medicine to secure the psychological well-being of its patients. Once they have become convinced of the need for such care, staff members of the department of medicine have a right to expect the liaison psychiatrist to make it available, and they object with justification to the fact that patients may be deprived of the psychological care they urgently require because they do not live in a prescribed geographical area, are financially ineligible, or do not interest the psychiatric staff. The liaison psychiatrist, who is in a position to witness these inequities at first hand, is obliged to bring them to the attention of the department of psychiatry, lest he forfeit the relationship he has worked to establish with the department of medicine. However, he frequently disturbs his psychiatric colleagues by requests that they reexamine their modus operandi (3).

Dr. Benson has accurately targeted the source of financial support as a major dilemma facing the liaison psychiatrist. Although one can bill for a psychiatric consultation, one cannot bill directly for liaison psychiatry efforts. (This clearly shows that third-party payers and federal, state, and city legislatures see a compelling difference between classical consultation psychiatry and liaison psychiatry, regardless of how psychiatrists themselves may feel about this division.)

There are currently four sources of funding for consultation/liaison psychiatry: 1) routine reimbursement for the standard psychiatric consultation; 2) hospital and medical school teaching appointments for liaison psychiatric teachers; 3) faculty stipends, and to a lesser extent, fellowship stipends for liaison psychiatry trainees funded by the Psychiatry Education Branch of the National Institute of Mental Health; and 4) innovative funding approaches that draw on consultation monies to provide liaison teaching and training.

The Renal-Psychiatric Modular Unit at Montefiore is an innovative approach that incorporates measures making it economically feasible to create a training position for a liaison psychiatric fellow (fourth-year resident) who will provide psychological care on an ongoing basis for chronic renal patients. Public Law 92-603 specifically allows Medicare payments of up to $250 per year for direct psychiatric consultation services to all renal transplant patients, regardless of age, for one year after surgery. Our program has been able to recruit fellows who agree to provide direct services to a limited number of renal patients, the total fees generated by such services to be equivalent to the stipend customarily paid fourth-year (fellowship year) residents, and to devote their remaining time to teaching psychological medicine in exchange for training in this subspecialty (4).

Finally, since the liaison psychiatrist does not take over the patient, he is not a competitor for the medically ill patient; in fact, he should enlarge the practice of his medical colleagues, who will not only be more able to identify psycho-social-medical distress, but who will also be in a position to offer treatment within the ongoing context of their medical practice.

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Ictal and Subictal Neurosis

SIR: I would like to make some comments regarding "Delinquency, Psychomotor Epileptic Symptoms, and Paranoid Ideation: A Triad," by Dorothy Otnow Lewis, M.D. (December 1976 issue). It has always taken courage to diagnose psychomotor epilepsy on the basis of symptoms alone in the absence of a corroborating EEG. Neurologists consistently deny the existence of the condition unless textbook symptoms are accompanied by abnormal EEGs. However, somewhere along the gradient of illness CNS events that are not severe enough to produce EEG changes are still capable of producing symptoms of spontaneous cerebral discharges.

Since both the limbic system and the temporal lobe are involved in the whole range of cognitive-emotional re-

sponses, it is obvious that some lesions in these areas may produce atypical manifestations, referred to as ictal and subictal neurosis (1). Many patients with such symptoms are considered to be psychoneurotics, psychopaths, or sociopaths, and are treated accordingly. Neurologists in such cases tend to confuse cause and effect—when an individual becomes aware of episodic, disruptive, acausal intrapsychic changes that may result in meaningless, asocial, or antisocial activity, he or she almost always experiences profound anxiety, and treatment of the accompanying anxiety or depression is then recommended.

Unless this "borderland" of epilepsy is recognized at the medical-school level, neurologists will continue to diagnose it as anxiety neurosis. Insufficient research has been devoted to the cognitive status of these episodically occurring percepts. Such neural activities are correlated with an immediately sensed quality; by giving them a symbolic representation, as psychiatrists are wont to do, a causal sequence is implied where none exists. The interviewer has to be cognizant of the fact that patients are aware of sensory data that they are unable to describe.

Beyond the theoretical interest of this phenomenon is a clinically important consideration. As a psychiatric consultant in an Army hospital I see many young men who are brought in as a result of violent or antisocial behavior. In a surprisingly large proportion of these instances, even a brief interview discloses a history of head injuries after which these men notice a change in behavior for which they cannot offer any explanations; in some cases there is a time lapse between the injury and such changes.

Dr. Lewis mentions that little work has been done on the prevalence and characteristics of the disorder in delinquents. Here I must disagree with her. There is a rich literature, which I mentioned in the bibliography of my book (1), and much work has been published since. The Dreyfus Medical Foundation, New York, N.Y., maintains the most complete file of information. Perhaps Dr. Lewis found so little reference to this syndrome because of the general reluctance to ascribe what appears to be a behavioral abnormality to discrete neurological pathology. Interactions between neurology and psychiatry in such cases require further work.

In some instances, discrete lesions, which are not always verifiable, of either the temporal lobes or parts of the limbic system may be behind the soft neurological and the pseudoneurological symptoms of psychopaths, persons with impulse disorders, hypochondriacs, and psychoneurotics.

One has to witness the nearly incredible authenticity of ictal automatisms to appreciate the intellectual effort psychiatrists must expend to avoid the trap of teleological reasoning. It requires a major reorientation of the thinking of both neurologists and psychiatrists to acknowledge the existence of this wide borderland.

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A.D. JONAS, M.D.
New York, N.Y.

Dr. Lewis Replies

SIR: I was pleased to receive Dr. Jonas's response to my paper. His experience with adults manifesting a variety of symptoms and behaviors complements our experiences with youngsters in trouble with the law. He raises important issues regarding the need to heighten the awareness of doctors in general and psychiatrists in particular to the existence of psychomotor phenomena and the need for psychiatrists to always take a meticulous medical history. I am grateful to him for sharing his experiences.

DOROTHY OTNOW LEWIS, M.D.
New Haven, Conn.

A Kudo for the Boards

SIR: Before I took the American Board of Psychiatry and Neurology oral examinations in Washington, D.C., I reread "Experiences of Psychiatry Board Exam Casualties: A Survey Report" by Martin R. Lipp, M.D. (March 1976 issue). I felt quite discouraged—I was working full-time and had an 18-month-old boy at home, which did not make studying for the Boards an easy task.

My husband and son accompanied me to the examination site. We found the hotel accommodations excellent. The registration was fast and Dr. Rudy's orientation was helpful. Busses were provided for transportation to the different hospitals, and the examinations were conducted according to schedule, with no delays. I passed the examinations and was certified in psychiatry.

To my colleagues who will be taking the examinations, I would like to say, do not be discouraged by "casualties." Let us hear more positive feedback from physicians who passed the Boards and found them to be quite a rewarding educational experience.

EDITH HAR-ESH, M.D.
Hyde Park, N.Y.

Tachycardia in Acute Schizophrenia: Assessment of Treatment Adequacy

SIR: Acute schizophrenia is often thought of strictly in terms of emotional and psychiatric symptoms, with no consideration of somatic signs. However, psychosis is often accompanied by signs of autonomic nervous system changes such as tachycardia, which can exist with no outward signs of agitation, nervousness, or anxiety. Tachycardia occurs in acute psychotic conditions such as schizophrenia and mania, and although it is not diagnostic of psychosis, its presence provides a fortuitous guide to the adequacy of treatment.

The mainstay in the treatment of acute schizophrenia is neuroleptic medications. By monitoring pulses several times a day in those psychotic patients who have elevated heart rates, the clinician can gain a reliable guide to the adequacy of the treatment. Tachycardia indicates insufficient treatment (usually inadequate doses of neuroleptics), while a normal pulse tends to indicate adequate therapy. This is true in spite of the fact that neuroleptic drugs, being anticholinergic, tend to increase heart rate.

In conclusion then, acutely psychotic schizophrenics often have elevated pulses, regardless of whether they are calm or agitated, and the heart rate decreases during adequate treatment with neuroleptic medications, despite their pharmacological tendency to increase heart rate.

We are currently in the process of conducting formal research on this observation and have started a pilot program

to assess pulse rates in acute schizophrenia and plot them against recovery rates.

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HERBERT WAGEMAKER, M.D.
Louisville, Ky.

Exhibitionism or Narcissism?

SIR: Marc H. Hollender, M.D., and associates described a patient purported to be a "striking case of a female exhibitionist" (April 1977 issue). Their patient, Ms A, is described with clarity and sensitivity, but her behavior does not seem "striking" and there are some reasons to doubt that hers is a case of "genital exhibitionism."

Essentially, Ms A, a stripper, is described as somewhat defective in reality testing and judgment, limited in emotional resources, and hungry for attention. When intoxicated she "flashes," exhibiting her breasts and genitals. Flashing is but one of a wide range of exhibitionistic behaviors that seem to abound in our culture (e.g., "mooning," "streaking," stripping, nude encounter groups, topless sunbathing, nudism). Ms A's flashing does resemble the behavior of the familiar male sexual variant. Yet flashing from doorways would appear to be commonplace in certain settings according to the descriptions of the activities of prostitutes in "red-light districts." Whether the motive for the behavior of these prostitutes is the solicitation of customers or, as in the case of Ms A, a means of attention getting would be hard to determine without specific knowledge of needs, wishes, and fantasies. Some combination of motives is likely. It would be reasonable to imagine that many of the denizens of such red-light districts would have personality structures and dynamics quite like those of Ms A.

An important issue is whether or not Ms A is indeed a genital exhibitionist rather than a body exhibitionist. Since she exhibits breasts and genitals and is essentially naked except for her raincoat, it is assumed that when she flashes she reveals most of her nakedness. Even if it could be assumed that what is most important to Ms A, in fact or fantasy, is that she exhibits her genitals, would her case still be properly classified as a sexual deviation? Certain criteria have to be met to distinguish exhibitionism as a sexual deviation from exhibitionism in the broader sense. The definition of exhibitionism used by the authors is taken from the subsection entitled "Perversion" in *Comprehensive Textbook of Psychiatry* (1). In the opening paragraph of this subsection it is stated, "Perversions are manifestly sexual in character. When the pathological impulses are released orgasm is achieved." Stoller states as part of his definition of perversion that it is an "habitual preferred aberration necessary for one's full satisfaction" (2). Ostrow and associates describe perversion as "an erotic technique" that one uses as a "complete sexual act" (3). Their definition requires that the behavior "leads to orgastic discharge." The typical exhibitionist exposes himself for sexual gratification, usually reaching orgasm through masturbation (4).

The case of Ms A appears to be neither unique nor a case of genital exhibitionism as it is usually defined. Her behavior does not sexually excite or gratify her. It is rather the regressive behavior, under the influence of alcohol, of an immature woman with intense body narcissism. The case does not seem to contribute much to the understanding of sexual deviation, despite the implications of the title and the overall manner of presentation.

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MILTON EBER, PH.D.
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Dr. Hollender and Associates Reply

SIR: Dr. Eber's disagreement with our paper is essentially based on the definition of genital exhibitionism, which we define as the deliberate exposure of sex organs under inappropriate conditions for the purpose of evoking a response in an observer. Such a definition permits the inclusion of the act as an attention-getting maneuver as well as the expression of castration anxiety. In Dr. Eber's definition, sexual gratification and orgasm are emphasized, thereby excluding acts based on pregenital needs.

We agree with Dr. Eber's contention that the phenomenon we described is "the regressive behavior . . . of an immature woman with intense body narcissism" and suspect that such a description might fit other women who are genital exhibitionists.

MARC H. HOLLENDER, M.D.
C. WINSTON BROWN, M.D.
HOWARD B. ROBACK, PH.D.
Nashville, Tenn.

Blood Types and Affective Disorders

SIR: Robert W. Shapiro, M.D., and associates reported in "ABO Blood Groups in Unipolar and Bipolar Manic-Depressive Patients" (February 1977 issue) that "A significantly higher percentage of bipolar patients than unipolar patients had blood group O, while a significantly higher percentage of unipolar than bipolar patients had blood group B." The authors assert quite reasonably that this finding supports the validity of the unipolar-bipolar distinction. Their further view that vulnerability to manic-depressive disorders may be related to membrane disturbances may be correct, but is not necessarily related to the implications of their report. Flemenbaum and Larsen (1) reviewed this topic and have shown some of the sources of error in the handling of statistics, but it is not on such grounds that Shapiro and associates' concept must be questioned.

If, indeed, there is a distinction in the frequencies of the O and A blood groups in manic-depressives as compared with the general population (as is shown in the table in Shapiro and associates' article), it would imply a locus on chromosome 9 for at least one genetic factor in these psychoses (2). One would then expect to find a familial pattern of inheritance relating the two phenomena. Such a pattern is seen, for example, in the nail-patella syndrome. This is instructive, for as Renwick and Lawler (3) showed, when a sufficiently

large number of families are studied the ABO frequencies do not differ from those of the general population. However, *within* families the inheritance of the syndrome occurs strictly in accordance with the blood type of the "founding" ancestor. Exceptions occur only as a result of recombination, at the rate of about 5%.

If, as seems likely, manic-depressive psychoses have existed in human populations for untold generations, there should not be a distinct difference in the ABO groups between the general population and the manic-depressives. We might consider a simple possible explanation for the data of Shapiro and associates: One "founding" ancestor, perhaps a manic with high political or military power, had blood type O, and through multiple matings in Denmark with OO, AO, BO females would have about a 66% chance of having OO progeny.

The proper way to examine this matter is through studies of families, but this is somewhat difficult. In the last two years, in the course of family studies, I have been able to examine only three families of individuals with bipolar affective disorders having afflicted members in at least two generations and with a sufficient number of cooperative relatives that such a relationship might be sought. In one family the data are not informative, but in the other two there is no segregation of blood type with clinical trait or state. Of course, such negative data are very slight and stand to be contraverted by positive family occurrences.

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WILLIAM J. TURNER, M.D.
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Dr. Shapiro and Associates Reply

SIR: Dr. Turner states that there should not be a distinct difference between the frequencies of ABO blood groups in the general population and in manic-depressive patients if manic-depressive illness has existed in human populations for untold generations. This statement applies only if one considers the relationship between manic-depressive disorder and ABO blood groups to be one of linkage. However, this statement does not apply if the relationship is one of casual association. Dr. Turner's example of the nail-patella syndrome suggests that he is thinking only in terms of linkage. The relationship between the nail-patella syndrome and the ABO blood group system is due to autosomal linkage, and this condition is not associated with ABO blood groups in unrelated patients. Our own observation of a higher frequency of blood group O in bipolar patients is probably more analogous to the well-known association between blood group O and duodenal ulcer. As Penrose (1) has pointed out, this association may be due to an effect of the O gene itself, rather than to that of an ABO-linked gene. Similarly, our own observation could be the result of an effect of the O gene itself.

Finally, we cannot entirely exclude the possibility of the "founder" effect hypothesized by Dr. Turner, although we find it rather unlikely, since the Danish population is very outbred and probably without detectable stratification.

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Ethics and Health Policy, *edited by Robert M. Veatch and Roy Branson. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1976, 324 pp., $15.00.*

The Institute of Society, Ethics and the Life Sciences, which published a highly regarded volume on the teaching of medical ethics, has established a research group on ethics and health policy. This book is the result of that effort. The tone set by the editors disavows simplistic solutions and clarifies differences among experts, giving the reader a much more realistic picture of the complexity of ethical issues as they relate to general medical practice, including psychiatry.

The book is divided into three sections. The first, Health Care Delivery: Fundamental Ethical Conflicts, presents a broad conceptual framework for examining the ethical implications of patient care in the social context. Leading scholars in the fields of philosophical and social ethics address and debate issues such as the right to health care. Also included in this section is a fascinating chapter on medicine in China. The second section, Ethics and Allocating Scarce Medical Resources, examines some of the moral dilemmas associated with advanced medical technology (e.g., the artificial heart). Section three, Ethics and Health Policy Planning, elucidates such issues as measuring economic benefits of health programs, community participation in health care decisions, technology assessment, and genetics.

The general level of scholarship in this book is excellent. Despite the theoretical excursions into philosophical and social ethics unfamiliar to most physicians, it reads extremely well. I found it refreshing that conflicting opinions and debate are highlighted.

Psychiatry and medicine cannot afford to ignore the issues raised in this book. However, theories of philosophical and social ethics and the theories found in the psychiatric field (developmental psychology, psychoanalytic theory, social systems theory, group dynamics, and knowledge regarding institutional behavior) that need to be brought together have not been in this book. Also, this particular volume lacks a sufficient psychological perspective. Such a perspective is generic to the study of individual and social ethics, which are unavoidably and profoundly psychological. The lack of a psychodynamic perspective gives a sense of one-dimensionality to the book's otherwise excellent content.

Not only this volume but a number of others written by philosophers, theologians, and social scientists on the subject of medical ethics tend to focus on exotic moral and ethical problems, e.g., when to prolong life, the feasibility of artificial hearts, and the control of genetic abnormalities. I believe it is much easier to debate these topics than it is to address the everyday nitty-gritty issues of moral conduct and rectitude that take place between physicians and patients. This point is especially important for the psychiatrist. Such issues extend beyond the one-to-one patient-therapist relationship. In dealing with patients the psychiatrist invariably deals with individuals who are related to other people in a social context—spouses, children, parents, and friends.

I feel that this book correctly links ethical principles of medical practice with issues of social justice. Despite minor shortcomings, the book is an important beginning. The intent of the editors is obviously directed toward interdisciplinary collaborative work. Physicians and psychiatrists are challenged to face squarely both matters that have to do with their own moral rectitude in behavior with patients (individual ethics) and their responsibilities to the community at large (social ethics). As Ortega said, "I am myself and my circumstance"; this includes the social context. In other words, psychiatrists and physicians must attend to the ethical problems arising in their relationships to their patients as well as to the system of care of which they and their patients are a part. Anything less is an abrogation of professional responsibility.

PHILIP WOOLLCOTT, JR., M.D.
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Psychiatric Education: Prologue to the 1980's: Report of the Conference on Education of Psychiatrists, Lake of the Ozarks, Missouri, June 9–15, 1975, *by Anne H. Rosenfeld. Washington, D.C., American Psychiatric Association, 1976, 512 pp., $15.00.*

The Working Papers of the 1975 Conference on Education of Psychiatrists. *Washington, D.C., American Psychiatric Association, 1976, 423 pp., $12.50 (paper).*

Psychiatric Education is a report of a conference that was held at the suggestion of the APA's Council on Medical Education and Career Development and APA's Committee on Medical Education. The council and the committee felt that in the last 10 years important social, economic, and political changes have taken place that profoundly affect the delivery of health care services, including mental health care services. They also felt that these changes would have important implications for the education of future psychiatrists.

When the conference was planned in the 1960s, there was a great deal of violence in our society—a struggle for civil rights, the demand for medical care for the poor, and demonstrations for an end to the Viet Nam conflict, for example. During this time, as a response to specific troubled areas, the Civil Rights Act was passed, Medicare and Medicaid developed, community mental health centers were built, and efforts were made to shorten medical school education and residency training.

By the time the conference was held in 1975, after Viet Nam and Watergate, there was a strong distrust of government, leadership, and authority. Through consumer groups and through Congress the public was demanding accountability, reflected in government attempts to control the cost and quality of medical service. At the same time, the federal government was reducing the funds spent for education of psychiatrists. Because the demand for mental health care is constantly changing and the form that the mental health sys-

tem will take in the 1980s is yet to be determined, the conference could deal with only general goals and general principles of education.

Psychiatric Education is based on the reports of seven preparatory commissions, reactions to each of these reports, propositional reports of 12 workshops, and proceedings of the conference plenary session. The following topics were discussed: goals and values, the resident (the student), faculty, educational methods, the environment, the economics of training, the social context of training, and evaluation of competency and quality control. Data are presented in each chapter to support the statements made. When such data were not available, normative views of very distinguished educators are supplied.

Unfortunately, very limited funds have been invested in research to investigate in depth the process of education for psychiatric residents. Such research might answer many of our questions. Some of the areas that need to be investigated might include the questions of what factors facilitate or impede the education of residents and what is the relative contribution to the process of education of faculty, students, educational methodology, and environment.

If there was one single focus in this conference it was evaluation and quality control. There was a general consensus that efforts must be made to measure the effectiveness of the educational process and the competency of the graduate. Knowledge is relatively easy to measure; skills and values are far more difficult to evaluate.

From this conference came a clear set of guidelines designed to enhance the broad knowledge of psychiatrists. A modification of these guidelines has already been published by the Residency Review Committee of Psychiatry and Neurology. These guidelines will be subjected to quality control and accountability by both the medical community and the public at large.

Anne Rosenfeld has captured the spirit of the conference very effectively. Her book can be recommended to any educator who is seriously interested in the education of psychiatric residents. Many educational principles found in *Psychiatric Education* could be applied to the education of medical students and child psychiatrists.

BERNARD HOLLAND, M.D.
Atlanta, Ga.

Maturation of the Therapeutic Community: An Organic Approach to Health and Mental Health, *by Maxwell Jones, M.D. New York, N.Y., Human Sciences Press (Behavioral Publications), 1976, 163 pp., $12.95.*

In this work Maxwell Jones has brought together in one brief volume many cogent observations from his almost 30 years of work with a variety of organizational systems. His purpose is to describe how a social systems approach can be useful in understanding interactional processes, how movement toward a more open social system can be beneficial to the society as a whole, and, specifically, how the open systems approach can enhance the functioning of a variety of mental health settings.

After a somewhat confusing and not well-focused foreword and introduction, the book moves rapidly to its major theme as Jones begins to show how a relatively static system can gain momentum toward openness and change through specific interactional methods. His descriptions of the preliminary or team-building phase, the process or action phase, and the evaluative or evolutionary phase are well written. They clearly outline and discuss principles that are valuable to any worker, professional or nonprofessional, in a mental health system. These processes are certain to be familiar to anyone who has worked in a mental health system, whether it be a hospital, community mental health center, or academic department of psychiatry. Jones also provides informative anecdotal material based on his personal experiences as a facilitator and/or interventionist in each of these three settings; these anecdotes help to illustrate his theses.

Jones only briefly examines the specific relevance of his theoretical positions for the practice of psychiatry; he raises more questions for me than he answers. However, it is clear from this book that the different organizational settings in which a psychiatrist chooses to practice may require different role orientations. The independent private practitioner rarely needs to be concerned with his or her role in a system, although this may change as peer review, Professional Standards Review Organizations (PSROs), and national health insurance become more prominent influences. In contrast, the psychiatrist who works within a system often has many problems in defining his or her role in relationship to the system and the other people in it. I believe this difference raises some important issues for training programs, specifically involving the mechanisms by which they prepare or fail to prepare psychiatric residents for work in a mental health system. For example, is it possible that some of the concern raised about the role of psychiatrists within some community mental health centers is related to the failure of training programs to adequately prepare the psychiatrist for participation in an open system? This question and others related to the role of the psychiatrist receive relatively little attention in *Maturation of the Therapeutic Community*.

Although Jones stresses the importance of evaluating the effectiveness of an open system as it affects therapeutic outcome, he does not discuss one of the most important consequences of the open systems approach, namely the recent emphasis on patients' rights. One must wonder whether this would have become as prominent an issue if it had not been antedated by the move toward an open systems approach in many mental health settings. A thorough examination of the open systems approach and its legal derivatives is needed, particularly as this relationship affects the quality of treatment and treatment outcome.

In summary, this book brings together many of Maxwell Jones's theories and precepts regarding organizational development as it pertains to mental health systems. For psychiatrists who work within one of these systems, an understanding of these principles is most relevant to their work.

ALLAN BEIGEL, M.D.
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Caring, *by Willard Gaylin, M.D. New York, N.Y., Alfred A. Knopf, 1976, 199 pp., $7.95.*

Dr. Willard Gaylin is a psychoanalyst, a professor of psychiatry, and president of the influential Institute of Society, Ethics and the Life Sciences. For many years and through a series of books, he has devoted his professional expertise to the cause of social justice. In *Caring*, he argues passionately that man is now in a crisis of self-confidence, increasingly convinced of his innate destructiveness and losing hope for his future.

Dr. Gaylin believes that this problem has been engen-

dered, at least in part, by the scientific errors of psychoanalysis and ethology that have one-sidedly focused on the hostile, destructive, selfish tendencies of our species. To surmount the gathering crisis of despair, Dr. Gaylin urges on us a corrected view of human nature:

> If there is one fact founded in his biology, essential to his survival and uniquely his own, it is that *Homo sapiens* is supremely a loving animal and a caring one. . . . I do not deny that there may be a genetic directive for aggression in mankind, but the genetic basis for love is far more obvious, and that is what I plan to explore here. (pp. 10–11)

To prove this thesis, Dr. Gaylin reviews newer researches in ethology, mother-infant interaction, psychoanalytic developmental theory, and object relations theory. Chapters on attachment, separation, identification, and conscience provide a fascinating new look at the relevant data in these vital areas of psychic development. The author's emphasis is on the ways in which dependency, tender emotions, loving care, and socially cooperative behaviors play a central role in the formation of human beings in human society. The case is convincing.

As a friendly critic of psychoanalysis, Dr. Gaylin discusses the historical reasons for the delay in achieving an appropriate balance in the psychoanalytic view of man. In "Totem and Taboo" (1), one of his early works, Freud recognized the need to root conscience and group cooperation in the evolutionary history of mankind—i.e., in man's genetic structure. However, as psychoanalysis became increasingly preoccupied with the nature of drive theory, and because "Totem and Taboo" seemed hopelessly Lamarckian in outlook, this early insight was lost. "Civilization and Its Discontents" (2), with its dramatic opposition of the self and society, became the standard of psychoanalytic sociology. Today's cultural climate, concerned with pollution, overpopulation, and nuclear warfare, is beginning to shift scientific interest from man's capacity for conquest to his capacity for cooperative survival. *Caring* is clearly one of a number of works in social biology that have begun to emphasize the constructive social capacities of the species.

Dr. Gaylin contends that man essentially designs himself. The social structure of a society, particularly the kind of mother-child interactions that each culture encourages, determines the nature of the child. Hope and self-confidence are necessary to encourage loving interactions and produce loving progeny:

> I do not despair at the state of the world; I despair at the current state of passive disenchantment and self-denigration. Given knowledge of the nature of the human being, such worry is warranted. And psychological definitions of the human state tend to be self-fulfilling prophecies. We may not be what we think we are—but what we think we are will determine in great part what we are to become. We must not design our future in terms of our disillusionment with self, for those designs, even if erroneously conceived, will influence the future development of our species. (pp. 170–171)

Although Dr. Gaylin conveys a sense of crisis, he is in fact a thoroughgoing optimist. His writing is dramatic, which may not be to everyone's taste, but he is bursting with ideas and interesting asides, and he manages to present masses of data with zest and enthusiasm. The scholarship is excellent, and the book is a valuable presentation of a newer point of view in the science of human behavior. *Caring* will be of interest as a review of the literature and as a source of new ideas for students and professionals. It can be understood by the intelligent nonprofessional reader as well. The footnotes and bibliography are excellent and are presented in a form that is a model for scholarly work.

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ARNOLD M. COOPER, M.D.
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Mysticism: Spiritual Quest or Psychic Disorder? Report 97, *by the Group for the Advancement of Psychiatry Committee on Psychiatry and Religion. New York, N.Y., GAP, 1976, 110 pp., $4.00 (paper).*

Many features of the mystical experience have remained puzzling to Western students of human nature on many counts, including its psychodynamics; its relation, if any, to psychosis; and its validity as a revelation of features of the universe inaccessible to waking consciousness. The growing popularity of mystical cults in the United States has created a new interest in these questions, and this admirable brief report represents the efforts of a group of psychoanalytically oriented psychiatrists to come to grips with them.

After describing the mystical experience and surveying the current mystical scene in the United States, the report goes on to an overview of Jewish and Christian mysticism, followed by thumbnail biographies of three widely different mystics in three cultures: Ramakrishna, Jacob Frank, and Ignatius Loyola. On the basis of these descriptions, the authors explore some possible psychodynamic sources of the mystical experience and its relation to psychoses and creativity. The biographies make it clear that mystical experiences are not confined to any one type of personality. Ramakrishna was emotionally unstable, suffered from psychotic-like episodes, and went into spontaneous trances; Jacob Frank was a charismatic person openly pursuing wealth and power; and Loyola was a hard-driving, obsessional ascetic.

The authors of the report conceptualize mystical trances as efforts to cope with an unacceptable outer reality by turning away from it to a gratifying inner one characterized by self-absorption and often a consoling reunion with symbolic representations of parents. Consistent with the authors' psychoanalytical orientation, these features are analogized to regression to an infantile state, which differs from psychotic regression in that it is facultative rather than obligatory, partial rather than complete, and compatible with forming close bonds with others rather than isolating. The authors have shown praiseworthy self-discipline in confining more adventurous psychoanalytic speculations to a brief epilogue.

When all is said and done, essential features of the mystical experience remain inexplicable in terms of Western cosmology. This report wisely confines itself to a consideration of features that can be empirically studied and offers much fascinating information, objectively presented and thoughtfully

discussed. It can be highly recommended as an introduction to the psychology of mysticism.

JEROME D. FRANK, M.D.
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Annual Progress in Child Psychiatry and Child Development 1976, *edited by Stella Chess, M.D., and Alexander Thomas, M.D. New York, N.Y., Brunner/Mazel, 1977, 744 pp., $17.50.*

For nine years the editors of this review have displayed a remarkable catholicity in assembling material related to the field of child development, the practice of child psychiatry, and the concerns of professionals and others about children. Developmental issues and theories have been a major concern of the editors; this is reflected in the selection of material from a series of serious and learned theorists.

As the editors have noted before and repeat in this year's *Annual Progress*, much of the material written on developmental issues tends to be speculation. They therefore include as a lead article a paper by Sameroff titled "Early Influences on Development: Fact or Fancy?" I agree with the editors that this article is a true milestone. It sets up a transactional model that emphasizes continuity and discontinuity in the developmental process of a child. It emphasizes the relationship of a child's progress to his or her environment in a systematic way that has only been suggested before. It may well warrent being labeled a classic.

In the 1975 *Annual Progress* the heading Social Issues and Public Policy appeared for the first time. There are no papers grouped under this topic in the present volume, but it appears to me that a host of articles could have appropriately fallen under that rubric. The appearance of such a large number of articles devoted to the concerns of society as well as clinical concerns sets a new tone for this particular volume.

The section on Racial Issues of the previous volumes has become Minority Issues. An outstanding paper by Gordon, "New Perspectives on Old Ideas in Education for the Minority Poor" as well as one by Robins and associates on "Arrests and Delinquency in Two Generations: A Study of Black Urban Families and Their Children" highlight this section. The former points out clearly what has and what has not happened in the educational efforts that have been made. The latter once more illustrates how difficult it is to move individuals out of a delinquent rut once the family is cast in that mold.

To continue the concerns about social issues, an entire section focuses on child abuse. Sibylle Escalona, in "Children in a Warring World," provides the opening for this series of articles, which continues with papers on drug abuse, professional abuse, and adolescent violence. It was difficult to choose the outstanding article in this group because so many of them are good. I feel, however, that one should not overlook King's paper on "The Ego and the Integration of Violence in Homicidal Youth" or Polier's calm and considered essay on "Professional Abuse of Children: Responsibility for the Delivery of Services."

One of the neglected areas in child psychiatry over the years has been that of the relationship of siblings and the influence of siblings on the development of an individual. To fill this vacuum the editors have chosen Bank and Kahn's outstanding paper "Sisterhood-Brotherhood Is Powerful." It leads off a section on the family that is complemented by a paper by Rosenthal and associates on "Parent-Child Relationships in Psychopathological Disorder in the Child." Using Minuchin's now familiar interest in the family, Minuchin and associates contribute a well-ordered clinical paper, "A Conceptual Model of Psychosomatic Illness in Children."

I have alternately despaired and exulted at the selection of papers in this series. This volume leads to a sense of joy once more. The joy is in the writing and the description (not, certainly, the reality) of the difficulties that society is having in handling problems of children. Akin to the description of society's failures outlined by Steiner in *The Children's Cause* (1), several of the articles mentioned above, and others, emphasize the fact that we understand many of the problems of children but lack an understanding of the pathology of the process by which we care for children.

It does appear, however, that the field of child psychiatry is producing something; that larger social problems are a concern of the field; and that we are looking at the relationship of issues derived from the field of psychiatry that have wide and concerned implications. If we were able to see as clearly the clinical process implicit in the methods by which we legislate and try to organize services for children, we might begin to make better progress in the whole field of child care.

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HENRY H. WORK, M.D.
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Double Bind: The Foundation of the Communicational Approach to the Family, *edited by Carlos E. Sluzki, M.D., and Donald C. Ransom, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1976, 347 pp., $19.75.*

When I was a medical student, the first paper on the double bind theory, "Toward a Theory of Schizophrenia" by Gregory Bateson, Don Jackson, Jay Haley, and John Weakland (1, pp. 201–227), created a great stir of debate in the psychiatric faculty. It appeared to be an idea that at once explained why schizophrenic patients were not only disturbed but had a defect in their thinking, what that defect might have to do with the communication process of the family, and how all that might be related to humor, hypnotism, psychotherapy, cybernetics, and linguistics. In other words, it was a Breakthrough. Twenty years later, the excitement has still not died down. The theory has been extended and clarified by the original authors and by their students and critics.

In *Double Bind*, Carlos Sluzki and Donald Ransom have brought together the essential original papers, 17 in all, and several chapters of commentary. Aside from the commentary, 6 of the pieces appear in print for the first time. The whole is arranged to convey the history of the development of an idea. Indeed, one of the most valuable new contributions is Haley's history of the project itself, with Bateson's and Weakland's comments on it.

This book is a scientific adventure; let me say at once that I recommend it to historians of science as well as to clinicians and researchers in the mental health field. It is also a monument to the extraordinary influence of Gregory Bateson. Those who found it difficult to get the message of his *Steps to an Ecology of Mind* (1) may find him better delin-

eated here as he and his colleagues debate with each other. Reading the debate, one realizes that we have learned from them the language and metaphors of communication theory; and no matter what we think, the way we think will never be the same.

The commentaries point out, repeatedly, that the project was not exclusively or even primarily interested in schizophrenia. Indeed, according to Haley's history, the project began with the study of communication patterns in small groups of humans and animals engaged in such activities as play and psychotherapy. They decided to concentrate on schizophrenia when their Rockefeller grant ran out and a more practical project had to be funded. By the end of the project (10 years later), each of the original group had turned to other concerns (hypnosis, psychotherapy, general family therapy, dolphins), but for academic psychiatry, an etiology of schizophrenia had been proposed and had to be tested. People started defining and counting double binds in family interactions. This research is masterfully reviewed here by Gina Abeles, a sociologist at Boston University, who concludes that whenever the concept was operationalized something essential to it seemed to have slipped away. This is because it is not an etiological agent but a language of description that is especially apt for schizophrenia in its family context but good for other situations as well. Like any language, it loses a great deal in the experimental translation from its natural context. This, the editors say in their commentary, is the whole point: the idea is not for testing, it is for thinking. In the rest of the book they collect very elegant examples of this idea.

Alan Watts, Jules Henry, Paul Watzlawick, Morton Schatzman, and Ronald Laing are all represented by fragments that show them at their best; clearly the double bind has taken each of them around an important corner in his explorations. Lyman Wynne presents a very moving article titled "On the Anguish, and Creative Passions, of Not Escaping Double Binds." It is an important piece for therapists.

I found a few disappointments: in his article on the clinical use of the concept Richard Rabkin begins by applying useful criticism to the separate definitions of paradox and double bind, but he promises us a new science of the double bind correctly defined and distinguished and does not deliver. Anthony Wilden and Tim Wilson have a think-tank piece about logic, magic, and economics that in telegraphic terms relates everything to everything else on a breathtaking level of generality that I have not seen since college term-paper days. Their conclusion is that we should prefer cooperation to competition, but if I were not convinced of that to start with I do not think this kind of intellectual tour de force would change my mind.

One piece especially exemplifies what the book is about. Carlos Sluzki and Eliseo Veron in "The Double Bind as a Universal Pathogenic Situation" apply the concept to the genesis of hysteria, phobia, and obsession, taking Fairbairn and learning theory as their point of departure. This shows in action how the double bind concept is useful: it is a general way of thinking about duplicity and unawareness in social systems. As such it has much the same function as the idea of the unconscious in Freudian psychoanalysis.

There is another interesting similarity between the Bateson group and Freud. Both turned aside from their development of a general theory to deal with schizophrenia as a special case. Freud's detour, the analysis of the Schreber case, has caused as much commotion, exegesis, and debate as has the double bind and for the same reason. The etiology of schizophrenia is the Great Prize in the quest of academic psychiatry, a company from which Freud was as removed in his day as was the Bateson group in the early 1950s. Both Freud and the Bateson group made presumptuous and provocative statements on the occasion of their entering the schizophrenia debate from the outside. Now that official psychiatry has accepted them as prophets in their own right, we should not go back and argue their diminishing claims to the etiology of schizophrenia. They have changed the way psychiatrists think about civilization and human relationships, and they—here I think particularly of Haley and Jackson in the Bateson group—have contributed immeasurably to the art and craft of psychotherapy.

This book defines the Bateson group's scientific position in multiple perspectives. It should not be read only for its contribution to the problem of schizophrenia or even the problem of communication and the family. As Weakland says in his final comment, the original paper was not so much a report as a command—to look at things differently. As such, it has put us all in a lasting and therapeutic bind.

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C. CHRISTIAN BEELS, M.D.
New York, N.Y.

The Hospitalized Adolescent: A Guide to Managing the Ill and Injured Youth, *by Adele D. Hofmann, R.D. Becker, and H. Paul Gabriel. New York, N.Y., Free Press (Macmillan Publishing Co.), 1976, 243 pp., $14.95.*

We always seem to be subdividing, specializing, subspecializing—even superspecializing. First there was medicine; then came a cloning into internal medicine and pediatric medicine, then psychiatric medicine and child psychiatry, down to two different limbs, liaison psychiatry and adolescent psychiatry. Must we forever divide, subdivide, and discriminate so discreetly? My conclusion is yes, assuming that proper attention is related to the whole. There is value in specifically focused knowledge and skills, along with a hazard or two.

The Hospitalized Adolescent is truly an important, good book. It is a special book for nonpsychiatric physicians who hospitalize adolescents for medical and surgical problems as well as a good book for those in child, adolescent, or liaison psychiatry who may consult on matters of psychological management of the hospitalized adolescent. The book evolves into a beautiful reweaving of adolescent medicine, psychiatry, and consultation and pulls them all together into a compassionate and knowledgeable statement on caring for the hospitalized adolescent with nonpsychiatric illness.

The book seems to me to be invaluable for every concerned professional dealing with the hospitalized adolescent. Aside from being an excellent primer for teaching purposes, it would be useful for our nonpsychiatric colleagues to read this thoroughly sensitive, well-balanced book. The volume's organization, the clearly labeled selected bibliography at the end of the book, and the detailed case histories woven throughout the text are all handled in an excellent manner. Topics covered include the critical issues of "normal" adolescence; ward interactions among patient, nurse, physician,

and other caretakers; direct patient care advice; a discussion of common behavioral problems; and the neurological, orthopedic, and psychological consequences that present unique problems for the adolescent.

The major reason for urging this book's dissemination among our medical, nursing, and allied professional colleagues is summarized best by Anna Freud's foreword:

> Illness in adolescence, as described by the authors, negates the very progression toward independent adult status which is the aim of this developmental stage. It threatens self-management of bodily functions, freedom from control by external authority; dashes hopes of winning athletic renown or attracting the other sex. No wonder that under these conditions the ill adolescent revolts—against medical or surgical interference which, to him, seems to aggravate matters, not relieve them; against nursing care which seems to return him to the infantile situation of having his body owned and handled by the adults; and against hospital rules which disregard his new need for self-determination. There exists, apparently, no easy way out of this impasse: it is either revolt or utter resignation, depression, and passivity.
>
> The authors of the book accomplish the almost incredible feat of viewing the whole turmoil not from the hospital's, the doctors', or the nurses' side, but from that of the adolescent patients themselves. They are confident that this is the method to circumvent the worst troubles; to allay irrational anxieties; to eliminate unnecessary aggravations; and to create, even in the face of these stressful circumstances, an atmosphere of trust, cooperation, and joint endeavor.

I know of no other resource which contains so much valuable, practical, and easily read information on the problems of the hospitalized adolescent or proposes such sensible preventions and solutions for those special problems.

RICHARD E. DAVIS, M.D.
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Family Therapy: Theory and Practice, *edited by Philip J. Guerin, Jr., M.D. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1976, 547 pp., $24.50.*

A.D. 2001 is not where family therapy is yet, and neither is this book. I question how many books on this topic we need; I own at least 20. This one is current and perhaps 5% ahead of last year's crop. There are 29 contributors and 31 chapters. Nine chapters are written by Murray Bowen or by his students Philip Guerin and Thomas Fogarty.

The book contains many historical and factual inaccuracies (see, for example, the section about the origins of family therapy in Philadelphia, pp. 10, 11). The chapter references have not been standardized, and I spotted several reference errors. However, this book will be valuable to younger family therapists; it is an up-to-date collection of views and practices by a variety of family therapists.

Murray Bowen explains and expands his family systems theory—the I-position, the differentiation of self scale, triangles, the family projection process by which parental undifferentiation impairs one or more of the children, emotional cutoff (a way people handle their unresolved emotional attachments to their parents), and the multigenerational transmission process ("If we follow the most impaired child through successive generations, we will see one line of descent producing individuals with lower and lower levels of differentiation"). Bowen now feels it may take 8 to 10 generations to produce the impairment seen in a schizophrenic person. Other elements of the Bowen theory include the importance of sibling position and his newer concept of societal regression. By his own admission (p. 89), Bowen's theory is not understood by many professionals. Many of his concepts appear to me to be elaborations of the earlier ideas of Harry Stack Sullivan.

John Weakland develops the cybernetic or systems approach to family interaction. Central to this theory is the following: "1) that specific behavior of all kinds is primarily an outcome or function of communicative interaction within a social system; and 2) that 'problems' consist of persisting undesired behavior."

Most of the authors subscribe to a systems model of family therapy, but the personal applications vary greatly. Innovative chapters by Carolyn Attneave, Norman Paul, Lynn Hoffman, Peggy Papp, Peter Laqueur, and others are worth reading. Several chapters focus attention on techniques with children.

This is a book for clinicians. It would probably be best read by sampling a chapter here or there (which I did on my second reading). It compares very favorably to other family therapy texts.

Carl Whitaker's chapter, "The Hindrance of Theory in Clinical Work," will be a remembered classic. I think it is a highlight of this book, dazzling in the professional skill, humor, humanity, and wisdom of its author: "I have a theory that theories are destructive. . . . Look. It's worse than you think. And if you think it's much worse."

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On the Origin of Schizophrenic Psychoses: Symposia Organized by the Interdisciplinary Society of Biological Psychiatry, *edited by H.M. van Praag. Amsterdam, The Netherlands, De Erven Bohn BV, 1975, 222 pp., $18.00 (paper).*

This is a book worth having by any standard, and it will be particularly valuable to most readers of this journal. Edited volumes are, in general, abominations. Some are collections of pieces that edited journals have rejected, and others are proceedings of conferences that took place many years in the past. This volume is of neither sort: it covers two congresses in Amsterdam of the Interdisciplinary Society of Biological Psychiatry held in September 1974 and January 1975. Even with such prompt publication, if this had been a conference in the United States the important contributions would have appeared by now as journal articles in publications generally read by the average American psychiatrist. In this volume, Seymour Kety is the only American contributor, and his sayings wear well with time anyway. The rest of the contributors are European, principally Danish, Dutch, and British, and they provide a remarkably broad and balanced survey of the ways Europeans attack the problem of schizophrenia. Many of these authors appear rarely or not at all in the more popular American psychiatric journals.

Like all symposium reports, the papers are uneven; some have obviously not been reviewed by an editor whose first language is English. However, all are interesting and some are brilliant. Topics range from neurochemistry to psycho-

analysis. Perhaps we have the Dutch tradition of intellectual tolerance to thank for such a refreshingly ecumenical volume, in which Sedwick's commentary on Laing and Cooper can repose cheek by jowl next to Hirsch's paper demonstrating beneficial effects from depot fluphenazine.

Because the volume contains 15 different papers, only a few can be singled out for special comment; this selection is by necessity largely a matter of personal taste. H.M. van Praag is well known to American psychiatrists; his contributions live up to what we have come to expect. I also found Hutt's "An Ethological Analysis of Autistic Behavior" particularly exceptional. The connection he makes between stereotypies and hyperarousal seems neatly defended. I realize now that I should have been more familiar with Hutt's work, but it does not appear regularly in the journals I keep up with. I mention this to illustrate how this volume has brought me in contact with work that my parochial habits have led me to neglect. I suspect other American psychiatrists will find this volume equally useful for similar reasons.

ENOCH CALLAWAY, M.D.
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Behavior and Brain Electrical Activity, *edited by Neil Burch and H.L. Altshuler. New York, N.Y., Plenum Publishing Corp., 1975, 555 pp., $32.50.*

This volume is the result of a symposium dealing with hard quantitative measurement of brain electrical activity. The first portion of the book deals primarily with electroencephalographic measurement of sleep profiles and their relationship to basic endocrine function. The change in electrical activity of the brain as a function of drug effects, biofeedback, and external electrical fields is also considered in a series of contributions. The final section of the volume is concerned with the behavioral patterns of violence and aggression.

The editors attempt to organize the large number of contributions into a meaningful volume in five sections. These include Sleep Profiles and Behavior, Measurement and Analysis of Electrical Activity, Change of State (particularly as induced by drugs and feedback effects), Maturation and Pathology, and Violence and Aggression. There is no question that this volume represents a major review of the important quantitative techniques for analyzing brain electrical activity in relationship to a number of important aspects of human and animal behavior. The contributors include well known and outstanding investigators.

Behavior and Brain Electrical Activity is a useful reference source for students and workers in this important interdisciplinary area of the neurosciences. This volume is recommended for all medical libraries as well as for researchers specifically interested in brain electrical activity, its quantitative measurement, and its relationship to behavior.

EDWARD F. DOMINO, M.D.
Ann Arbor, Mich.

Acute Drug Abuse Emergencies: A Treatment Manual, *edited by Peter G. Bourne, M.D. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1976, 305 pp., $16.50.*

Once in a while a book appears that has potential for repeated editions. *Acute Drug Abuse Emergencies* could well become the equivalent of *Current Therapies* in the field of alcohol and drug abuse treatment. It is, in fact, a second edition, updating *A Treatment Manual for Acute Drug Abuse Emergencies*, which was published by the National Clearinghouse for Drug Abuse Information in 1974 with the encouragement of APA's Task Force on Drugs and Drug Abuse Education. Dr. Bourne did the original editing and organizing of the book while he was Assistant Director of the Special Action Office of Drug Abuse Prevention (SAODAP), long before he attained his current position of influence in the health care system.

The book is organized into 9 sections containing 28 chapters. The initial section on differential diagnosis is followed by sections dealing with opiates, CNS depressants, CNS stimulants, hallucinogens, marijuana, inhalants, alcohol, and special problems. The 41 contributing authors, 16 of whom are psychiatrists, represent a cross section of medical specialties.

The real strength of this book as a clinical reference is its index, which occupies 15% of the pages. Here are to be found useful references ranging from the use of pentylenetetrazol (Metrazol), picrotoxin, caffeine, and strychnine in treating alcoholic intoxication to the entry "zinc sulfate, never use as an emetic (p. 282)." The latter entry will get the reader involved in Eric Comstock's excellent chapter on "Chemotherapy for Drug Abuse Related Emergencies." Browsing through the index can provide informative fun as well as turning up such items as "complications of amphetamine use by athletes (p. 116)" and "psychiatrists more impressed with marihuana's dangers than academicians (p. 154)."

The most distracting aspect of the book is the variability in style and format used by the contributing authors. This variability is most evident in the references and in the organization of each chapter. Dole's chapter on "Management of the Opiate Abstinence Syndrome" has no references, while Glaser's chapter on "Inhalation Psychosis" and Victor's chapter on "Alcohol Intoxication and the Withdrawal Syndrome" each has 3 pages or more of references. Five chapters use the British reference style, which does not include article titles. My own preference is for the *American Journal of Psychiatry* reference style, which is more likely to stimulate me to look up a citation.

I hope that, in addition to exercising more editorial strength in future editions, Dr. Bourne can add some additional clinical material. A chapter on the management of acute medical complications of alcoholism would be useful. Included would be the alcohol-disulfiram (Antabuse) reaction, which unnecessarily frightens many physicians and limits the use of disulfiram as a valuable adjunct to the treatment of alcoholism. The management of neonatal alcohol withdrawal and other CNS depressant withdrawal would help physicians deal with the fetal alcohol syndrome and other newly recognized, related, and unfortunately common conditions. A specific chapter on the management of such behavioral problems as suicide, violence, manipulations, family turmoil, and child abuse might help clinicians feel more comfortable with alcohol- and drug-dependent patients who are often medically underserved as a result of these behaviors.

Acute Drug Abuse Emergencies can be useful for every branch of medical practice. It is of particular value to psychiatrists working in general hospitals, who may be called on by other physicians encountering alcohol or drug abuse problems. Dr. Bourne has made a good start toward the goal he presents in his preface, that of meeting "this largely unmet need in developing more generally disseminated expertise in

aiding the drug abuser." I hope he will continue his work with future editions.

JOHN N. CHAPPEL, M.D.
Reno, Nev.

Understanding EEG: An Introduction to Electroencephalography, *by Donald Scott. Philadelphia, Pa., J.B. Lippincott Co., 1976, 241 pp., $16.00.*

Clinical EEG reports on the charts of many psychiatric patients are frequently misunderstood by the physician who requested them. This is not surprising because the principles of this method are not adequately taught either at the undergraduate level or as a part of psychiatric residency training. Considering the cumulative price of many thousands of EEG records and reports, this is an expensive misunderstanding.

There are good EEG textbooks available, but these were designed primarily for the neurologist specializing in EEG or for the EEG technician. Dr. Scott has written an introductory textbook for the nonspecialist, drawing on his extensive experience with undergraduates and postgraduates at a busy EEG department of a teaching hospital in London.

The introductory chapters on recording technique and general description of normal and abnormal EEG are concise and easy to understand. The chapters on various disease states not only describe the EEG changes but clearly spell out the limitations of the method. False-positive and false-negative findings are discussed, and there is an unusual and very useful chapter titled "The EEG Report" that explains not only how to write one but also how to read one. A detailed and extensive glossary of EEG terms is appended.

Textbooks on EEG generally provide very little information on drug effects; this book is no exception. The EEGs of a majority of psychiatric patients are modified by psychoactive drugs; this complicates the clinical interpretation of the tracings. Additional information on EEG drug effects should be obtained elsewhere (for example, see Fink's essay[1]).

Scott's book is well written and easy to understand. It should be read by those who order EEG reports and expect to understand them.

REFERENCE

1. Fink M: The electroencephalogram in clinical psychiatry, in Biological Psychiatry. Edited by Mendels J. New York, John Wiley & Sons, 1973, pp 331–344

JAN VOLAVKA, M.D., PH.D.
St. Louis, Mo.

Psychotherapy of the Borderline Adult: A Developmental Approach, *by James F. Masterson, M.D. New York, N.Y., Brunner/Mazel, 1976, 369 pp., $17.50.*

In this volume the author travels to the increasingly popular land of the borderline patient. He begins with a 2-chapter outline of an object relations theory of ego development. These are followed by 17 chapters containing reports of both supportive and reconstructive psychotherapy with borderline patients. The final chapter is a restatement of object relations theory with some reference to the many questions about ego organization that remain unanswered. The style is straightforward but repetitious, and there is a tendency in the theoretical sections to gloss over the complexities of conflicting hypotheses.

The author acknowledges his major debt in formulating his theory to Margaret Mahler. He also cites Kernberg at length and refers to Kohut's work. He does not, however, define the relationship between their theories and those of either the classicists or the ego psychologists. In consequence, the system Masterson presents is deceptively simple, as are the treatment approaches that spring from the theory.

By identifying confrontation as the principal technique of the initial phase of treatment, the author seems to encourage an earlier ventilation of aggression than most therapists would advise. It is true that a careful reading of the clinical sessions suggests that, perhaps intuitively, Masterson prepares the borderline patient for confrontational clarification with ego support and by bolstering adaptive responses to anxiety. The sections on theory give little attention to such techniques, however. Also missing from the discussion are criteria for treatability and for termination of therapy.

The achievements of the book are considerably more modest than its cost. Were it not for that deterrent, *Psychotherapy of the Borderline Adult* might be recommended to the novice as a relatively painless introduction to a broad and formidable subject.

ROBERT J. CAMPBELL, M.D.
New York, N.Y.

Empirical Studies of Alcoholism, *edited by Gerald Goldstein and Charles Neuringer. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1976, 256 pp., $16.00.*

This monograph reviews some of the major topics in current psychological research in alcoholism. These include the use of psychological tests for the study of identification, prediction, and treatment; experimental investigations of tension reduction models; behavioral approaches to treatment; perceptual and cognitive deficit; neuropsychological studies of Korsakoff patients; typology of hospitalized alcoholic patients; and neuropsychological investigations.

Neuringer and Clopton review and critique the use of psychological tests for the study of the identification, prediction, and treatment of alcoholism. Included in their discussion are projective techniques (Rorschach, Thematic Apperception Test, Rosenzweig Picture Frustration Test, and Bender-Gestalt), psychometric personality tests, the MMPI (alcoholism scales, the prediction of future alcoholism, and response to treatment), and characteristics of alcoholic subjects identified by objective personality tests. Neuringer and Clopton conclude that, as yet, with the exception of the MMPI, these tests have been found to be of little value in diagnosing alcoholism.

Higgins examines the experimental investigations of tension reduction models of alcoholism, the problem in evaluating the tension reduction hypothesis, stress-induced drinking, and continued drinking in the face of aversive consequences. Following a careful evaluation, Higgins concludes that the specific role of tension reduction in the etiology of either normal or abusive drinking remains obscure.

Denney divides the behavioral approaches to the treatment of alcoholism into three categories: aversion conditioning (chemical, electrical, and verbal), operant procedures (contingent punishment and contingent reinforce-

ment), and broad-spectrum behavioral treatment. Denney views the shift from aversion conditioning to operant conditioning procedures as a fundamental change in treatment orientation and interprets the use of broad-spectrum behavioral treatment as indicating an acceptance of the multiple determinants influencing alcoholism. The following elements have been incorporated into the broad-spectrum treatment approach: specific measures for the physical rehabilitation of the patient; aversion conditioning to decrease the patient's urge to drink; relaxation, desensitization, and aversion training procedures seeking to eliminate the patient's social anxiety; alternative sources of reinforcement (e.g., hobbies and sports); and the enlistment of significant persons in the patient's environment for support. However, Denney emphasizes that this approach may at times be associated with a tendency to overlook the individual patient and rely on shotgun programs.

Goldstein reexamines the nature of the perceptual and cognitive deficit in alcoholic subjects, paying considerable attention to the concept of field dependence and field independence. Although this is a well-established observation, the explanation for this phenomenon is far from clear. One group of investigators view field dependence in alcoholic subjects as a predisposing factor present before acquisition of the alcohol addiction. However, others interpret this phenomenon as a manifestation of some specific consequence of alcoholism and/or situational variables that bear little relationship to the personality of the alcoholic subject or his or her cognitive style before becoming alcoholic.

Goldstein also reviews the alcoholic individual's ability to respond to a variety of testing procedures using different types of configurations and compares the temporal stability of performance levels. His findings suggest that alcoholism is associated with a series of partially reversible perceptual motor and cognitive deficits. He discusses their relation to the understanding of the alcoholic individual. Goldstein reports this characteristic among the more prominent descriptions making up a composite picture of the alcoholic patient who is most likely to drop out of treatment.

Butters and Cermak study the severe memory deficit of alcoholic Korsakoff patients. They center their attentions on the phenomena of anterograde amnesia from the standpoint of short-term memory, proactive interference, and encoding. Their evaluations indicate that Korsakoff patients are sensitive to proactive interference and that they are also deficient in the depth of their encoding. There also appears to be a general limitation in the amount of information Korsakoff patients are capable of processing in any given period of time as well as increasing evidence that Korsakoff patients have visuoperceptive impairments, an observation that suggests these patients may have an impairment in nonverbal memory that parallels their deficit in verbal analysis. Thus it appears that Korsakoff patients may have two separate neuropsychological syndromes: one characterized by anterograde amnesia, short-term memory deficits, and limitation in analysis of verbal materials; the other characterized by deficits on a number of perceptual tests that involve the analysis of complex unfamiliar visual stimuli. Other aspects of this matrix relate to motor memory and olfactory deficits and a comparison of the memory disorders of alcoholic Korsakoff patients, post-encephalitic patients, and patients with Huntington's disease.

Shelly and Goldstein present their findings on an objectively derived typology of hospitalized alcoholic patients. Surprisingly, this indicates that alcoholic patients who were employed immediately before hospitalization in an occupation with relatively high socioeconomic status and who performed in a normal range on the psychological tests were less likely to have a period of abstinence during the follow-up year than were other patients.

Tartar concludes the monograph with a summation of neuropsychological investigations of alcoholism. He finds that the impairment patterns of chronic alcoholic subjects is similar to that of persons suffering from acute brain damage. There is some discussion on the reversibility of the impairment, the rule of antecedent neuropsychological impairment, and the implications of these for treatment.

The 8 chapters are well documented by extensive bibliographies. The presentation is also enriched with 23 figures, 13 tables, and an author and subject index. For the student of alcoholism, the presentation is a scholarly assessment of the psychological progress made in the investigations of alcoholism in recent years. It can be recommended as a useful reference.

ALBERT A. KURLAND, M.D.
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Further Studies of Drug Users, Treatment Typologies, and Assessment of Outcomes During Treatment in DARP. The Effectiveness of Drug Abuse Treatment, Vol. III, *edited by S.B. Sells and D. Dwayne Simpson. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1976, 456 pp., $22.50.*

Evaluation of Treatment Outcomes for 1971–1972 DARP Admission Cohort. The Effectiveness of Drug Abuse Treatment, Vol. IV, *edited by S.B. Sells and D. Dwayne Simpson. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1976, 476 pp., $22.50.*

In the first two volumes of what will be a five-volume series, Sells and Simpson and their coworkers analyzed data on 11,000 patients admitted to 23 federally supported drug treatment programs between June 1969 and June 1971. In volumes III and IV, they present studies on a second cohort of 15,831 patients admitted to 36 federally supported treatment agencies between June 1971 and May 1972. The findings presented here are comparable to those presented in the first two volumes. In the first year of treatment (we will have to wait for volume V for studies extending beyond one year), patients were able to decrease illicit drug use, become less criminally active, and improve their general functioning. However, they could not make significant gains in employment.

In these volumes the authors continue to refine the patient and treatment typologies they developed in the earlier volumes. Four basic treatment modalities are validated: methadone maintenance, therapeutic communities, drug-free treatment, and detoxification. Through the process of field study with identification of relevant factors and complicated statistical analyses the authors arrive at a sophisticated classification of treatments.

In methadone maintenance, for example, they describe two subtypes, methadone maintenance-change oriented (MM-CO) and methadone maintenance-adaptive (MM-A). The essential difference between these treatment approaches is that MM-CO programs place high demands on patient behavior while MM-A programs are "softer." These subtypes are easily identifiable from a clinical perspective, and, as the authors demonstrate, they are effective with different types of patients.

In volume IV a comparison of treatments indicates that methadone maintenance programs are superior to therapeutic communities, drug-free treatment, and detoxification in retaining patients in treatment. Surprisingly, MM-CO programs have higher retention rates than MM-A programs. The finding reported in volume IV that all treatments have some success and that different types of patients respond to different treatments is powerful confirmation of the multimodality concept. One hopes this will have a sobering effect on those who insist that their approach is the only one deserving funding. The data do not confirm the popular idea that alcoholism rates rise in drug-addicted patient populations. Little change was observed between the pretreatment and during-treatment prevalences of alcoholism. The findings in all four volumes of *The Effectiveness of Drug Abuse Treatment* highlight the need for combining treatment for drug abuse and social programming—social programming for repair of educational deficiencies and training for entry into the legitimate job market.

Sells and Simpson's work deserves the widest possible attention. It is methodologically sound, is based on large cohorts of addicts, and represents the best data we have. These data directly contradict the current widespread idea in the lay and medical press that drug abuse treatments, particularly methadone maintenance, are failures.

In addition to a contribution to the field of substance abuse, this work represents a landmark contribution to the assessment of social and mental health services. It will serve as a model for the continuing effort of evaluation and improvement of service efforts for many years to come. Dr. Lois Chatham of the U.S. Department of Health, Education, and Welfare is to be commended for her foresight in generating this effort.

EDWARD C. SENAY, M.D.
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Alcohol and Alcohol Problems: New Thinking and New Directions, *edited by William J. Filstead, Jean J. Rossi, and Mark Keller. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1976, 291 pp, $15.00.*

This book emanates from a 1973 conference at the Alcoholism Rehabilitation Center of Lutheran General Hospital in Des Plaines, Ill. Thus it suffers from the usual defects of such an arrangement: the contributions are four years old and the chapters are uneven, repetitious and, in some cases, in need of editorial surgery.

The book starts out with two interesting historical reviews by Mark Keller and Robert Strauss. Headway is lost, however, in a chapter by Seldon Bacon that, because of its length and wordiness, tends to lose the reader.

Henry Murphree's chapter, "Some Possible Origins of Alcoholism," is in my opinion the high point of the book. In an admirably succinct manner, Murphree reviews the state of knowledge on this vital topic and makes some important observations; e.g., that addiction is not a sufficient reason for alcoholic individuals to continue to drink or to return to drinking after abstinence. His comments about the high achieved with alcohol are worthy of a direct quotation:

> This state is more or less pleasant to large numbers of people, and to my mind it is probably the most common reason why people drink alcoholic beverages. For most, it is merely a harmless, transient state, and the regression may indeed be adaptive. But for some it seems to become so compelling as to override all other considerations, even the elementary needs for food, clothing, and shelter.

How sensible a point of view, yet it has been of only modest interest to clinical researchers.

Mello's chapter on biological research is a brief review that is to a degree repetitive of Murphree's. Of greatest interest here is the possibility that the presence of type IV lipoprotein abnormality may be a marker for people at greatest risk for alcohol-induced injury to the liver, heart, and pancreas. Mello is particularly concerned that basic research, which I gather means biological research, is not being given sufficient weight through federal research funding. She sees some arrogant, faceless bureaucracy as behind this, which will come as a surprise to those conscientious scientists who review proposals and assign priorities to them.

Rossi and Filstead critically review the present state of the art of diagnosis and treatment; they are not very complimentary. They describe the uses of labels and the demand that patients fit them and copouts on treatment failures by blaming the patient's lack of motivation. They also observe that the personal characteristics of the therapist are more important than training and credentials. Although these are reasonably correct observations, I felt they could have been somewhat softened.

The chapter on evaluation by Schuckit and Cahalan is the second high point of the book. It is a primer on program evaluation and a critique of past efforts that would be very helpful to someone about to embark on testing the effectiveness of his or her program. Their plea that we not confuse true program effectiveness with cost-benefit analysis is important today in light of the fact that business managers are invading the health care field with their eyes glued to the bottom line rather than focused on the best treatment outcome.

In the last chapter Gusfield tackles the toughest issue of all—prevention. One can hardly fault him for coming up empty. His observations are that nothing seems to have worked and that projects greeted with optimism have turned out to be "Hawthorne effects." He makes an intriguing suggestion that, lacking a primary prevention strategy, we should put our money into building a world in which alcohol-impaired people are less likely to come to grief. Examples are safer cars and highways and a restructuring of the work week to leave more days for drinking without experiencing absenteeism.

In total, although the subtitle of this book may not have been realized, the general quality of the book—the effort expended by those at the conference—makes it a useful addition to the library of alcohologists.

ROBERT A. MOORE, M.D.
San Diego, Calif.

Rorschach Content Interpretation, *by Edward Aronow, Ph.D., and Marvin Reznikoff, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1976, 340 pp., $19.50.*

The primary purpose of this text is to call attention to "content approaches" of the Rorschach test. According to the introduction, "Hermann Rorschach favored the perceptual factors, and the major emphasis of the Rorschach

schools and Rorschach textbooks has, likewise, been on perceptual factors" (p. xii).

The Rorschach sophisticate reading this text will find it difficult to avoid musing over the history of Roschach's test and the tomes of literature that emerged following the publication of his original text in 1921. *Rorschach Content Interpretation*, by virtue of its erudite format, is equally a tribute to the history of Rorschach's test as it is to the contemporary issues of content interpretation. In the fact that it discusses idiographic concepts (content), this text revisits the belabored issues of the nomothetic versus the idiographic approaches to the study of personality originally described by Allport in 1937. This book gives testament to the fact that the idiographic data of content can be evaluated as well within a nomothetic framework, even though measures of reliability and validity have not proven consistently promising.

This volume is considered the fullest attempt to document, classify, and evaluate the majority of the tomes of literature concerning interest content. Its publication is heralded even from the point of view of its use as a reference source. Beginning with a brief history of inkblot techniques (chapter 1), the authors introduce a variety of approaches in studying content interpretation. They start with the inception of such interpretation by the original investigator, referencing all of the pertinent literature published since concerning each individual method and evaluating the current status of the validity and reliability of each. They accomplish this task with scholarly erudition. The passing parade of Rorschach investigators cited for their original contributions of a variety of content scales in their own research efforts are too numerous to cite in this review; the list would comprise a Rorschach test anthology in itself.

This text opens with a lamenting tone of despair in citing Buros' poignant discounting of the validity of Rorschach's test (p. 1). It closes with a note of equal despair: Laurance Shaffer comments that the test never fulfilled its promise (p. 312). The authors state,

> Clinical Psychology has been led down a blind alley in electing to approach the Rorschach Test primarily from a perceptual standpoint. It is our hope that with the emphasis placed primarily on content and the analysis of verbalizations, the Rorschach Test will, in deed, fulfill its promise as a significant valid instrument." (p. 315)

They conclude that the Holtzman Inkblot Test (HIT) is more amenable to research use, the Rorschach more to clinical applications.

Although Rorschach emphasized the function of perception in his test, he also referred to apperception. The apperceptive functions (such as content) are still partially a function of the perceptual processes in each of the 10 test figures. In fact, chapter 11, which discusses the stimulus characteristics of the Rorschach blots, attests to this interdependency. The validity and reliability of content interpretation is partially dependent on the perceptual stimuli afforded by each card for which there are normative data.

It is surprising that the authors did not seize on the opportunity to refer to one of the most natural and logical theoretical frameworks to emphasize the interdependence between perception and interest content in the Rorschach, i.e., the Gestalt theory of perception. All of the Gestalt processes of leveling, sharpening, and normalizing, basic to the "law of Pregnanz" are involved in the final emergence of interest content to any one specific detail. In this respect, an innovative and most provocative use of Rorschach content from a psychotherapeutic as well as research point of view awaits those who apply the principles of Gestalt psychotherapy to the content interpretation of the Rorschach, especially from the point of view of innovative therapeutic strategies.

H. BARRY MOLISH, PH.D.
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Methods in Brain Research, *edited by Philip B. Bradley. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1975, 544 pp., $47.50.*

In the past quarter century neurobiology has expanded its body of knowledge at an exponential rate. As a result, it is now appropriate to ask not only what is known but how it is known. Because neurobiology has attracted workers from diverse disciplines, the editor of *Methods in Brain Research* concluded that a volume was needed on multidisciplinary approaches to brain research with an emphasis on methodology. During the genesis of this work, several multivolume works on method have appeared. These works plus the desire to limit this effort to a single volume led the editor to choose areas in which important methodological advances have recently been made.

A listing of the chapter headings will give an impression of the diversity of techniques discussed. The volume opens with a chapter on neuroanatomical methods; this chapter focuses on methods of selectively staining various neural structures. Chapters on autoradiography, subcellular fractionation, and microchemical techniques follow. A chapter on cytofluorometry describes microspectrofluorometric analysis, which has been spectacularly used by Swedish workers. A chapter cryptically titled "Collection Techniques" deals with the isolation of active substances, usually neurotransmitters. Electrophysiology has become old hat and receives attention only in a discussion of microelectrode techniques. The last of the micromethods deals with using tissue culture to provide various "model" neural systems. The four remaining chapters deal with behavioral techniques, including operant conditioning; a relatively brief chapter on the study of memory consolidation using intracranial self-stimulation; neuroethology, which addresses itself to the role of neural mechanisms in the normal behavior of animals in their natural environment; and an exceedingly brief chapter on psychosurgery.

The volume traverses the usual perils of a work by many authors. The editor has rejected the cookbook approach, but the individual authors have interpreted this mandate with varying degrees of rigor ranging from such descriptions as "centrifuge 56,000g × 120 min, synaptic membranes recoverable from the 1.2–1.0 and the 1.0–0.9 M interfaces" to an introspective, unfocused, and inconclusive attempt to decide whether or not "psychosurgery" should be performed on human beings. Chapters that fall in between such extremes prove useful and can provide a deeper understanding of the bases and limitations of various methodologies in neurobiology. On balance, the volume may fall to the charge that it is too complex for the uninitiated and inadequate for the cognescenti.

DAVID D. DALY, M.D., PH.D.
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The Handbook of Gestalt Therapy, *edited by Chris Hatcher and Philip Himelstein. New York, N.Y., Jason Aronson, 1976, 795 pp., $25.00.*

The publication of a handbook is an important event for a body of knowledge because it indicates a new maturity. Thus the appearance of *The Handbook of Gestalt Therapy* is to be greeted with enthusiasm by those who have been interested in and followed the development of the subject. This handbook should be especially useful to those seeking to learn about Gestalt therapy for the first time. Articles by Joen Fagan and Claudio Naranjo on technique deserve special attention for their clarity and incisiveness; there are also many other excellent papers. A special strength of the book is its excellent index—something that has been sadly lacking in previous writings on Gestalt therapy. There is also a rather complete listing of films, audiovisual materials, and writings available on the subject. The book is divided into four parts: The Contributions of Frederick S. Perls, Contemporary Theory, Techniques, and Perspectives and Resources.

This handbook is really a collection of previously published articles on Gestalt therapy from books and journals. Only 11 of the 32 chapters were written specifically for the handbook. Although generally good, the work is marred by the spotty quality of some of the chapters. leaving the reader to wonder why they were included at all. It is also difficult to understand why the authors selected these articles to represent the whole of Gestalt therapy. For example, in 200 pages on the contributions of Perls, 1 chapter was selected from each of 3 of his books, *Gestalt Therapy Verbatim* (1), *Gestalt Therapy* (2), and *In and Out of the Garbage Pail* (his autobiography) (3). No explanation is given for the selection of these chapters over others. The grouping of articles that were written for many different purposes produces a hodgepodge of styles that detracts from the book's readability. One might say that the book as a whole does not form a very cohesive Gestalt (sic).

The editors of the handbook, who are Gestalt therapists, manifest in this volume an often observed phenomenon. True to their therapeutic perspectives, Gestalt therapists often write for themselves rather than for the reader. They are concerned with the experience of writing rather than with the product itself, thereby placing the purposes of therapy and publication in conflict. This book is representative of many of the problems with Gestalt therapy writings. Authors seem to be less concerned with a smoothly reading product than with the process and experience of production. For example, an interview with Jim Simkin about the history of Gestalt therapy contains five different notations such as "(unclear)" and "(too low)" on the first page. Would it really have been too much to expect of the editor to return to the source to obtain an understandable response?

In the last section of the book an article by Steven Applebaum compares psychoanalysis with Gestalt therapy and reveals the obvious: the two are much more closely related than the proponents of either have been willing to admit. It has always seemed that Gestalt therapy could easily be viewed as an extension of psychoanalytic ego psychology if it had not been for matters of personality and power. The separation of these bodies of knowledge is more political than theoretical, and each is poorer for it.

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ARNOLD R. BEISSER, M.D.
Los Angeles, Calif.

Mental Health Information Systems: Design and Implementation, *by David J. Kupfer, Michael Levine, and John A. Nelson. New York, N.Y., Marcel Dekker, 1976, 151 pp., $19.50.*

This book is the 19th volume in a series of monographs and textbooks on library and information science edited by Allen Kent, Director of the Office of Communication Programs at the University of Pittsburgh. In five chapters the authors present an overview of information systems and mental health programs that includes system design considerations, suggestions on how to support an information system, and a description of the development of specific systems at the Connecticut Mental Health Center in New Haven and the Western Psychiatric Institute and Clinic in Pittsburgh. In these times of strange bedfellows—administrators, insurance carriers, government, accreditation agencies, lawyers, and enormous health care delivery systems—the book presents a valuable overview of what to do and what to avoid in the development of a comprehensive information system.

Although the authors assert that the primary aim of a management information system in mental health should be the improvement of patient care, and some of Kupfer's research papers may certainly achieve that goal, the reader is left with the feeling that, at present, such systems are primarily useful to placate and relate with management, administration, insurance carriers, government accreditation agencies, and the law. Thus a management information system is a series of carefully planned procedures that facilitate the systematic gathering of data that can be processed into information that, in turn, enables people to make the institution run effectively. The management information system is actually used for the following purposes: 1) to produce reports for the charts, 2) to monitor quality of care, 3) to provide a data base for evaluation studies and clinical research, 4) to gather data in order to strengthen the fiscal system, 5) to provide data for administrative planning, and 6) to provide information for teaching.

From reading this book one senses the critical interstitial role of information as an administrative and management tool, as a means of interdisciplinary communication, and as a necessity for accountability, peer review, and audit studies. Its impact on patient care is very hard to evaluate.

Of considerable interest are the historical insights into the development of the systems in Connecticut and Pittsburgh. The evolution of the position of director of information at Pittsburgh and the single-portal entry system for workup and case disposition described are particularly worthy of consideration. The book is recommended as an introduction to the KDS forms and charting system. These should be seriously considered by all who anticipate changing currently inadequate medical records.

MONTE JAY MELDMAN, M.D.
Des Plaines, Ill.

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Chemotherapy in Psychiatry, *by Ross J. Baldessarini, M.D. Cambridge, Mass., Harvard University Press, 1977, 196 pp., $9.95.*

Psychotherapy and Growth: A Family Systems Perspective, *by W. Robert Beavers, M.D. New York, N.Y., Brunner/Mazel, 1977, 378 pp., $17.50.*

Hypnosis for the Seriously Curious, *by Kenneth S. Bowers. Monterey, Calif., Brooks/Cole Publishing Co. (Wadsworth Publishing Co.), 1976, 166 pp., $6.95 (paper).*

Family Dance: Building Positive Relationships Through Family Therapy, *by Warren M. Brodey. New York, N.Y., Anchor Press (Doubleday & Co.), 1977, 138 pp., $2.95 (paper).*

Human Sexuality: New Directions in American Catholic Thought, *commissioned by the Catholic Theological Society of America. Ramsey, N.J., Paulist Press, 1977, 316 pp., $8.50.*

Hero, Artist, Sage, or Saint? A Survey of Views on What Is Variously Called Mental Health, Normality, Maturity, Self-Actualization, and Human Fulfillment, *by Richard W. Coan. New York, N.Y., Columbia University Press, 1977, 316 pp., $20.00; $6.50 (paper).*

Nebraska Symposium on Motivation 1976, *edited by James K. Cole and Alvin W. Landfield. Lincoln, Neb., University of Nebraska Press, 1977, 359 pp., $16.95; $7.95 (paper).*

Jewish Ethno-Psychiatry: A Manual for Inservice and House Staff Education, *by the Federation of Jewish Philanthropies of New York, Commission on Synagogue Relations, Task Force on Mental Health and Judaism, Committee on Residency Training. New York, N.Y., Federation of Jewish Philanthropies, 1977, 80 pp., $3.00 (paper).*

The Septal Nuclei. Advances in Behavioral Biology, Vol. 20, *edited by Jon R. DeFrance. New York, N.Y., Plenum Press, 1976, 536 pp., $39.50.*

Crime and Personality, *by H.J. Eysenck. Boston, Mass., Routledge & Kegan Paul, 1977, 217 pp., $8.95.*

Adolescent Psychiatry, Vol. V: Developmental and Clinical Studies, *edited by Sherman C. Feinstein and Peter L. Giovacchini. New York, N.Y., Jason Aronson, 1977, 487 pp., $20.00.*

Psychiatric Drugs for the Non-Medical Mental Health Worker, *by William Goldsmith, M.D. Springfield, Ill., Charles C Thomas, 1977, 162 pp., $11.50.*

Profiles in Social Work, *by Mary L. Gottesfeld and Mary E. Pharis. New York, N.Y., Human Sciences Press, 1977, 238 pp., $14.95.*

Is This Really What I Want to Do? *by Dr. Jerry Greenwald. Pasadena, Calif., Ward Ritchie Press, 1977, 207 pp., $8.95.*

Uncommon Therapy: The Psychiatric Techniques of Milton H. Erickson, M.D., *by Jay Haley. New York, N.Y., Norton Library (W.W. Norton & Co.), 1977, 313 pp., $3.95 (paper).*

The Physician and Sexuality in Victorian America, *by John S. Haller and Robin M. Haller. New York, N.Y., Norton Library (W.W. Norton & Co.), 1977, 324 pp., $3.95 (paper).*

Existential Metapsychiatry, *by Thomas Hora. New York, N.Y., Seabury Press, 1977, 234 pp., $12.95.*

Freud and the Dilemmas of Psychology, *by Marie Jahoda. New York, N.Y., Basic Books, 1977, 180 pp., $10.00.*

Love, Guilt and Reparation and Other Works, 1921–1945, *by Melanie Klein. New York, N.Y., Delta (Dell Publishing Co.), 1977, 444 pp., $4.95 (paper).*

The Restoration of the Self, *by Heinz Kohut. New York, N.Y., International Universities Press, 1977, 327 pp., $17.50.*

Psychopathology and Politics, *by Harold D. Lasswell. Chicago, Ill., University of Chicago Press, 1977, 335 pp., $5.95 (paper).*

Ethological Psychiatry: Psychopathology in the Context of Evolutionary Biology, *edited by Michael T. McGuire, M.D., and Lynn A. Fairbanks, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1977, 219 pp., $15.00.*

The Sleep Instinct, *by Ray Meddis. Boston, Mass., Routledge & Kegan Paul, 1977, 143 pp., $9.75.*

Human Sleep and Its Disorders, *by Wallace B. Mendelson, J. Christian Gillin, and Richard Jed Wyatt. New York, N.Y., Plenum Press, 1977, 255 pp., $19.50.*

The Freudian Paradigm: Psychoanalysis and Scientific Thought, *edited by Md. Mujeeb-ur-Rahman, Ph.D. Chicago, Ill., Nelson-Hall, 1977, 404 pp., $17.50; $9.95 (paper).*

From Teenage to Young Manhood: A Psychological Study, *by Daniel Offer, M.D., and Judith Baskin Offer, with the assistance of Eric Ostrov, Ph.D. New York, N.Y., Basic Books, 1975, 254 pp., $12.95.*

Emerging Concepts of Alcohol Dependence, *by E. Mansell Pattison, Mark B. Sobell, and Linda C. Sobell. New York, N.Y., Springer Publishing Co., 1977, 352 pp., $18.95.*

A Reassessment of the Concept of Criminality, *by Eggert Petersen. Copenhagen, Denmark, Munksgaard (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1977, 134 pp., no price listed (paper).*

The Healing Alliance, *by Margaret Elmendorf Raymond, M.S.W., Andrew Edmund Slaby, M.D., M.P.H., and Julian Lieb, M.D. New York, N.Y., Norton Library (W.W. Norton & Co.), 1977, 287 pp., $3.95 (paper).*

A Short Textbook of Psychiatry, 2nd ed., *by W.L. Linford Rees. Philadelphia, Pa., J.B. Lippincott Co., 1976, 327 pp., $7.50 (paper).*

Habilitation of the Handicapped: New Dimensions in Programs for the Developmentally Disabled, *by Marvin Rosen, Gerald B. Clark, and Marvin S. Kivitz. Baltimore, Md., University Park Press, 1977, 366 pp., $18.50.*

Future Facts: A Forecast of the World As We Will Know It Before the End of the Century, *by Stephen Rosen. New York, N.Y., Touchstone (Simon and Schuster), 1976, 522 pp., $5.95 (paper).*

Group Psychotherapy and Group Function, revised ed., *edited by Max Rosenbaum and Milton M. Berger. New York, N.Y., Basic Books, 1975, 794 pp., $15.00.*

Reading Disability: A Human Approach to Learning, 3rd ed., *by Florence G. Roswell and Gladys Natchez. New York, N.Y., Basic Books, 1977, 300 pp., $13.50.*

The Clinical Management of Muscle Disease: A Practical Manual of Diagnosis and Treatment, *by Irwin M. Siegel, M.D. Philadelphia, Pa., J.B. Lippincott Co., 1977, 156 pp., $10.00.*

The Silva Mind Control Method, *by José Silva and Philip Miele. New York, N.Y., Simon and Schuster, 1977, 200 pp., $8.95.*

Understanding Human Behavior in Health and Illness, *edited by Richard C. Simons, M.D., and Herbert Pardes, M.D. Baltimore, Md., Williams & Wilkins Co., 1977, 704 pp., $21.95.*

Organizing Against Crime: Redeveloping the Neighborhood, *by Anthony Sorrentino. New York, N.Y., Human Sciences Press, 1977, 267 pp., $14.95.*

C.G. Jung: The Haunted Prophet, *by Paul J. Stern. New York, N.Y., Delta (Dell Publishing Co.), 1977, 262 pp., $3.95 (paper).*

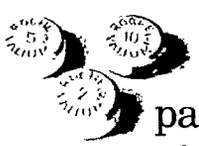
Biofeedback: How to Control Your Body, Improve Your Health, and Increase Your Effectiveness, *by Robert M. Stern, Ph.D., and William J. Ray, Ph.D. Homewood, Ill., Dow Jones-Irwin, 1977, 191 pp., $9.95.*

The Mental Status Examination in Neurology, *by Richard L. Strub, M.D., and F. William Black, Ph.D. Philadelphia, Pa., F.A. Davis Co., 1977, 173 pp., $7.95 (paper).*

Bringing Up Children Overseas: A Guide for Families, *by Sidney Werkman, M.D. New York, N.Y., Basic Books, 1977, 215 pp., $9.95.*

Pediatric Psychopharmacology: A Practical Guide to Clinical Application, *by James H. White, M.D. Baltimore, Md., Williams & Wilkins Co., 1977, 178 pp., $13.50 (paper).*

Mental Retardation and Developmental Disabilities: An Annual Review, Vol. IX, *edited by Joseph Wortis, M.D. New York, N.Y., Brunner/Mazel, 1977, 320 pp., no price listed.*



In the psychoneurotic patient, the pattern of anxiety – psychophysiological complaints and secondary depressive symptoms – often responds to Valium®(diazepam)

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Before prescribing, please consult complete product information, a summary of which appears on the following page.

In the psychoneurotic patient

Valium® (diazepam) IV

2-mg, 5-mg, 10-mg scored tablets

can relieve anxiety and its secondary depressive symptoms

Initial calming in hours

Your anxious psychoneurotic patient will find the prompt antianxiety action of Valium (diazepam) highly reassuring. It's immediate, tangible proof that his medication is working.

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You've come to expect a certain kind of response with Valium—and that's the kind you usually get. A pronounced antianxiety response. A response that starts to benefit the anxious patient almost immediately.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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Coverage and Utilization of Care for Mental Conditions Under Health Insurance —Various Studies, 1973–74

By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

This book reports six studies that add to the growing body of data demonstrating the feasibility of covering mental illness under health insurance. Although in some ways it may be considered a supplement to APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (Reed, Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

- Utilization of mental benefits under the Blue Cross and Blue Shield plan for federal employees, documenting that a "plateau" in the use of these benefits was reached in 1973–74.
- Comparison of benefits for mental and other illnesses under selected employee health benefit plans.
- Coverage of mental illness under collective bargaining agreements of selected unions.
- Utilization of care for mental conditions under the Canadian health insurance program, which gives the same coverage for mental as for other conditions.
- Updating of information on Blue Cross benefits for hospital care of mental illness.
- Data from selected Blue Cross and Blue Shield plans on coverage and utilization of mental conditions, with emphasis on major medical coverage.

80pp. Paperbound

Single copy, $4.00; 10–49 copies; $3.80 each; 50 or more copies, $3.20 each.

SPECIAL OFFER: One copy of this book (regular price, $4.00) and one copy of **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (regular price, $6.50) for $8.50.

Publications Services Division
American Psychiatric Association
1700 18th St., N.W., Washington, D.C. 20009

Please send me _____ copies of **COVERAGE AND UTILIZATION OF CARE FOR MENTAL CONDITIONS UNDER HEALTH INSURANCE—VARIOUS STUDIES, 1973–74**. Single copy, $4.00; 10–49 copies, $3.80 each; 50 or more copies, $3.20 each.

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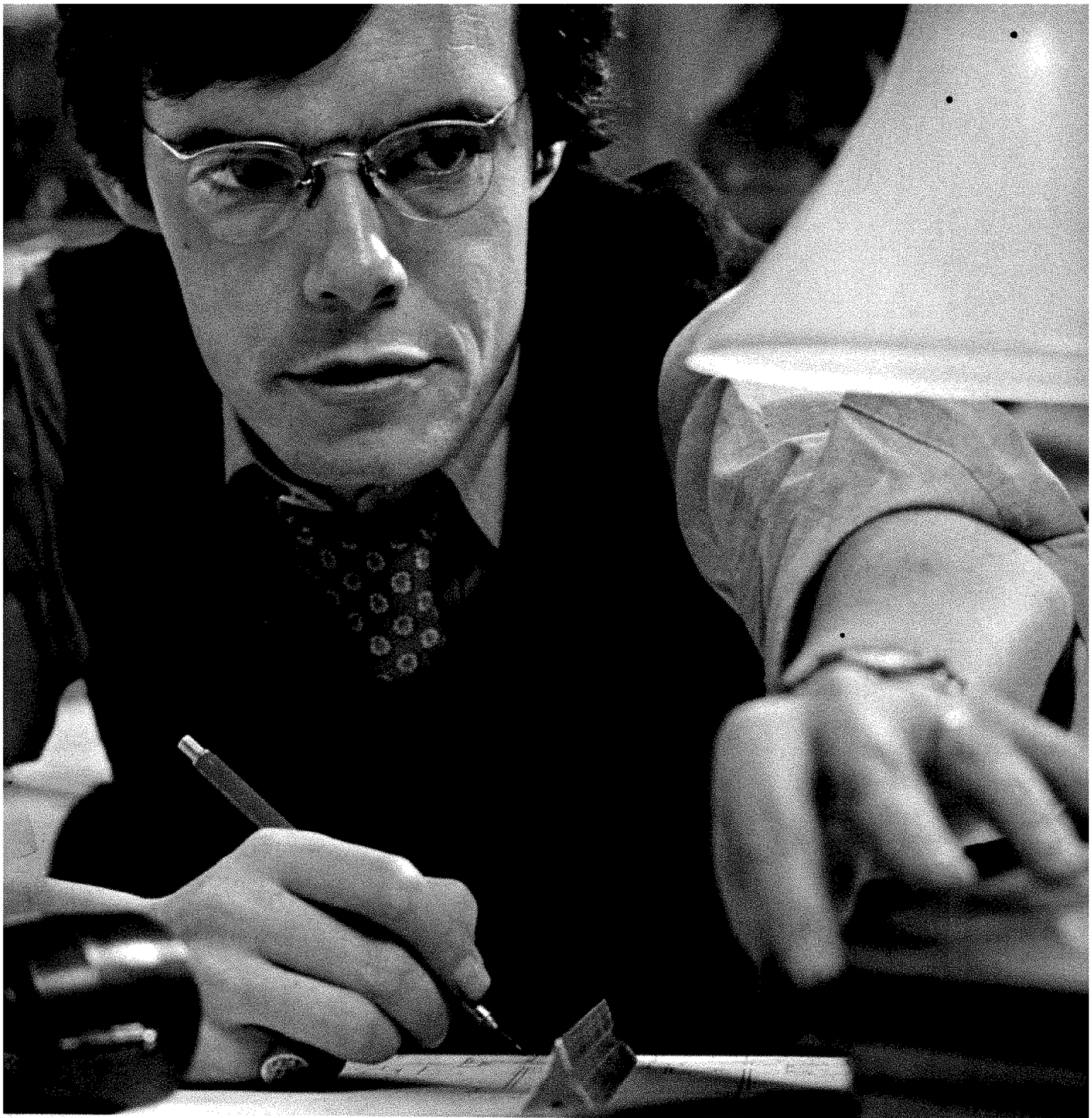
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Phillip H.* was an engineer with a problem... but now he's solving problems for industry

Patient name Phillip H.*	**Sex:** Male	**Age:** 29
Marital status S. (M.) W. D.	**Occupation:** Mechanical engineer	

History:	Studious, bright, only son; much older sister. Father an invalid; mother, a successful business woman. A "loner" with multiple psychosomatic illnesses, he started dating late and married first girl friend, several years older
Onset of illness:	Upset at wife's pregnancy, expressed strong guilt feelings after her miscarriage; said people accused him of "murder." Unable to eat or sleep properly, felt he was "losing his mind." Unable to work, obtained leave of absence and was referred to psychiatrist
Diagnosis:	Acute schizophrenia
Treatment:	LOXITANE® Loxapine Succinate 25 mg b.i.d. increased to 50 mg t.i.d. over 2 weeks. Supportive psychotherapy three times a week
Therapeutic course:	First week, eating and sleeping better. Guilt feelings expressed less often. Mild akathisia responded to appropriate antiparkinson medication
	Second week, communicating better. Feared that "fatherhood would develop into rivalry," feared loss of wife's love
	Third week, no delusions or hallucinations, relating better, but still to only one person at a time
	Affect still blunted at one month, otherwise asymptomatic. LOXITANE decreased to 50 mg b.i.d.; antiparkinson medication continued
Follow-up:	10 weeks; at work for one week, seems to be adjusting well. Antiparkinson medication discontinued; psychotherapy weekly, LOXITANE 25 mg t.i.d.

*Not an actual patient. The collection of clinical data upon which this simulation is built illustrates the action of LOXITANE® Loxapine Succinate as reported in clinical studies.



He's adjusting well in his job, and with psychotherapy and medication, he also can be helped to feel more secure, less threatened in his relationships with others.

Loxitane®
LOXAPINE Lederle SUCCINATE

For the symptoms that interrupt careers

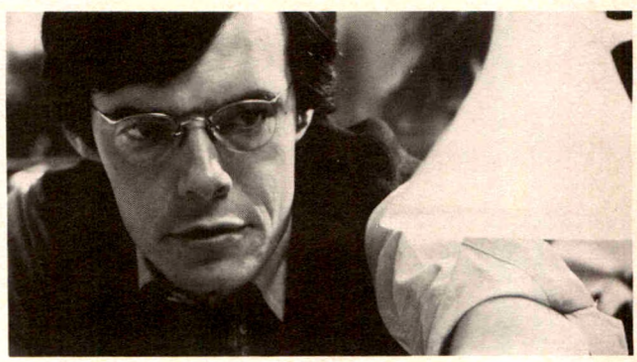
See LOXITANE prescribing information on following page for indications, warnings and precautions and for more detailed information concerning side effects.

Loxitane®
LOXAPINE SUCCINATE
Lederle

For the symptoms that interrupt careers

Recommended Daily Dosage

Initial Dosage	MILD 10 mg b.i.d.	MODERATE 10 mg t.i.d. or q.i.d.	SEVERE 25 mg b.i.d.
First 7 to 10 Days	Increase dosage until psychotic symptoms are controlled. Dosage should not exceed 250 mg/day. Usual dosage during titration: 50 to 150 mg/day.		
Maintenance Dosage	Adjust to lowest effective level. Usual maintenance dosage: 60 to 100 mg/day. Many patients are controlled with dosages as low as 20 to 60 mg/day.		



Brief Summary

LOXITANE® Loxapine Succinate Capsules
OR
LOXITANE® C Loxapine Hydrochloride Oral Concentrate

Description: A dibenzoxazepine compound, representing a new subclass of tricyclic antipsychotic agent, chemically distinct from the thioxanthenes, butyrophenones, and phenothiazines.

Indications: For the manifestations of schizophrenia. Loxapine has not been evaluated for the management of behavioral complications in mental retardation, and therefore cannot be recommended.

Contraindications: In comatose or severe, drug-induced depressed states (alcohol, barbiturates, narcotics, etc.); and in individuals with known hypersensitivity to the drug.

Warnings: In Pregnancy: Safe use during pregnancy or lactation has not been established; in pregnancy, nursing mothers, or women of child-bearing potential, weigh potential benefits against possible hazards to mother and child. No embryotoxicity or teratology was observed in studies in rats, rabbits or dogs, although with the exception of one rabbit study, the highest dosage was only two times the maximum recommended human dose and in some studies they were below this dose. Perinatal studies have shown renal papillary abnormalities in offspring of rats treated from mid-pregnancy with doses of 0.6 and 1.8 mg./kg., doses which approximate the usual human dose but which are considerably below the maximum recommended human dose. **In Children:** Studies have not been performed in children; therefore this drug is not for use below the age of 16.

May impair mental and/or physical abilities, especially during the first few days of therapy; warn ambulatory patients about activities requiring alertness (*e.g.*, operating vehicles or machinery), and about concomitant use of alcohol and other CNS depressants.

Precautions: Use with extreme caution in patients with history of convulsive disorders; seizures have been reported in epileptics receiving this drug at antipsychotic dose levels, and may occur even with maintenance of routine anticonvulsant drug therapy. Has an antiemetic effect in animals; this effect in man may mask signs of overdosage of toxic drugs and obscure conditions such as intestinal obstruction and brain tumor. Use with caution in patients with cardiovascular disease. Increased pulse rates in the majority of patients receiving antipsychotic doses and transient hypotension have been reported. In hypotension requiring vasopressor therapy, preferred drugs may be norepinephrine or angiotensin. Usual doses of epinephrine may be ineffective because of inhibition of its vasopressor effect by loxapine. Possibility of ocular toxicity from loxapine cannot be excluded at this time, therefore observe carefully for pigmentary retinopathy and lenticular pigmentation (observed in some patients receiving certain other antipsychotic drugs for prolonged periods). Because of possible anticholinergic action, use with caution in patients with glaucoma or a tendency to urinary retention, particularly with concomitant administration of anticholinergic-type antiparkinson medication.

Adverse Reactions: CNS: Adverse effects other than extrapyramidal, infrequent. Drowsiness, usually mild, may occur at beginning of therapy or when dosage is increased, usually subsiding with continued therapy. Incidence of sedation is less than that of certain aliphatic phenothiazines and slightly more than piperazine phenothiazines. Dizziness, faintness, staggering gait, muscle twitching, weakness, and confusional states have been reported. Extrapyramidal reactions during use of this drug have been reported frequently, often during the first few days of treatment. In most patients, these involve Parkinson-like symptoms such as tremor, rigidity, excessive salivation, masked facies, akathisia, usually not severe and controlled by dosage reduction or use of antiparkinson drugs in usual dosage. *Less frequent, but more severe:* Dystonias, including spasms of muscles of neck and face, tongue protrusion, oculogyric movement; dyskinesia in the form of choreo-athetoid movements. These sometimes require reduction or temporary withdrawal of drug dosage in addition to appropriate counteractive drugs. *Persistent Tardive Dyskinesia:* May appear in some patients on long-term therapy, or after therapy has been discontinued; the risk greater in the elderly, especially females, on high dosage. These symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of tongue, face, mouth or jaw (*e.g.*, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements, sometimes accompanied by involuntary movements of extremities). No known effective treatment; discontinue all antipsychotic agents if these symptoms appear. The necessity to reinstitute treatment or increase dosage, or switch to a different antipsychotic agent, may mask syndrome. If medication is stopped when fine vermicular movements of the tongue occur, the syndrome may not develop. *Cardiovascular:* Tachycardia, hypotension, hypertension, lightheadedness, syncope. A few cases of EKG changes similar to those seen with phenothiazines have been reported, not known to be related to loxapine use. *Skin:* Dermatitis, edema (puffiness of face), pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of uncertain etiology seen in a few patients during hot summer months. *Anticholinergic:* Dry mouth, nasal congestion, constipation, blurred vision—more likely to occur with concomitant use of antiparkinson agents. *Other:* Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of uncertain etiology.

Dosage and Administration: Administered orally, usually in 2 to 4 divided doses a day. Adjust dosage to patient's need relative to severity of symptoms and history of response to antipsychotic drugs. Recommended initial dosage is 10 mg. b.i.d.; in severely disturbed patients up to 50 mg. daily. Increase dosage during first 7 to 10 days until psychotic symptoms are controlled. Usual therapeutic and maintenance range is 60 mg.-100 mg. daily. More than 250 mg. daily is not recommended. Maintenance dosage should be at lowest level to control symptoms; many patients have been maintained on 20 mg. to 60 mg. daily.

LOXITANE C Oral Concentrate should be mixed with orange and grapefruit juice shortly before administration. Use only enclosed calibrated dropper.

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Pearl River, New York 10965

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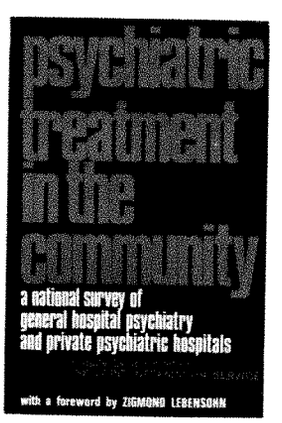
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Where The Action Is—

For more than a decade the general hospital psychiatric unit has been the principal locus of inpatient treatment. This latest Joint Information Service national survey brings up to date its earlier—and the original—study of this important component of the psychiatric scene. It reveals that the remarkable rate of growth held up, showing an increase of 50 percent in the number of units between 1963 and 1971, and an increase of 46 percent in the number of admissions. And it verifies that general hospital psychiatry has become increasingly comprehensive, with an impressively high level of outpatient service, emergency service, and even consultation to community agencies. Indeed, the general hospital accounts for several times as much service and activity as the widely heralded federally supported community mental health center.

This study also involves the private psychiatric hospitals, which have not changed much in number but are admitting about 10,000 more patients than in 1964—and they, too, are providing a remarkably comprehensive program.

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...with a low incidence of adverse side effects on

MOBAN®
molindone HCl

- an effective antipsychotic agent
- not a phenothiazine

FAVORABLE BENEFIT/RISK PROFILE, WHICH MAY ENHANCE COMPLIANCE*

NO incidence	Tolerance development Hepatotoxic changes Ophthalmological changes Excessive weight gain Impotence Decreased libido Skin pigmentation Thyroid function changes Persistent tardive dyskinesia
LOW incidence	Sedation Adverse drug interactions Hypotension Hematological changes Lactation Amenorrhea Autonomic changes Skin rash Nonpersistent symptoms of tardive dyskinesia
MODERATE incidence	Extrapyramidal reactions

*This table compiled from clinical and field reports, according to the best available information.

Further advantages with MOBAN®

The pharmacological effect from a single oral dose persists for 24-36 hours, permitting *h.s.* dosage, once the daily dosage level is established.

Several clinicians have reported that obese patients on MOBAN® frequently experienced a marked reduction in weight toward normal weight.

How to use MOBAN®

The usual starting dose is 50-75 mg/day. You can increase to 100 mg/day in 3-4 days. Titrate individually up or down depending on patient response. Some chronic, treatment-resistant patients may require an increase to 400 mg/day. (However, the long-term safety of 400 mg/day has not been established.) Please refer to complete prescribing information for further dosage instructions.

For complete prescribing information, please see next page

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DUPONT

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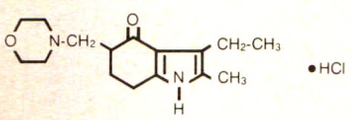
MOBAN®

molindone HCl

5 mg	10 mg	25 mg
Endo	Endo	Endo

DESCRIPTION MOBAN® (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN® is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN® (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN® antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN® exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN® (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN® being excreted in urine and feces.

INDICATIONS MOBAN® (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN® (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in the pregnant patient have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN® (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN® (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS Some patients receiving MOBAN® (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN®. Caution should be exercised where increased activity may be harmful.

MOBAN® does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN® has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological

Extrapyramidal Reactions

Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia

Motor restlessness may occur early.

Parkinson Syndrome

Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome

Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System

Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological

There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN® may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects

Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with MOBAN® therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN®.

Cardiovascular

Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological

Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN® (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN®.

Skin

Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN® usage alone.

MOBAN® (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN® is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia

Although rarely reported with MOBAN® (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN® (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50-75 mg/day.

—Increase to 100 mg/day in 3 or 4 days.

—Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.

—An increase to 225 mg/day may be required in patients with severe symptomatology.

—Some chronic, treatment resistant patients, may require up to 400 mg/day (however, the long term safety of 400 mg/day has not been established).

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

Dosages may be administered once a day.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN® (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN® is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN® (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN® (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidote must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, while the emetic effect of apomorphine is blocked by MOBAN® in animals, this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN® from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN® is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based on general principles, the amount of unmetabolized MOBAN® in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles of 100 and 1000 with potencies and colors as follows: 5 mg orange, 10 mg lavender, 25 mg light green.

*Benadryl—Trademark, Parke Davis and Co.
*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
*Akineton—Trademark, Knoll Pharmaceutical Co.

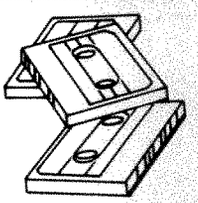
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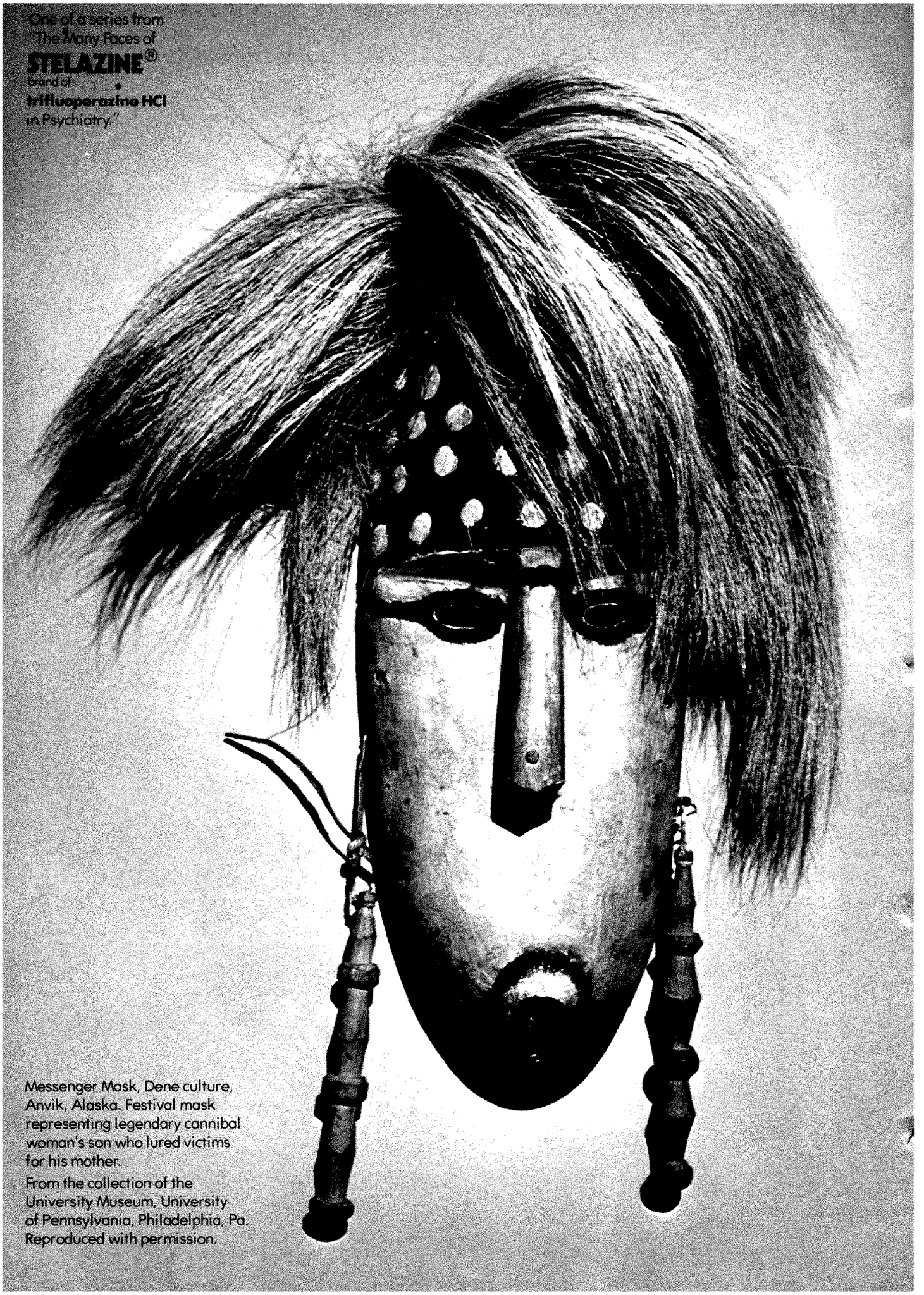
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Messenger Mask, Dene culture, Anvik, Alaska. Festival mask representing legendary cannibal woman's son who lured victims for his mother.
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REMOVE THE MASK OF SCHIZOPHRENIC SYMPTOMS

'STELAZINE' PROVIDES EFFECTIVE CONTROL OF HALLUCINATIONS, DELUSIONS, ANXIETY AND OTHER SCHIZOPHRENIC SYMPTOMS IN A WIDE RANGE OF PATIENTS.

FROM THE WITHDRAWN AND APATHETIC TO THE ANXIOUS AND OVERACTIVE, 'STELAZINE' HELPS RETURN THE PSYCHOTIC PATIENT TO REALITY AND CAN FACILITATE THERAPEUTIC CONTACT AND RAPPORT.

- EFFECTIVELY CONTROLS PSYCHOTIC SYMPTOMS
- SELDOM CAUSES EXCESSIVE SEDATION
- CONVENIENT B.I.D. DOSAGE

Before prescribing, see complete prescribing information in SK&F literature or *PDR*. The following is a brief summary.

Indications
Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudo-parkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions; peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 1 mg., 2 mg., 5 mg. and 10 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only); Injection, 2 mg./ml.; and Concentrate (intended for institutional use only) 10 mg./ml.

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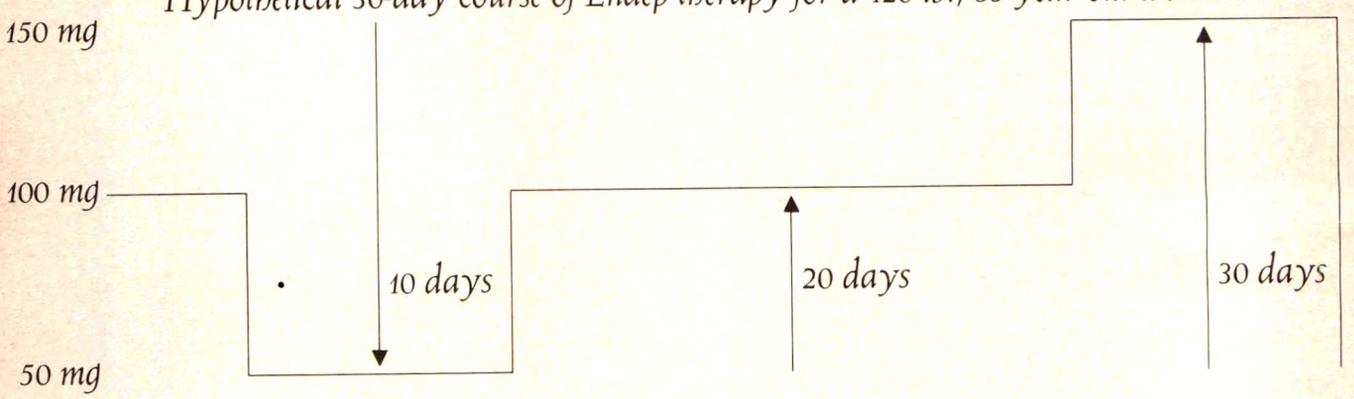
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Helps schizophrenic patients become more responsive.

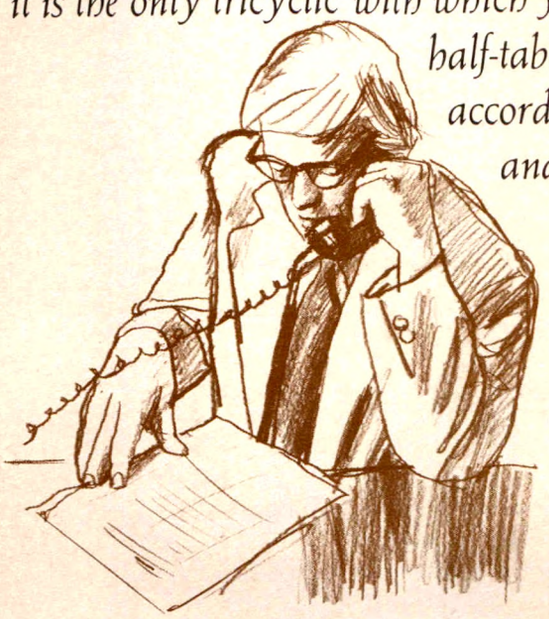
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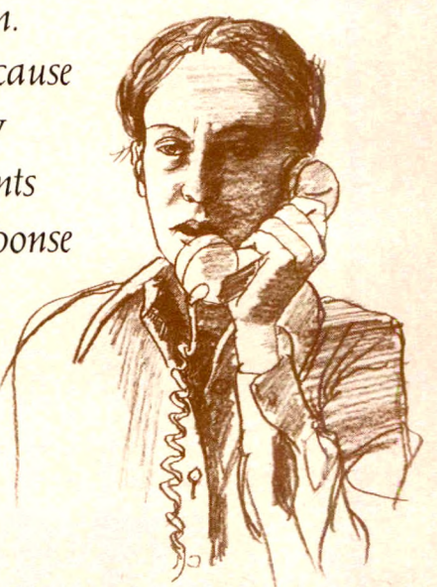
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Half-dose adjustments h.s., depending on progress or side effects.

Because therapeutic levels of amitriptyline vary greatly from patient to patient, reaching the optimal dose requires careful titration. Endep (amitriptyline HCl) greatly facilitates this process because it is the only tricyclic with which you can adjust dosage by half-tablet increments or decrements according to the therapeutic response and/or side effects.





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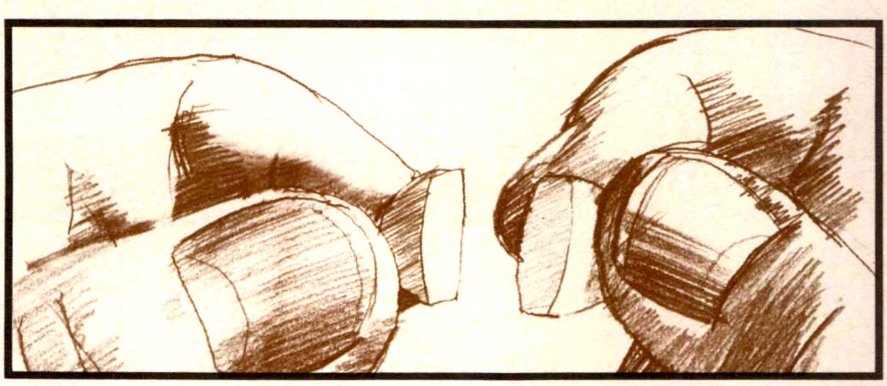
titration more practical:

Flexibility promotes optimal response and economy

The wide range of six different tablet strengths—from 10 mg to 150 mg—permits you to prescribe the strength best suited for the individual patient. Because you won't need a new Rx, you can save your patient the cost of an extra prescription fee. And, by prescribing the daily dose at bedtime, you can prescribe the higher strength at the outset and help the patient economize on the prescription price per milligram.

Scored tablets obviate need for new Rx

Half-tablet dosage adjustments with Endep (amitriptyline HCl) are possible because every tablet is scored. You can prescribe a single strength and still have the advantage of adjusting dosage without resorting to a new Rx.



Endep®

amitriptyline HCl/Roche

The half-strength advantage

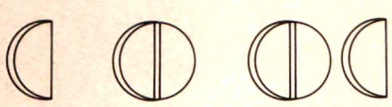
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Practicality is a fundamental fact about Endep®

amitriptyline HCl/Roche

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Compliance with practical regimen promotes optimal response.

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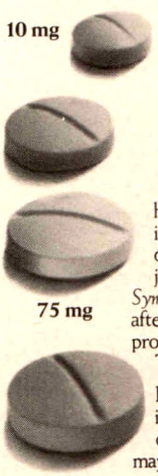
Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels.



Other: Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels, increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier.

Initial Adult Dosage: Outpatients—25 mg *t.i.d.*; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate—50 to 100 mg *h.s.*, gradually increasing *h.s.* dose up to 150 mg/day. Hospitalized Patients—Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day.

Adolescent and Elderly Patients: In general, 10 mg *t.i.d.* with 20 mg *h.s.* may be satisfactory for those who do not tolerate higher doses.

Maintenance Dosage: With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg *b.i.d.* to *q.i.d.*, or 10 mg *q.i.d.* Continue maintenance therapy 3 months or longer to avoid relapse.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg, 75-mg, 100-mg and 150-mg scored tablets—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg, 100 mg and 150 mg), available singly and in trays of 10.

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The Bermuda Hospitals' Board requires two (2) Consultant Psychiatrists for St. Brendan's Hospital to commence on 1st April, 1978. The Hospital is fully accredited by the Canadian Council for Hospital Accreditation.

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The hospital has 170 beds and is divided into eight distinct ward areas two of which care for the mentally retarded. It serves the total Island population of 60,000.

The theme of the psychiatric service is community oriented and an active rehabilitation programme is practiced. In addition, consulting services are provided to the Social Services Department, Sheltered Workshops, Child and Family Services, Rehabilitation Hostels, Drug and Alcohol Addiction Services, community based Geriatric Facilities, the Prison Services and the Island's modern General Hospital. Many of these programmes have recently been developed and there is therefore ample opportunity to develop and put into practice one's own interests and ideas.

Extra experience in psychogeriatrics, or neuropsychiatry would be valuable but not essential as, in selecting the candidate, emphasis will be on good all round psychiatric training and experience and the ability to share with colleagues the continued development of the service.

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Further information is available, on request.

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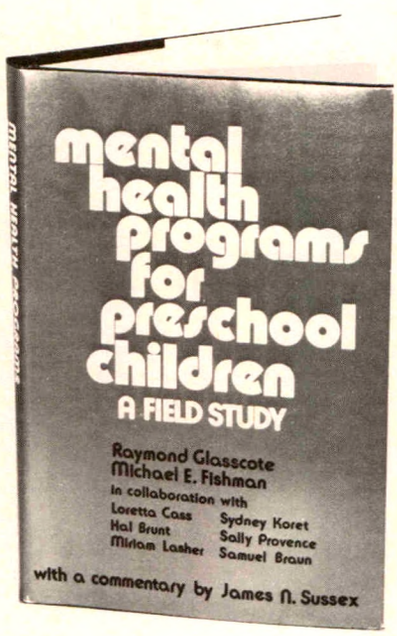
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BERMUDA

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This latest Joint Information Service field study provides a concise, comprehensive overview of the complex matter of the very young American child and his mental health, and then describes in intimate detail seven unique programs for pre-school children with problems—from North Hollywood's Dubnoff Center, emphasizing education as its primary therapeutic modality, to Baltimore's Martin Luther King, Jr. Parent-Child Center, serving two housing projects, served by Johns Hopkins, and largely governed by its own people. All different in treatment approach and philosophy, the programs described are uniform in their dedication to innovation in improving service to the emotionally disturbed very young child, and, equally, to the emotional well being of all young children.

This thoughtful and perceptive description of what has been created by some of the best informed, most experienced and thoughtful specialists in mental health services for very young children should be welcome to all involved in the mental health of children and a casebook for those dealing with child and family mental health.



An articulate exposition of the immense problems and encouraging accomplishments in a particularly difficult area of endeavor . . . . Beyond their concern with the manifestly ill child, the authors intriguingly explore what may be needed to enhance the mental good health of all young children . . . . A significant contribution.

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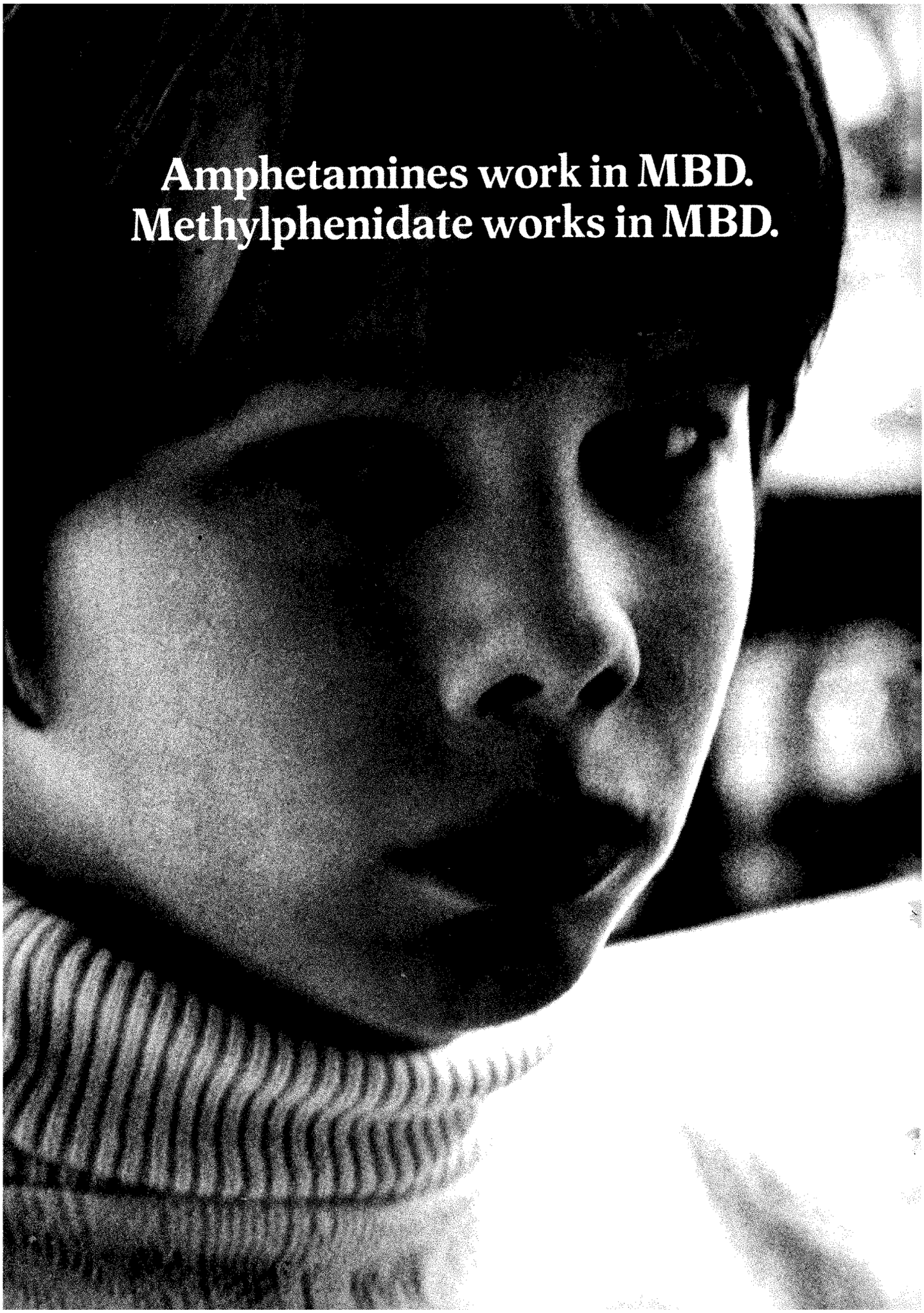
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(pemoline)

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Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally (once or twice a year) to determine if behavioral symptoms are sufficient to require continued therapy.

When not to use medication

Cylert should not be used for (and will not be effective in) simple cases of overactivity in school age children.

Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

The physician should rely on a complete history of the child and a thorough description of symptoms from both parents and teacher before postulating a diagnosis of MBD.

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Please see next page for Prescribing Information.

7083202 R1

Cylert and Cylert Chewable Tablets

(pemoline)

Prescribing Information

Description: Cylert (pemoline) is a central nervous system stimulant. Pemoline is structurally dissimilar to the amphetamines and methylphenidate.

It is an oxazolidine compound and is chemically identified as 2-amino-5-phenyl-2-oxazolin-4-one.

Pemoline is a white, tasteless, odorless powder, relatively insoluble (less than 1 mg./ml.) in water, chloroform, ether, acetone, and benzene; its solubility in 95% ethyl alcohol is 2.2 mg./ml.

Cylert (pemoline) is supplied as tablets for oral administration.

Clinical Pharmacology: Cylert (pemoline) has a pharmacological activity similar to that of other known central nervous system stimulants; however, it has minimal sympathomimetic effects. Although studies indicate that pemoline may act in animals through dopaminergic mechanisms, the exact mechanism and site of action of the drug in man is not known.

The serum half-life of pemoline is approximately 12 hours. Peak serum levels of the drug occur within 2 to 4 hours after ingestion of a single dose. Multiple dose studies in adults at several dose levels indicate that steady state is reached in approximately 2 to 3 days.

Metabolites of pemoline include pemoline conjugate, pemoline dione, mandelic acid, and unidentified polar compounds. Cylert is excreted primarily by the kidneys; approximately 75% of an oral dose is recovered in the urine within 24 hours. Approximately 43% of pemoline is excreted unchanged.

Cylert (pemoline) has a gradual onset of action in children with minimal brain dysfunction. Using the recommended schedule of dosage titration, significant clinical benefit may not be evident until the third or fourth week of drug administration.

Indications: *MINIMAL BRAIN DYSFUNCTION IN CHILDREN*—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: The cause of minimal brain dysfunction (MBD) is unknown. Diagnosis of MBD involves the use of medical, psychological, educational, and social tools, since no single diagnostic test is adequate.

MBD is characterized by chronic moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindications: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See ADVERSE REACTIONS.)

Warnings: Cylert is not recommended for children less than 6 years of age since its safety and efficacy in this age group have not been established.

Sufficient data on the safety and the efficacy of the long-term use of Cylert in children with minimal brain dysfunction are not yet available.

A temporary suppression of the predicted growth rate (weight and/or height gain) has been reported for children receiving long-term stimulant therapy. A definite causal relationship between stimulant drugs and this finding has not been established.

Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans. Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates. However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline. Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) for use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laboratory animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on the fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannabilization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conception.

Adverse Reactions: Insomnia is the most frequently reported side effect of Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear to be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

Overdosage: Signs and symptoms of acute Cylert overdosage may include agitation, restlessness, hallucinations, tachycardia. The treatment for an acute overdosage of pemoline is essentially the same as that for an overdosage of any CNS stimulant. Management is primarily symptomatic and may include induction of emesis or gastric lavage, sedation, and other appropriate supportive measures.

Results of preliminary studies in dogs indicate that extracorporeal hemodialysis may be useful in the management of Cylert overdosage; forced diuresis and peritoneal dialysis appear to be of little value.

Dosage and Administration: Cylert (pemoline) is administered as a single oral dose each morning.

The recommended starting dose is 37.5 mg./day. This daily dose should be gradually increased by 18.75 mg. at one week intervals until the desired clinical response is obtained. The effective daily dose for most patients will range from 56.25 to 75 mg. The maximum recommended daily dose of pemoline is 112.5 mg.

Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally (once or twice a year) to determine if behavioral symptoms are sufficient to require continued therapy.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

18.75 mg. tablets (yellow-colored) in bottles of 100 (NDC 0074-6025-13)

37.5 mg. tablets (orange-colored) in bottles of 100 (NDC 0074-6057-13)

75 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-6073-13)

Cylert Chewable is supplied as monogrammed, grooved tablets in one dosage strength:

37.5 mg. tablets (orange-colored) in bottles of 100 (NDC 0074-6088-13)

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Just Published . . .

The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

Paperback edition—$5.00 each, may be ordered from the AMERICAN PSYCHIATRIC ASSOCIATION, Publication Sales, 1700 18th St., N.W., Washington, D.C. 20009.

Hardback edition—$7.95 may be ordered from Basic Books, Inc., 10 East 53rd Street, New York, New York 10022.

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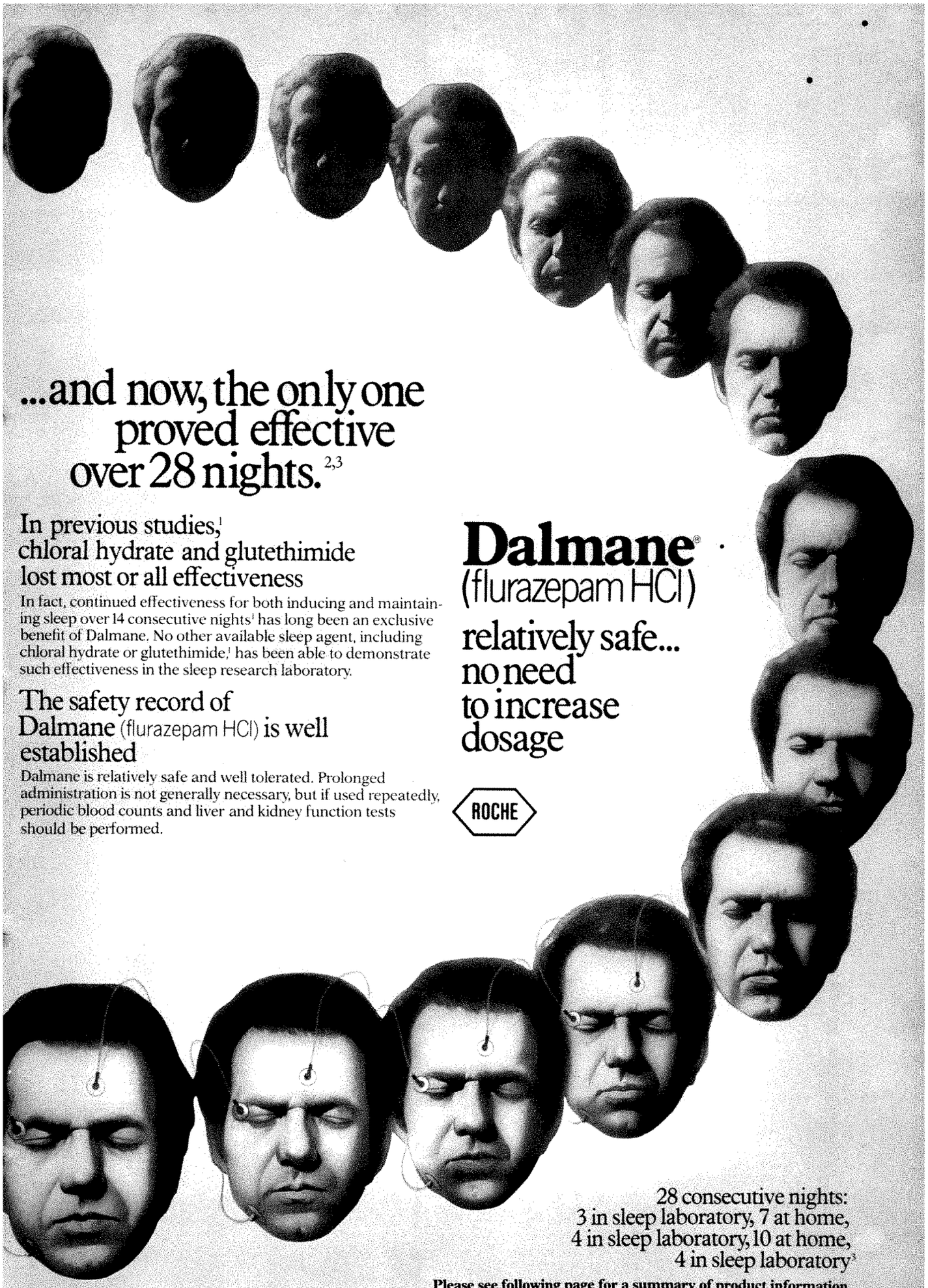
TOTAL SLEEP TIME (MEAN)[3]
(5 patients with insomnia)

*Significantly increased over baseline placebo ($p < .05$)

For those patients who need it, Dalmane (flurazepam HCl) alone provides continued effectiveness[2,3]

Since insomnia is often transient and intermittent, the prolonged administration of a hypnotic is generally not necessary or recommended. But for those patients whose insomnia is a chronic problem, the continued effectiveness of Dalmane is a decided benefit. Even at the end of a 28-night medication period, patients with chronic insomnia were falling asleep faster, spending less time awake during the night, and sleeping substantially longer on Dalmane 30 mg *h.s.* than on baseline placebo nights (average results). This effectiveness was proved[2] and confirmed[3] in two independent sleep research studies using a new 47-night protocol. When similarly evaluated, pentobarbital was ineffective at the end of two weeks.[2]

14 consecutive nights:
3 in sleep laboratory, 8 at home,
3 in sleep laboratory



...and now, the only one proved effective over 28 nights.[2,3]

In previous studies,[1] chloral hydrate and glutethimide lost most or all effectiveness

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Dalmane®
(flurazepam HCl)

relatively safe... no need to increase dosage

ROCHE

28 consecutive nights:
3 in sleep laboratory, 7 at home,
4 in sleep laboratory, 10 at home,
4 in sleep laboratory[3]

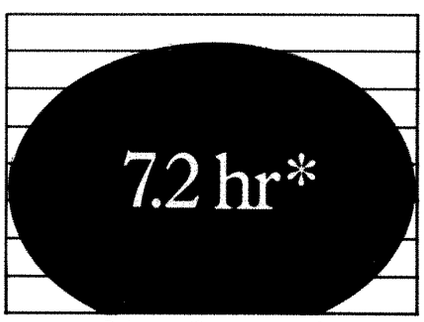
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1,5,7,14,28 nights... as needed, the effectiveness of Dalmane® one *h.s.* (flurazepam HCl) C IV meets the challenge of insomnia

One 30-mg capsule *h.s.*—usual adult dosage (15 mg may suffice in some patients).
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- **Patients fall asleep rapidly, sleep longer on a single *h.s.* dose**
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TOTAL SLEEP TIME (OVERALL MEAN)[3]
(5 patients with insomnia)



Dalmane (flurazepam HCl)
nights 1-3, 12-14, 26-28

*Significantly increased over baseline placebo ($p < .05$).

REFERENCES:

1. Kales A, *et al*: *Arch Gen Psychiatry* 23:226-232, Sep 1970
2. Kales A, *et al*: *Clin Pharmacol Ther* 18:356-363, Sep 1975
3. Dement WC, *et al*: Long-term effectiveness of flurazepam 30 mg *h.s.* on chronic insomniacs. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, Jun 30-Jul 4, 1975

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

***Usage in Pregnancy:* Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane (flurazepam HCl), a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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"I'm just a shadow of what I used to be."

That is what depressed individuals may feel is the substance of their being. There is no pleasure, no joy – nothing grows – and in the cold shadow of depression their activities are inhibited, while initiative may be eroded or destroyed. The tragedy is that they can see that others are able to live on the brighter side but they, themselves, cannot reach it on their own.

Your experience in treating depression, and Tofranil-**PM** can help light the way.

Tofranil-**PM**®
imipramine **pamoate**

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Unsurpassed effectiveness among tricyclics in relieving symptoms of depression.

Before prescribing Tofranil-PM, please review a summary of the prescribing information on the back of this page.

A83

Tofranil-PM®
imipramine pamoate

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As sleep disturbances, fatigue, and other depressive symptoms are relieved, mood and motivation may be markedly improved.

Patients are usually alert and capable of functioning at more normal levels of behavior.

Tofranil-PM encourages patient compliance because one capsule lasts from bedtime to bedtime.

Good results are usually seen at the starting dose of one 75-mg capsule h.s.

For many patients, dosage can be safely increased to 150 mg daily.

As with all tricyclics, sedation may occur. Please caution patients against driving or operating dangerous machinery.

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)
(B) 98-146-840-A(9/75) 667120

For complete details, including dosage and administration, please refer to the full prescribing information.

GEIGY Pharmaceuticals
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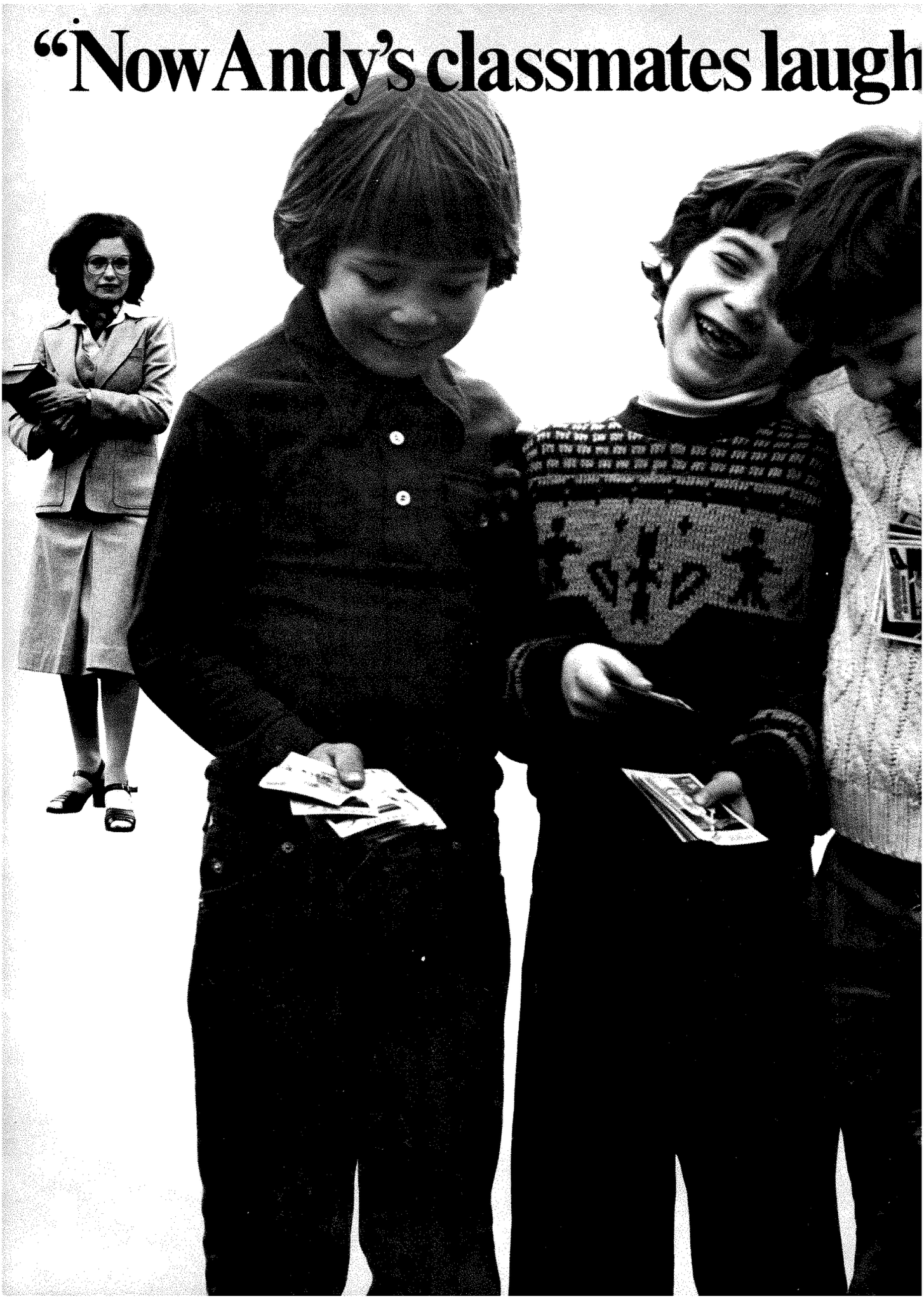
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"Now Andy's classmates laugh

with him, not at him"

Today, direct coaching in appropriate social interaction, role playing, and encounter techniques are being used to help hyperactive children overcome disruptive social behavior patterns that often cause them to become objects of ridicule to their peers.

Ritalin, when indicated, can also play a useful role in this respect, as improving social interaction in the MBD child often involves reducing hyperactivity. Indeed, as one investigator[1] has reported, with Ritalin therapy, the MBD child can be less aggressive, less impulsive, and better able to interact with his peers.

Other studies also report improved social behavior of MBD children on Ritalin.[2,3]

A drug of choice when treatment of MBD is indicated,[4,5] controlled studies and clinical experience have demonstrated the ability of Ritalin to effectively reduce hyperactivity,[1,2] distractibility,[1,3] and disorganized behavior[1,3,6-8] in the MBD child.

And these symptomatic improvements achieved with Ritalin often result in the child being more responsive to the nonpharmacological measures of the MBD management team.[8]

Although side effects – insomnia and appetite loss – are occasionally seen with Ritalin, they are less frequent or severe than with dextroamphetamine.[4,10]

Therapy with Ritalin should be undertaken only after a medical diagnosis of MBD has been made. Dosage should be periodically interrupted. Often these interruptions reveal some "stabilization" in the child's behavior even without medication. In some MBD children they permit a reduction in dosage and eventual discontinuance of drug therapy.

Ritalin...Only when medication is indicated

Ritalin® C II
(methylphenidate)
An effective member of the MBD management team



CIBA

Please turn page for brief prescribing information.



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(methylphenidate)
Only when medication is indicated

Ritalin® hydrochloride
(methylphenidate hydrochloride)
TABLETS

INDICATION

Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)

Special Diagnostic Considerations

Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.

Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS

Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS

Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy

Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS

Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS

Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION

Children with Minimal Brain Dysfunction (6 years and over).

Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.

If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.

Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED

Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-Pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500, and 1000.

Consult complete product literature before prescribing.

C76-16 Rev. 7/76

CIBA Pharmaceutical Company
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Summit, New Jersey 07901

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Tranxene® CL IV

CAPSULES
SINGLE DOSE TABLETS

CLORAZEPATE DIPOTASSIUM 4306CB **Brief Summary**

ABBOTT

ACTIONS—Pharmacologically, TRANXENE (clorazepate dipotassium) has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine.

INDICATIONS—TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE for long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS—TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS—TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS—In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS—The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION—

For symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg TRANXENE initially, followed by 30 to 60 mg in divided doses; 2nd 24 hours, 45 to 90 mg in divided doses; 3rd 24 hours, 22.5 to 45 mg in divided doses; 4th day, 15 to 30 mg in divided doses. Thereafter gradually reduce to 7.5 to 15 mg daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS—If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE (clorazepate dipotassium) is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE—Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed, since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY—Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption–Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED—TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

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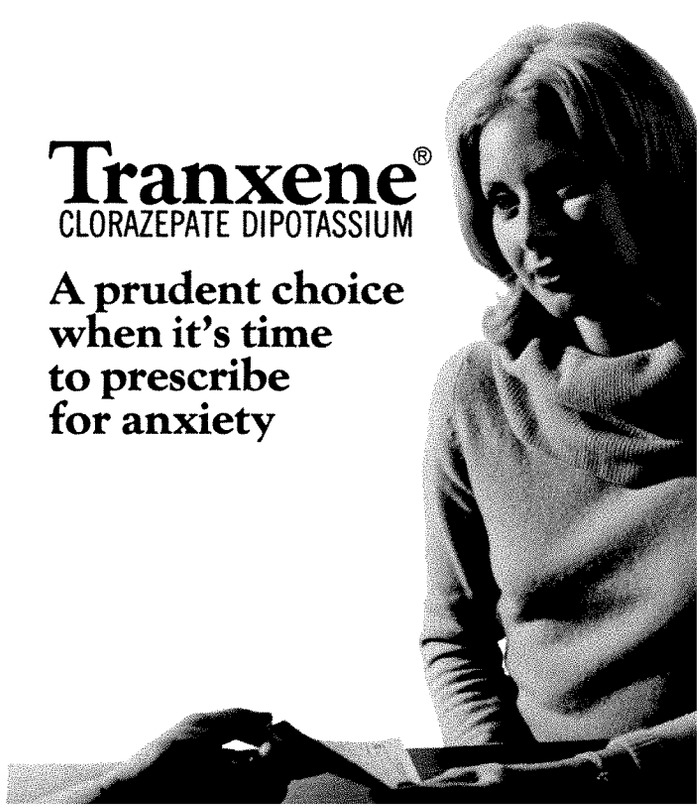
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In this issue

The Successful Professional Woman: On Being Married To One
By Theodore Nadelson and Leon Eisenberg

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3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
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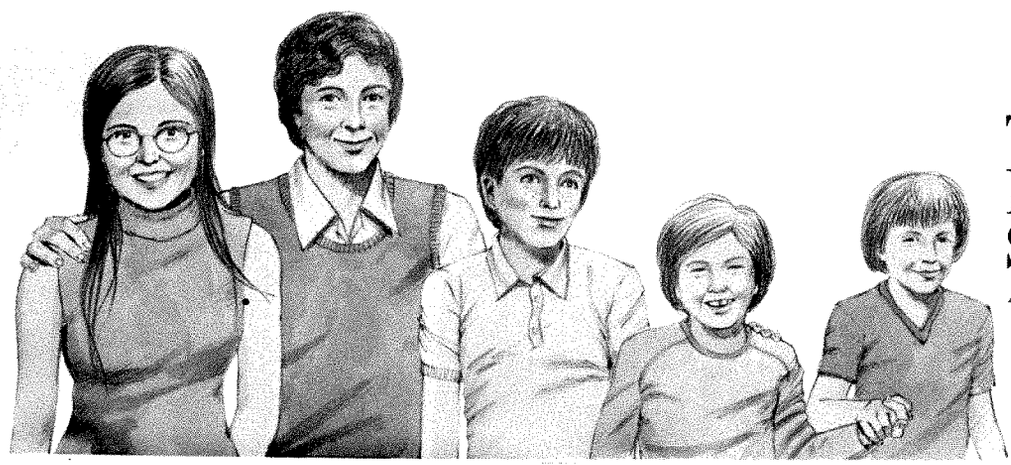
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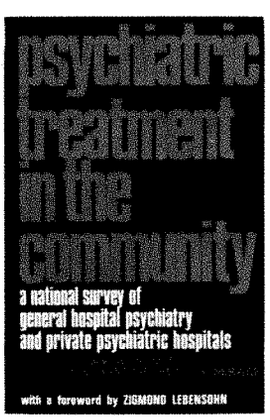
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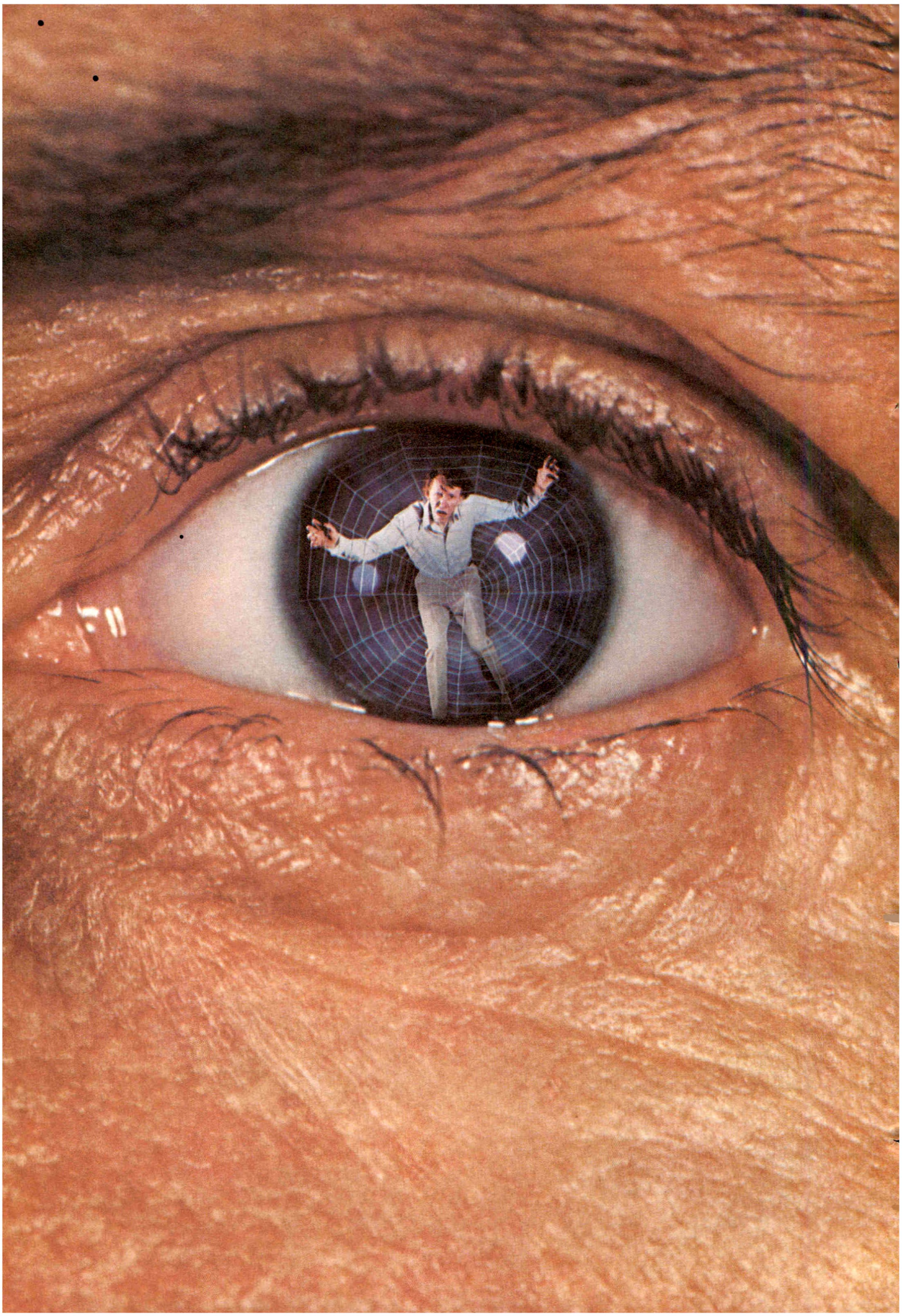
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Actions. Navane is a psychotropic agent of the thioxanthene series. Navane possesses certain chemical and pharmacological similarities to the piperazine phenothiazines and differences from the aliphatic group of phenothiazines. Navane's mode of action has not been clearly established.

Indications. Navane is effective in the management of manifestations of psychotic disorders.

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy* – Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children—The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration – As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution—For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate—In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg. of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

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Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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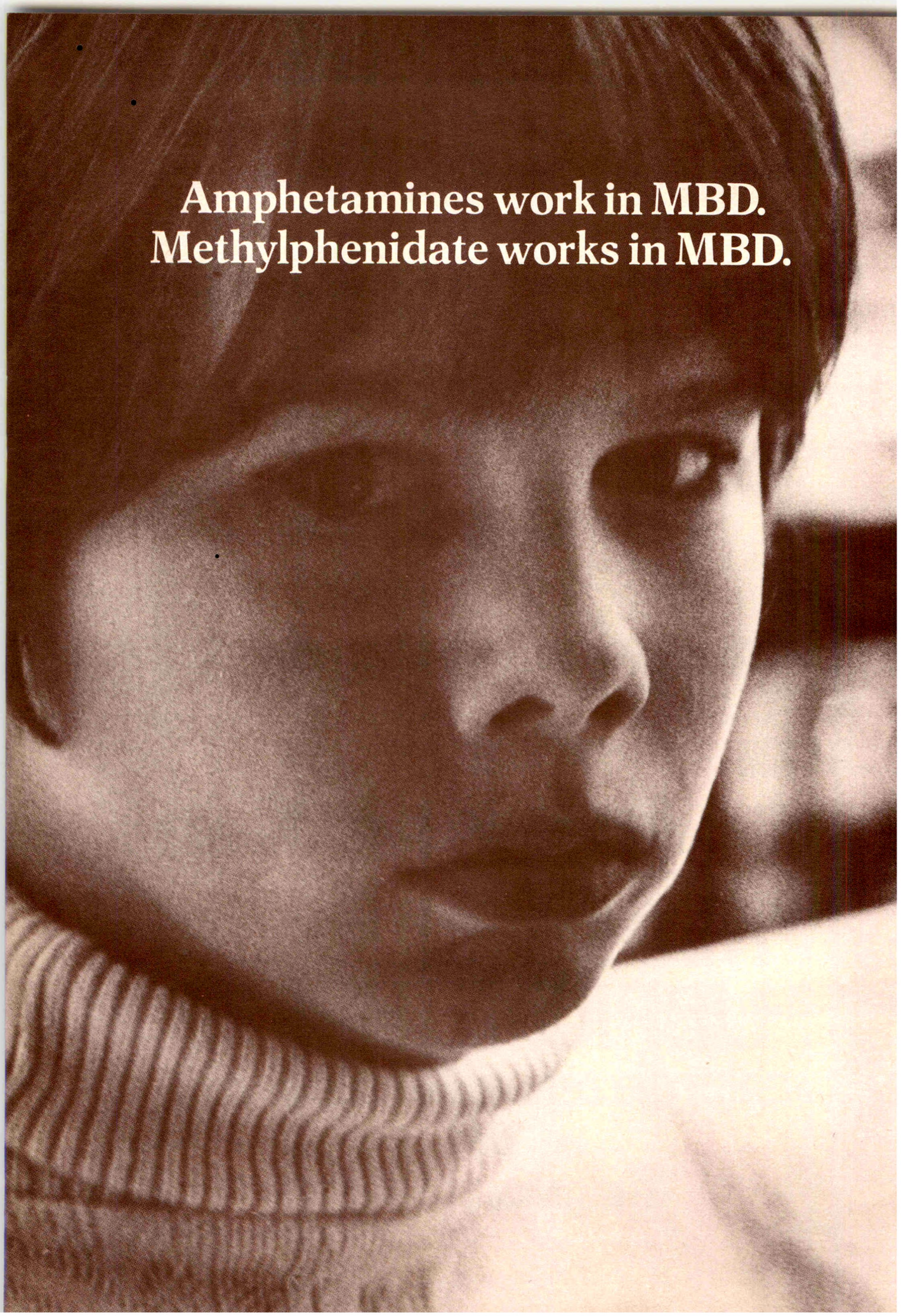
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Cylert (pemoline) is supplied as tablets for oral administration.

Clinical Pharmacology: Cylert (pemoline) has a pharmacological activity similar to that of other known central nervous system stimulants; however, it has minimal sympathomimetic effects. Although studies indicate that pemoline may act in animals through dopaminergic mechanisms, the exact mechanism and site of action of the drug in man is not known.

The serum half-life of pemoline is approximately 12 hours. Peak serum levels of the drug occur within 2 to 4 hours after ingestion of a single dose. Multiple dose studies in adults at several dose levels indicate that steady state is reached in approximately 2 to 3 days.

Metabolites of pemoline include pemoline conjugate, pemoline dione, mandelic acid, and unidentified polar compounds. Cylert is excreted primarily by the kidneys; approximately 75% of an oral dose is recovered in the urine within 24 hours. Approximately 43% of pemoline is excreted unchanged.

Cylert (pemoline) has a gradual onset of action in children with minimal brain dysfunction. Using the recommended schedule of dosage titration, significant clinical benefit may not be evident until the third or fourth week of drug administration.

Indications: *MINIMAL BRAIN DYSFUNCTION IN CHILDREN*—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: The cause of minimal brain dysfunction (MBD) is unknown. Diagnosis of MBD involves the use of medical, psychological, educational, and social tools, since no single diagnostic test is adequate.

MBD is characterized by chronic moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindications: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See ADVERSE REACTIONS.)

Warnings: Cylert is not recommended for children less than 6 years of age since its safety and efficacy in this age group have not been established.

Sufficient data on the safety and the efficacy of the long-term use of Cylert in children with minimal brain dysfunction are not yet available.

A temporary suppression of the predicted growth rate (weight and/or height gain) has been reported for children receiving long-term stimulant therapy. A definite causal relationship between stimulant drugs and this finding has not been established.

Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans. Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates. However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline. Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) for use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laboratory animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on the fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannabilization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conception.

Adverse Reactions: Insomnia is the most frequently reported side effect of Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear to be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

Overdosage: Signs and symptoms of acute Cylert overdosage may include agitation, restlessness, hallucinations, tachycardia. The treatment for an acute overdosage of pemoline is essentially the same as that for an overdosage of any CNS stimulant. Management is primarily symptomatic and may include induction of emesis or gastric lavage, sedation, and other appropriate supportive measures.

Results of preliminary studies in dogs indicate that extracorporeal hemodialysis may be useful in the management of Cylert overdosage; forced diuresis and peritoneal dialysis appear to be of little value.

Dosage and Administration: Cylert (pemoline) is administered as a single oral dose each morning.

The recommended starting dose is 37.5 mg./day. This daily dose should be gradually increased by 18.75 mg. at one week intervals until the desired clinical response is obtained. The effective daily dose for most patients will range from 56.25 to 75 mg. The maximum recommended daily dose of pemoline is 112.5 mg.

Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally (once or twice a year) to determine if behavioral symptoms are sufficient to require continued therapy.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

18.75 mg. tablets (yellow-colored) in bottles of 100 (NDC 0074-6025-13)

37.5 mg. tablets (orange-colored) in bottles of 100 (NDC 0074-6057-13)

75 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-6073-13)

Cylert Chewable is supplied as monogrammed, grooved tablets in one dosage strength:

37.5 mg. tablets (orange-colored) in bottles of 100 (NDC 0074-6088-13)

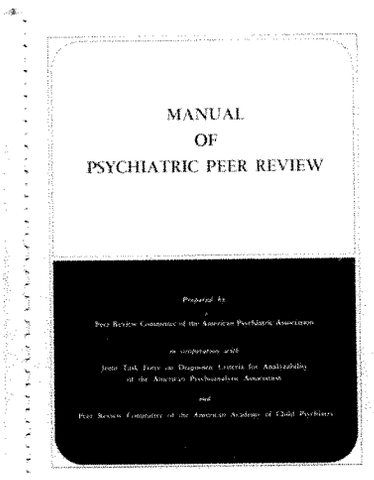
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"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

Robert W. Gibson, M.D.
President

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For those patients who need it, Dalmane (flurazepam HCl) alone provides continued effectiveness[2,3]

Since insomnia is often transient and intermittent, the prolonged administration of a hypnotic is generally not necessary or recommended. But for those patients whose insomnia is a chronic problem, the continued effectiveness of Dalmane is a decided benefit. Even at the end of a 28-night medication period, patients with chronic insomnia were falling asleep faster, spending less time awake during the night, and sleeping substantially longer on Dalmane 30 mg *h.s.* than on baseline placebo nights (average results). This effectiveness was proved[2] and confirmed[3] in two independent sleep research studies using a new 47-night protocol. When similarly evaluated, pentobarbital was ineffective at the end of two weeks.[2]

TOTAL SLEEP TIME (MEAN)[3]
(5 patients with insomnia)

*Significantly increased over baseline placebo ($p < .05$).

14 consecutive nights:
3 in sleep laboratory, 8 at home,
3 in sleep laboratory



...and now, the only one proved effective over 28 nights.[2,3]

In previous studies,[1] chloral hydrate and glutethimide lost most or all effectiveness

In fact, continued effectiveness for both inducing and maintaining sleep over 14 consecutive nights[1] has long been an exclusive benefit of Dalmane. No other available sleep agent, including chloral hydrate or glutethimide,[1] has been able to demonstrate such effectiveness in the sleep research laboratory.

The safety record of Dalmane (flurazepam HCl) is well established

Dalmane is relatively safe and well tolerated. Prolonged administration is not generally necessary, but if used repeatedly, periodic blood counts and liver and kidney function tests should be performed.

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(flurazepam HCl)

relatively safe... no need to increase dosage

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28 consecutive nights:
3 in sleep laboratory, 7 at home,
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4 in sleep laboratory[3]

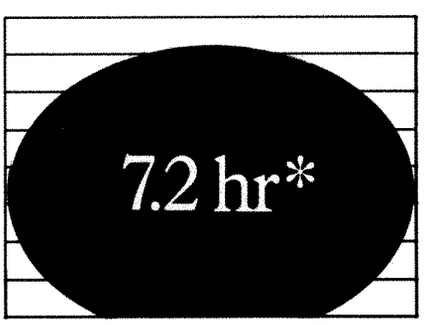
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1,5,7,14,28 nights... as needed, the effectiveness of Dalmane® one *h.s.* (flurazepam HCl) C IV meets the challenge of insomnia

One 30-mg capsule *h.s.*—usual adult dosage (15 mg may suffice in some patients).
One 15-mg capsule *h.s.*—initial dosage for elderly or debilitated patients.

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TOTAL SLEEP TIME (OVERALL MEAN)[3]
(5 patients with insomnia)



Dalmane (flurazepam HCl)
nights 1-3, 12-14, 26-28

*Significantly increased over baseline placebo ($p < .05$).

REFERENCES:

1. Kales A, *et al*: *Arch Gen Psychiatry* *23*:226-232, Sep 1970
2. Kales A, *et al*: *Clin Pharmacol Ther* *18*:356-363, Sep 1975
3. Dement WC, *et al*: Long-term effectiveness of flurazepam 30 mg *h.s.* on chronic insomniacs. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, Jun 30-Jul 4, 1975

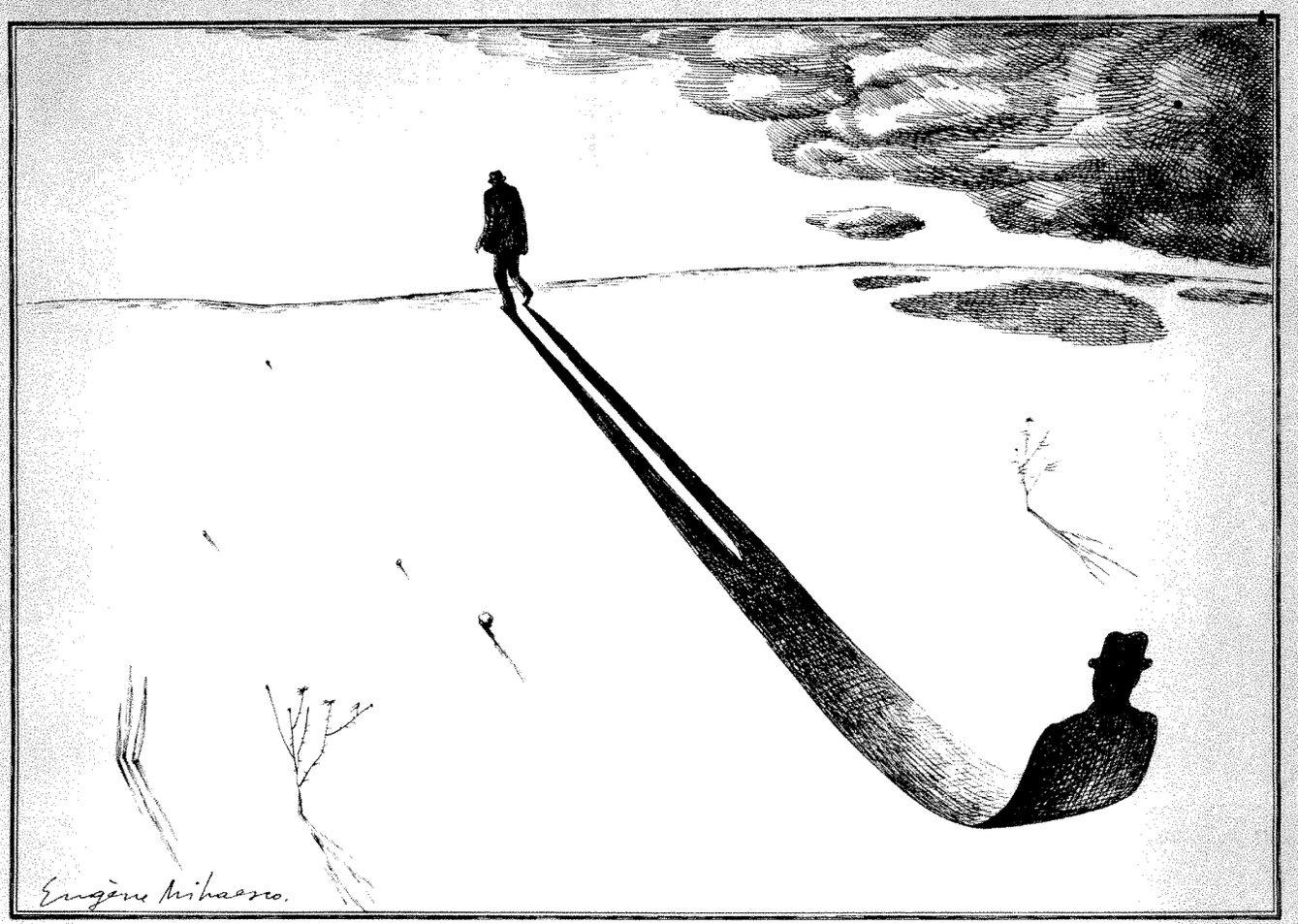
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Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

***Usage in Pregnancy:* Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane (flurazepam HCl), a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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"I'm just a shadow of what I used to be."

That is what depressed individuals may feel is the substance of their being. There is no pleasure, no joy – nothing grows – and in the cold shadow of depression their activities are inhibited, while initiative may be eroded or destroyed. The tragedy is that they can see that others are able to live on the brighter side but they, themselves, cannot reach it on their own.

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imipramine **pamoate**

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As anxiety, agitation, sleep disturbances, and other depressive symptoms are relieved, mood and motivation may be markedly improved.

Patients are usually alert and capable of functioning at more normal levels of behavior.

Tofranil-PM encourages patient compliance because one capsule lasts from bedtime to bedtime.

Good results are usually seen at the starting dose of one 75-mg capsule h.s.

For many patients, dosage can be safely increased to 150 mg daily.

As with all tricyclics, sedation may occur. Please caution patients against driving or operating dangerous machinery.

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

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brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;

—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;

—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;

—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;

—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)
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For complete details, including dosage and administration, please refer to the full prescribing information.

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"Now Andy's classmates laugh



with him, not at him"



Today, direct coaching in appropriate social interaction, role playing, and encounter techniques are being used to help hyperactive children overcome disruptive social behavior patterns that often cause them to become objects of ridicule to their peers.

Ritalin, when indicated, can also play a useful role in this respect, as improving social interaction in the MBD child often involves reducing hyperactivity. Indeed, as one investigator[1] has reported, with Ritalin therapy, the MBD child can be less aggressive, less impulsive, and better able to interact with his peers. Other studies also report improved social behavior of MBD children on Ritalin.[2,3]

A drug of choice when treatment of MBD is indicated,[4,5] controlled studies and clinical experience have demonstrated the ability of Ritalin to effectively reduce hyperactivity,[1,2] distractibility,[1,3] and disorganized behavior[1,3,6-8] in the MBD child.

And these symptomatic improvements achieved with Ritalin often result in the child being more responsive to the nonpharmacological measures of the MBD management team.[8]

Although side effects – insomnia and appetite loss – are occasionally seen with Ritalin, they are less frequent or severe than with dextroamphetamine.[4,10]

Therapy with Ritalin should be undertaken only after a medical diagnosis of MBD has been made. Dosage should be periodically interrupted. Often these interruptions reveal some "stabilization" in the child's behavior even without medication. In some MBD children they permit a reduction in dosage and eventual discontinuance of drug therapy.

Ritalin...Only when medication is indicated

Ritalin® (II)
(methylphenidate)
An effective member of the MBD management team



C I B A

Please turn page for brief prescribing information.



Ritalin®
(methylphenidate)
Only when medication is indicated

Ritalin® hydrochloride
(methylphenidate hydrochloride)
TABLETS

INDICATION

Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)

Special Diagnostic Considerations

Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.

Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS

Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS

Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy

Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS

Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS

Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION

Children with Minimal Brain Dysfunction (6 years and over).

Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.

If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.

Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED

Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-Pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500, and 1000.

Consult complete product literature before prescribing.

C76-16 Rev. 7/76

CIBA Pharmaceutical Company
Division of CIBA-GEIGY Corporation
Summit, New Jersey 07901

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CIBA

Dual Problem: marked agitation with depression

...a common problem in psychiatric practice.



MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information, please see last page of this advertisement.

Dual-action TRIAVIL®

containing perphenazine and amitriptyline HCl

Marked agitation accompanying depression is a common problem in psychiatric practice, a problem often requiring dual-action therapy to effectively cope with both components of the disorder. TRIAVIL 4-25 helps treat both. A formulation particularly suited to psychiatric practice when higher doses are required, TRIAVIL 4-25 affords the psychiatrist the opportunity to treat marked agitation—each tablet containing 4 mg of the tranquilizer, perphenazine—while helping to relieve the coexisting depression with 25 mg of the tricyclic antidepressant, amitriptyline HCl.



helps treat both the marked agitation and the depression

An antidepressant alone may be adequate when the accompanying anxiety is mild, but dual-action TRIAVIL should prove more appropriate when the anxiety or agitation (accompanying depression) is moderate or severe.

The four formulations of TRIAVIL permit the psychiatrist to treat a relatively broad spectrum of depressed patients who manifest symptoms ranging from moderate anxiety to severe agitation, and allow for individualized dosage adjustment.

The advantages of dual-action TRIAVIL in psychiatric practice.

With TRIAVIL, as anxiety or agitation with depression is relieved, the patient may become more accessible and cooperative. Many symptoms such as insomnia, fatigue, anorexia, and functional G.I. complaints are frequently alleviated; symptomatic relief may enable the patient to function more effectively in his daily activities, and the psychotherapeutic process itself may be aided.

TRIAVIL simplifies therapy; encourages compliance.

A single tablet encourages patients to take medication properly and reduces the risk of dosage confusion and error. Cost of therapy to the patient is usually less than when the components are prescribed individually. To date, clinical evaluations of TRIAVIL have revealed no undesirable reactions peculiar to the combination.

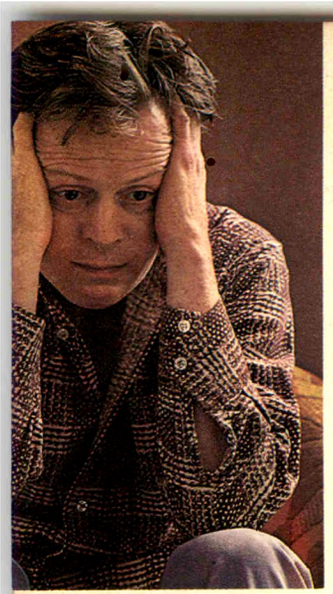
Treatment with TRIAVIL– a balanced view.

TRIAVIL is contraindicated in CNS depression from drugs, in the presence of evidence of bone marrow depression, and in patients hypersensitive to phenothiazines or amitriptyline. It should not be used during the acute recovery phase following myocardial infarction or in patients who have received an MAOI within two weeks. Patients with cardiovascular disorders should be watched closely. It is not recommended in children or during pregnancy. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. Antiemetic effect may obscure toxicity due to other drugs or mask other disorders. Since suicide is a possibility in any depressive illness, patients should not have access to large quantities of the drug. Hospitalize as soon as possible any patient suspected of having taken an overdose.



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For a brief summary of prescribing information, please see following page.



DUAL-ACTION Triavil®

containing perphenazine and amitriptyline HCl

for moderate to severe anxiety or agitation and depression

Available:
TRIAVIL® 2-25: Each tablet contains 2 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 2-10: Each tablet contains 2 mg perphenazine and 10 mg amitriptyline HCl.
TRIAVIL® 4-25: Each tablet contains 4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains 4 mg perphenazine and 10 mg amitriptyline HCl.

INITIAL THERAPY FOR MANY PATIENTS
TRIAVIL® 2-25 (or TRIAVIL® 4-25) t.i.d. or q.i.d.

FOR FLEXIBILITY IN ADJUSTING MAINTENANCE THERAPY
TRIAVIL® 2-10 (or TRIAVIL® 4-10)

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric crisi hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinsonism have been reported and can usually be controlled by the concomitant use o effective antiparkinsonian drugs and/or by reduction in dosage, but som times persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term therapy o may occur after drug therapy with phenothiazines and related agents ha been discontinued. The risk appears to be greater in elderly patients on hig dose therapy, especially females. Symptoms are persistent and in som patients appear to be irreversible. The syndrome is characterized by rhyt mical involuntary movements of the tongue, face, mouth, or jaw. Involu tary movements of the extremities sometimes occur. There is no know treatment for tardive dyskinesia; antiparkinsonism agents usually do not all viate the symptoms. It is advised that all antipsychotic agents be discontin ued if the above symptoms appear. If treatment is reinstituted, or dosage o the particular drug increased, or another drug substituted, the syndrome ma be masked. Fine vermicular movements of the tongue may be an early sig of the syndrome. The full-blown syndrome may not develop if medication stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, erythem urticaria, eczema, up to exfoliative dermatitis); other allergic reaction (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions) peripheral edema; reversed epinephrine effect; hyperglycemia; endocrin disturbances (lactation, galactorrhea, gynecomastia, disturbances of me strual cycle); altered cerebrospinal fluid proteins; paradoxical excitemen hypertension, hypotension, tachycardia, and ECG abnormalities (quinidin like effect); reactivation of psychotic processes; catatonic-like states; aut nomic reactions, such as dry mouth or salivation, headache, anorexi nausea, vomiting, constipation, obstipation, urinary frequency or inco tinence, blurred vision, nasal congestion, and a change in pulse rate; othe adverse reactions reported with various phenothiazine compounds, but n with perphenazine, include grand mal convulsions, cerebral edema, pol phagia, pigmentary retinopathy, photophobia, skin pigmentation, and failur of ejaculation.

The phenothiazine compounds have produced blood dyscrasias (panc topenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosinoph lia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur after lon term administration of some phenothiazines. Although it has not bee reported in patients receiving TRIAVIL, the possibility that it might occu should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia hav also been reported.

Amitriptyline: Note: Listing includes a few reactions not reported for thi drug, but which have occurred with other pharmacologically similar tricycli antidepressant drugs and must be considered when amitriptyline is admini tered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpitation myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neuromu cular:* Confusional states; disturbed concentration; disorientation; delusions hallucinations; excitement; anxiety; restlessness; insomnia; nightmares numbness, tingling, and paresthesias of the extremities; peripheral neur pathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (ant diuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred visio disturbance of accommodation; increased intraocular pressure; constipatio paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic:* Ski rash; urticaria; photosensitization; edema of face and tongue. *Hematologic* Bone marrow depression including agranulocytosis; leukopenia; eosinoph lia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastric di tress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; paroti swelling; black tongue. Rarely hepatitis (including altered liver function an jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male breast enlargement and galactorrhea in the female; increased or decrease libido; elevated or lowered blood sugar levels. *Other:* Dizziness, weakness fatigue; headache; weight gain or loss; increased perspiration; urinar frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* Abrup cessation after prolonged administration may produce nausea, headache, an malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an overdosag should be admitted to a hospital as soon as possible. Treatment is sympt matic and supportive. However, the intravenous administration of 1–3 m of physostigmine salicylate is reported to reverse the symptoms of tricycli antidepressant poisoning. Because physostigmine is rapidly metabolized, th dosage of physostigmine should be repeated as required particularly if lif threatening signs such as arrhythmias, convulsions, and deep coma recur c persist after the initial dosage of physostigmine. On this basis, in sever overdosage with perphenazine-amitriptyline combinations, symptomati treatment of central anticholinergic effects with physostigmine salicylat should be considered.

J 6TR06 R 3 (DC 6613212)

For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486.

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NOW FROM SANDOZ

For the treatment of depression

PAMELOR®

(nortriptyline HCl) NF

25mg. Capsules



Provides 2 important benefits for both patients and clinicians.

- PAMELOR® begins to improve sleep patterns within a week.
- PAMELOR® allows patients to remain calm yet alert.

For Brief Summary please see following page.

NOW FROM SANDOZ

PAMELOR®
(nortriptyline HCl) NF
25 mg. Capsules

The antidepressant with 2 important clinical benefits

PAMELOR® begins to improve sleep patterns within a week

- PAMELOR® helps relieve the sleep problems that accompany depression: difficulty in falling asleep, restless sleep, and early-morning awakening. An improved pattern of sleep begins to appear in some patients within the first week of therapy.
- Overall improvement is often observed by the end of the second week. Maximum improvement with Pamelor®, as with other antidepressants, may require a longer period of therapy, particularly in severe depressive illnesses.

PAMELOR® allows patients to remain calm yet alert

- Helps improve depressed mood with a low level of stimulatory effect.
- PAMELOR® also has a mild calming action to help relieve the anxiety and tension that are often symptoms of depression. Because PAMELOR® rarely causes excessive drowsiness, patients are more alert and better able to function. Nevertheless, they should be cautioned about driving or operating machinery.

Indications: For relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Hypersensitivity. Should not be given concomitantly with MAO inhibitors or within 2 weeks of the use of this drug since hyperpyretic crises, severe convulsions, and fatalities have occurred when similar tricyclic antidepressants were used in such combinations. Cross-sensitivity with other dibenzazepines is a possibility. Contraindicated during acute recovery period after myocardial infarction.

Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: The pharmacologic similarities among the tricyclic antidepressant drugs require that each of the following reactions be considered when nortriptyline is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though these are not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

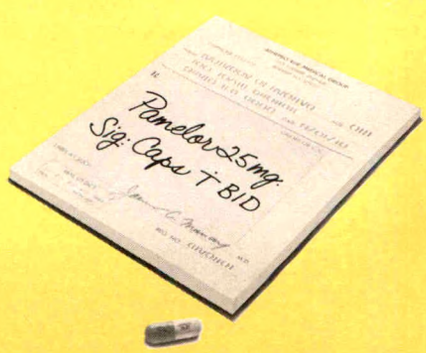
Dosage and Administration: *Usual adult dose*—25 mg. three or four times daily; dosage should begin at a low level and increase as required. *Elderly and Adolescent:* 30 to 50 mg. per day, in divided doses. Doses above 100 mg. per day and use in children are not recommended. If a patient develops minor side effects, the dosage should be reduced. The drug should be discontinued promptly if adverse effects of a serious nature or allergic manifestations occur.

How Supplied: Capsules 10 mg. and 25 mg.; solution 10 mg./5 cc.

For more detailed information see full prescribing information.

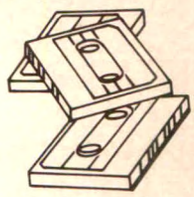
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CASSETTE LECTURES*
OF THE 130th ANNUAL MEETING
AMERICAN PSYCHIATRIC ASSOCIATION



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- USING THE RESEARCH DIAGNOSTIC CRITERIA TO STUDY AFFECTIVE DISORDERS
- NEUROTIC DEPRESSION: A SYSTEMATIC ANALYSIS OF ITS MULTIPLE MEANINGS
- FAMILIAL FACTORS IN AFFECTIVE DISORDERS
- PERSONALITY ATTRIBUTES AND AFFECTIVE DISORDERS
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- FAMILY DYNAMICS AND MANIC-DEPRESSIVE ILLNESS
- PHENOMENOLOGY OF DEPRESSION IN ADOLESCENCE
- SEROTONIN AND NOREPINEPHRINE SUBGROUPS IN DEPRESSION

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- FACTORS RELATED TO TARDIVE DYSKINESIA
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her "voices" came back.
Tomorrow she's
going back to work.*
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Haldol®
(haloperidol)
tablets/concentrate/injection

For prompt control with minimal risk of hypotension or oversedation

Highly effective in a wide range of psychotic symptoms[1-7]
such as hallucinations, delusions, suspiciousness, hostility, mania, psychomotor agitation, etc., in both acute and chronic disorders.

Helps keep patients functioning and avoid hospitalization.[1-3]
HALDOL haloperidol usually leaves most patients alert and responsive, able to function at work and at home without impairment, and more amenable to other therapeutic measures.

Permits aggressive titration to effective dosage levels[1,4]
up to 100 mg/day orally (with minimal risk of usual troublesome reactions) to achieve optimal response when patient is inadequately controlled at lower dosage...to help you rapidly control and stabilize new patients, promptly regain control during periods of exacerbation.

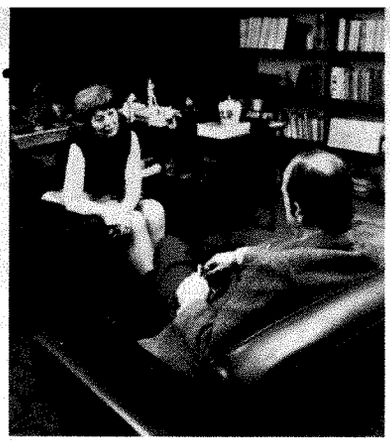
Common side effects easily controlled.[1,2,4]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Reduces risk of certain troublesome reactions.[1,2,4-6]
Transient hypotension occurs rarely and severe orthostatic hypotension has not been reported. Marked sedation is rare, although some instances of drowsiness have been reported. In addition, with chronic use, it is unlikely to cause hepatic damage, serious hematologic reactions, photosensitivity reactions and skin rashes...and has minimal effect on renal function.

Please turn page for information relating to Indications, Contraindications, Warnings, Precautions and Adverse Reactions.

Important: Full directions for use should be read before HALDOL haloperidol is administered or prescribed.



Haldol®
(haloperidol)
tablets/concentrate/injection

For prompt control of psychotic symptoms with minimal risk of hypotension or oversedation

A Dosage Form for Every Need:

5 tablet strengths for convenience in individualizing dosage: ½ mg., 1 mg., 2 mg., 5 mg. and 10 mg.

A tasteless, odorless, colorless liquid concentrate for better patient acceptability: 2 mg. per ml.

A rapid-acting injection for psychiatric emergencies: 5 mg. per ml., with 1.8 mg. methylparaben and 0.2 mg. propylparaben per ml., and lactic acid for pH adjustment to 3.4±0.2.

Contraindications: Severe depression, coma, CNS depression due to centrally-acting depressants, Parkinson's disease, hypersensitivity to the drug.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Usage in Children: Safety and effectiveness not established; not recommended in pediatric age group.

Combined Use with Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases. *Withdrawal Emergent Neurological Signs:* Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn. *Persistent Tardive Dyskinesia:* Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent. *Other CNS Effects:* Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms. ***Cardiovascular Effects:*** Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions:*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed. HALDOL tablets are manufactured by McNeil Laboratories Co., Dorado, Puerto Rico 00646. 5/77

References:

1. Man, P.L.: Dis. Nerv. Syst. *34*:113 (Feb.) 1973. **2.** Ayd, F.J., Jr.: Med. Sci. *18*:55 (Oct.) 1967. **3.** Rapp, M.S.: Can. Psychiatr. Assoc. J. *15*:73 (Feb.) 1970. **4.** Howard, J.S.: Dis. Nerv. Syst. *35*:458 (Oct.) 1974. **5.** Gerle, B.: Clin. Trials J. *3*:380 (Feb.) 1966. **6.** Abuzzahab, F.S., Sr.: Psychosomatics *11*:188 (May-June) 1970. **7.** Darling, H.F.: Dis. Nerv. Syst. *34*:364 (Oct.-Nov.) 1973.

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Coverage and Utilization of Care for Mental Conditions Under Health Insurance —Various Studies, 1973–74

By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

This book reports six studies that add to the growing body of data demonstrating the feasibility of covering mental illness under health insurance. Although in some ways it may be considered a supplement to APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (Reed, Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

- Utilization of mental benefits under the Blue Cross and Blue Shield plan for federal employees, documenting that a "plateau" in the use of these benefits was reached in 1973–74.
- Comparison of benefits for mental and other illnesses under selected employee health benefit plans.
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- Utilization of care for mental conditions under the Canadian health insurance program, which gives the same coverage for mental as for other conditions.
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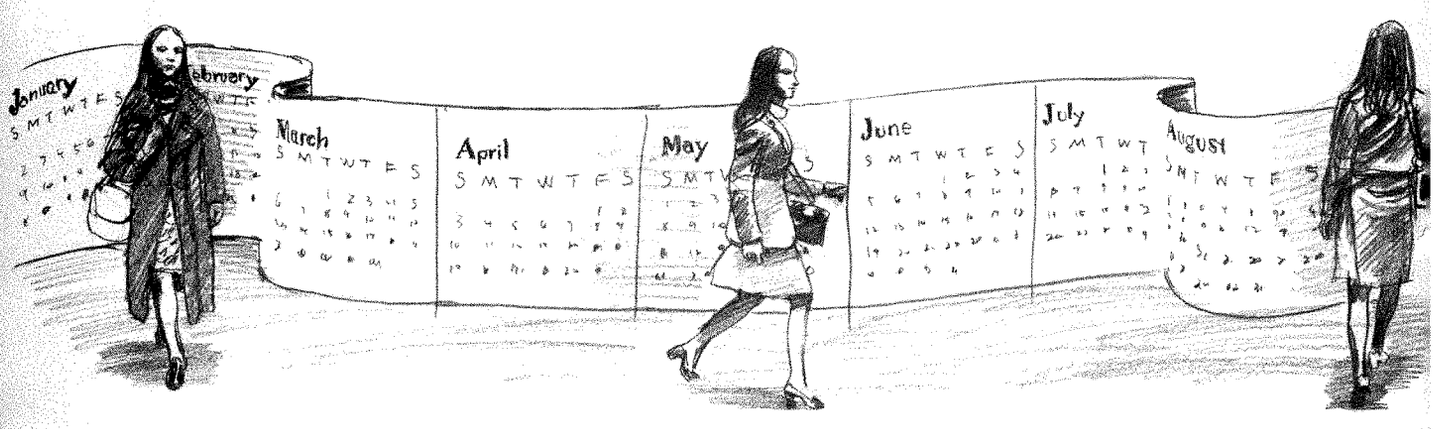
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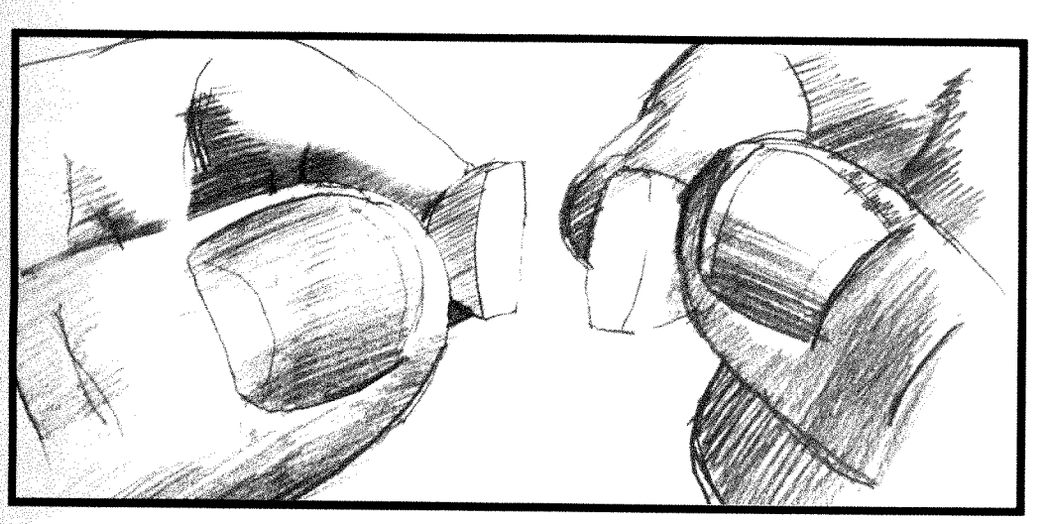
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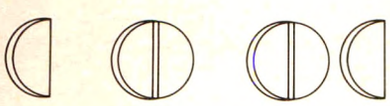
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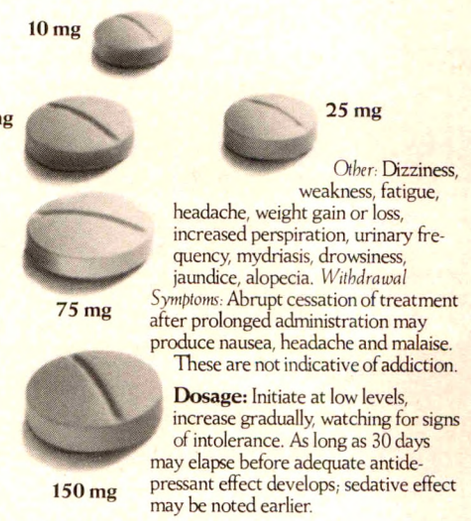
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Single daily *h.s.* dosage promotes compliance and economy.

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Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels, increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier.

Initial Adult Dosage: Outpatients—25 mg *t.i.d.*; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate—50 to 100 mg *h.s.*, gradually increasing *h.s.* dose up to 150 mg/day. Hospitalized Patients—Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day.

Adolescent and Elderly Patients: In general, 10 mg *t.i.d.* with 20 mg *h.s.* may be satisfactory for those who do not tolerate higher doses.

Maintenance Dosage: With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg *b.i.d.* to *q.i.d.*, or 10 mg *q.i.d.* Continue maintenance therapy 3 months or longer to avoid relapse.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg, 75-mg, 100-mg and 150-mg scored tablets—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg, 100 mg and 150 mg), available singly and in trays of 10.

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The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

Paperback edition—$5.00 each, may be ordered from the AMERICAN PSYCHIATRIC ASSOCIATION, Publication Sales, 1700 18th St., N.W., Washington, D.C. 20009.

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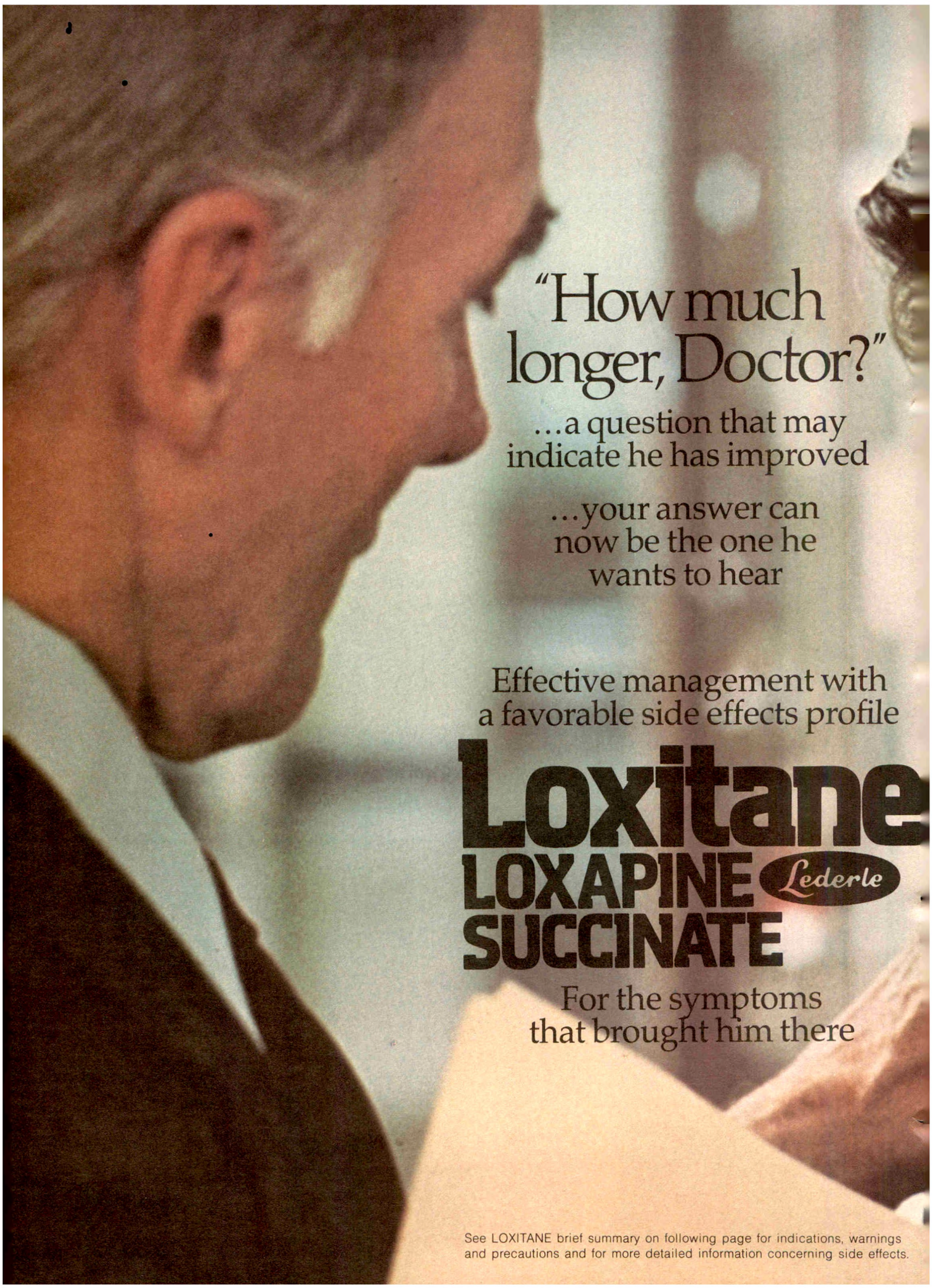
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...a question that may indicate he has improved
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Effective management with a favorable side effects profile
Loxitane
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For the symptoms that brought him there
See LOXITANE brief summary on following page for indications, warnings and precautions and for more detailed information concerning side effects.



Name	Sex	Age
Bert H.*	Male	27
Marital Status (S) M W D	**Occupation** Supermarket clerk	

CASE SUMMARY SHEET

History:
Left school at 16. Good in math; poor reader. Youngest child (only son); two married sisters well adjusted. Father died six years ago; mother, two years ago. Patient attractive, precise in manner. Few friends, no girlfriend. Lives alone

Onset of current illness:
When customer questioned addition of grocery slip, patient became abusive, said he "didn't steal." Threw slip at customer; hit store manager; tried to break cash register. Police brought him to hospital

Diagnosis:
Acute schizophrenia

Treatment:
LOXITANE® C Loxapine Hydrochloride Oral Concentrate 50 mg stat then 20 mg q.i.d. increased over 3 weeks to 35 mg q.i.d. Psychotherapy twice weekly

Therapeutic course:
First week: Calmer, sometimes curses, not physically violent. Believes cash register is part of a "conspiracy" to discredit him. Facial tremors appeared which responded to antiparkinsonism medication

Second week: Delusions persist but less often expressed. Not verbally abusive. Cooperative with ward regimen. LOXITANE C 30 mg q.i.d.

Third week: Boasts he "figures" better than the cash register. No further delusions. Switched to LOXITANE® Loxapine Succinate Capsules 35 mg q.i.d.

Fourth week: Says "girls are messy, always leaving makeup around." Encouraged to discuss relationships with people. Likes the OT worker because she is "neat and tidy"

Fifth week: In therapy sessions, speaks of "loneliness" and "fear of making mistakes." Appears close to premorbid behavior. LOXITANE decreased 25 mg q.i.d., antiparkinsonism medication discontinued

Sixth week: Asked to see eldest sister. Patient asking about day passes

Follow-up:
Tenth week: Sister took patient out for the day. Patient pleased but has difficulty expressing emotion. Asked about leaving the hospital; not upset when told he is not ready yet. LOXITANE 20 mg t.i.d. and 10 mg h.s. Group therapy once weekly

*Not an actual patient. The collection of clinical data upon which this simulation is built illustrates the action of LOXITANE® Loxapine Succinate as reported in clinical studies.

Loxitane®
LOXAPINE Lederle SUCCINATE

For the symptoms that brought him there

Recommended Daily Dosage

Initial Dosage	MILD 10 mg b.i.d.	MODERATE 10 mg t.i.d. or q.i.d.	SEVERE 25 mg b.i.d.
First 7 to 10 Days	Increase dosage until psychotic symptoms are controlled Dosage should not exceed 250 mg/day Usual dosage during titration: 50 to 150 mg/day		
Maintenance Dosage	Adjust to lowest effective level Usual maintenance dosage: 60 to 100 mg/day Many patients are controlled with dosages as low as 20 to 60 mg/day		



Brief Summary

LOXITANE® Loxapine Succinate Capsules
OR
LOXITANE® C Loxapine Hydrochloride Oral Concentrate

Description: A dibenzoxazepine compound, representing a new subclass of tricyclic antipsychotic agent, chemically distinct from the thioxanthenes, butyrophenones, and phenothiazines.

Indications: For the manifestations of schizophrenia. Loxapine has not been evaluated for the management of behavioral complications in mental retardation, and therefore cannot be recommended.

Contraindications: In comatose or severe, drug-induced depressed states (alcohol, barbiturates, narcotics, etc.); and in individuals with known hypersensitivity to the drug.

Warnings: In Pregnancy: Safe use during pregnancy or lactation has not been established; in pregnancy, nursing mothers, or women of child-bearing potential, weigh potential benefits against possible hazards to mother and child. No embryotoxicity or teratology was observed in studies in rats, rabbits or dogs, although with the exception of one rabbit study, the highest dosage was only two times the maximum recommended human dose and in some studies they were below this dose. Perinatal studies have shown renal papillary abnormalities in offspring of rats treated from mid-pregnancy with doses of 0.6 and 1.8 mg./kg., doses which approximate the usual human dose but which are considerably below the maximum recommended human dose. **In Children:** Studies have not been performed in children; therefore this drug is not for use below the age of 16.

May impair mental and/or physical abilities, especially during the first few days of therapy; warn ambulatory patients about activities requiring alertness (e.g., operating vehicles or machinery), and about concomitant use of alcohol and other CNS depressants.

Precautions: Use with extreme caution in patients with history of convulsive disorders; seizures have been reported in epileptics receiving this drug at antipsychotic dose levels, and may occur even with maintenance of routine anticonvulsant drug therapy. Has an antiemetic effect in animals; this effect in man may mask signs of overdosage of toxic drugs and obscure conditions such as intestinal obstruction and brain tumor. Use with caution in patients with cardiovascular disease. Increased pulse rates in the majority of patients receiving antipsychotic doses and transient hypotension have been reported. In hypotension requiring vasopressor therapy, preferred drugs may be norepinephrine or angiotensin. Usual doses of epinephrine may be ineffective because of inhibition of its vasopressor effect by loxapine. Possibility of ocular toxicity from loxapine cannot be excluded at this time, therefore observe carefully for pigmentary retinopathy and lenticular pigmentation (observed in some patients receiving certain other antipsychotic drugs for prolonged periods). Because of possible anticholinergic action, use with caution in patients with glaucoma or a tendency to urinary retention, particularly with concomitant administration of anticholinergic-type antiparkinson medication.

Adverse Reactions: CNS: Adverse effects other than extrapyramidal, infrequent. Drowsiness, usually mild, may occur at beginning of therapy or when dosage is increased; usually subsiding with continued therapy. Incidence of sedation is less than that of certain aliphatic phenothiazines and slightly more than piperazine phenothiazines. Dizziness, faintness, staggering gait, muscle twitching, weakness, and confusional states have been reported. Extrapyramidal reactions during use of this drug have been reported frequently, often during the first few days of treatment. In most patients, these involve Parkinson-like symptoms such as tremor, rigidity, excessive salivation, masked facies, akathisia, usually not severe and controlled by dosage reduction or use of antiparkinson drugs in usual dosage. *Less frequent, but more severe:* Dystonias, including spasms of muscles of neck and face, tongue protrusion, oculogyric movement; dyskinesia in the form of choreo-athetoid movements. These sometimes require reduction or temporary withdrawal of drug dosage in addition to appropriate counteractive drugs. *Persistent Tardive Dyskinesia:* May appear in some patients on long-term therapy, or after therapy has been discontinued; the risk greater in the elderly, especially females, on high dosage. These symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements, sometimes accompanied by involuntary movements of extremities). No known effective treatment; discontinue all antipsychotic agents if these symptoms appear. The necessity to reinstitute treatment or increase dosage, or switch to a different antipsychotic agent, may mask syndrome. If medication is stopped when fine vermicular movements of the tongue occur, the syndrome may not develop. *Cardiovascular:* Tachycardia, hypotension, hypertension, lightheadedness, syncope. A few cases of EKG changes similar to those seen with phenothiazines have been reported, not known to be related to loxapine use. *Skin:* Dermatitis, edema (puffiness of face), pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of uncertain etiology seen in a few patients during hot summer months. *Anticholinergic:* Dry mouth, nasal congestion, constipation, blurred vision—more likely to occur with concomitant use of antiparkinson agents. *Other:* Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of uncertain etiology.

Dosage and Administration: Administered orally, usually in 2 to 4 divided doses a day. Adjust dosage to patient's need relative to severity of symptoms and history of response to antipsychotic drugs. Recommended initial dosage is 10 mg. b.i.d.; in severely disturbed patients up to 50 mg. daily. Increase dosage during first 7 to 10 days until psychotic symptoms are controlled. Usual therapeutic and maintenance range is 60 mg.-100 mg. daily. More than 250 mg. daily is not recommended. Maintenance dosage should be at lowest level to control symptoms; many patients have been maintained on 20 mg. to 60 mg. daily.

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In the hospitalized patient...
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ANTIPSYCHOTIC EFFICACY... INFREQUENT EXTRAPYRAMIDAL SYMPTOMS



Antipsychotic medication should not interfere with the patient's ability to participate in your total therapeutic program. That is why Mellaril (thioridazine) is an excellent choice. It is highly effective, and although extrapyramidal symptoms are characteristic of this class of drug, with Mellaril (thioridazine) extrapyramidal stimulation—notably pseudoparkinsonism—is infrequent. Adding an antiparkinsonian agent—which can cause its own side effects—can usually be avoided.

Mellaril (thioridazine) is not habituating and usually does not cause euphoria or undue sedation. (But, warn patients about undertaking activities requiring complete mental alertness.) And Mellaril (thioridazine) is contraindicated in patients with severe hypotensive or hypertensive heart disease.

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TABLETS: 50 mg, 100 mg, 150 mg, and 200 mg thioridazine HCl, USP

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Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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The Successful Professional Woman: On Being Married To One

BY THEODORE NADELSON, M.D., AND LEON EISENBERG, M.D.

Femininity and professionalism traditionally have been regarded as incompatible. From their own experience and that of colleagues and patients, the authors argue that female personhood is enriched by professional commitment. In consequence, the woman with a career becomes a more stimulating spouse and parent. Problems of role differentiation and of time allocation stress the dual-career marriage, but coping with those stresses enhances personal growth and leads to a more rewarding marital relationship. The authors consider themselves the most fortunate of men.

FOOLS write papers that are autobiographical; wiser men confine their confessions to their analysts.[1] If further proof of folly be needed, note our temerity as men in daring to opine about the conditions of women's lives. Our hubris fairly invites criticism from colleagues, from readers, worse yet, from our wives. Clearly, we are not easily deterred. In our role as participant-observers in that most intense of relationships with successful women—being married to them—we insist on our right to something to say on the matter.

This essay is not based on generalizations from a sample of two. Our personal observations have been augmented by reflecting on the men we have known as friends, colleagues, and patients over our professional life-times. Preparing this paper offered us an opportunity for collaboratively taking stock, as friends, members of the same academic community, and fathers of children adolescent and older. We share one final perspective: we write in celebration of wives as well as marriages we consider hugely successful—which is not to say without problems to be resolved and tensions to be managed. And yet, for our investment in marriage, we have in return experienced interpersonal adventures of greater intensity than we had been able to imagine in the romantic dreams of our adolescence. Indeed, the most forceful realization from our common reminiscences is that fulfillment in marriage, much as in individual maturation, is enhanced by coping with and overcoming developmental crises.

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[1]S. Freud, personal communication.

WHAT IS A "SUCCESSFUL WOMAN"?

The term "successful woman" highlights the ambiguity of the adjective "successful" when it modifies the noun "woman." Literally, it refers to success in professional (nonmarital) life. Associatively, it implies success in being a woman (i.e., being feminine) as well. The dual connotation underscores the double standard that is prevalent in our sexist society. For, in contrast, the term "successful man" would at once be understood to describe one who is outstanding in his work role rather than in his sex or paternal role (that is, a successful lover or father).

Traditionally, the status of a wife is ascribed rather than achieved; that is, it is derivative from the social position of her husband. Thus, in most studies that include social class as an important variable, the children of married women are assigned a place in the social hierarchy corresponding to their father's income, occupation, and education. Census inquiries into the occupation of "head of household" automatically report that of the husband, unless there is no husband present. It has been taken as a social given that when the marriage is intact, the male is the head of the household.

This definition has not changed despite the fact that half the women in this country are in the paid labor force, including 46% of those with children (1, 2). What had been a "deviant" role for women a half century ago has become the modal one. The tipping point has been reached; it will be the unpaid housewife, not the working wife, who will feel social pressure to justi-

fy her role (3). Many women work because they want to and derive important personal satisfactions from an expanded social network, from the sense of accomplishment, and from the independence that comes from generating an income. However, many work out of necessity in jobs they find distasteful, a description that applies equally well to much of the male labor force (4). If we recite the obvious, it is to highlight the contrast between the ordinary work of most women and men and the work our physician wives and we are privileged to do. Our reflections, therefore, are limited to marriages in which the wife has a commitment to a professional career (5). That commitment is evident from the high motivation necessary to master career tasks and from the importance of occupational achievement to personal satisfaction (6–8).

Success, like status, has both a public and a private meaning. At a public level, success can be measured by the applause of others, by income level, by position in an organizational hierarchy and, for women, by being one among the very few who have attained a given level. Thus, a woman who is an associate professor or a dean is, in an important sense, more successful than a man occupying the same position because there are so few like her. As psychiatrists know all too well, the private conviction of success is no simple correlate of public recognition. To generalize from the limited sample of perhaps 40 professional women we have known, the successful woman is more likely to doubt her own worth than her male counterpart. It is not that we do not know men who remain convinced of their own incompetence despite external acclaim, but that the syndrome does appear to be more prevalent in successful women.

OCCUPATIONAL HAZARDS FOR PROFESSIONAL WOMEN

One does not have far to search for the sources of this discontent. In all disparaged groups, negative attitudes toward one's reference group all too readily become internalized. To be a successful woman is to be dealt with as an exception, which serves to reinforce the concept of inherent group inferiority. The stereotypes of the professional woman almost always incorporate the dogma of her "mannishness" and physical unattractiveness. To be regarded as feminine continues to be important to most women. Indeed, compliments to women on their "brainiess" may evoke a need to be reassured that they are not ugly. The husband of the successful professional has a role that is in no sense unique in meeting his wife's need for reassurance of her worth as a person (just as he needs this from her), but it is one that he may fail to recognize because it may seem absurd that a woman so competent and so attractive should need to be told the obvious.

Furthermore, he often finds his wife asking him advice about professional matters in which her judgment is superior to his own. Occasionally, this is nothing more than a ploy to make husbands feel needed, and, for that, we acknowledge our gratitude. Most often, it is not advice that is being sought but support for her own judgment. She is moving in realms where few women have moved before her, and she may doubt her legitimacy in situations in which men take their authority for granted. When she sets out a major concept at a committee meeting, discussion proceeds as if she had been silent; in the summary at the meeting's end, her idea may be included but accredited to a male colleague or, at best, left unattributed. Moreover, she is more likely to experience open challenge and covert sabotage because she is a woman in an executive position. Both men and women are more likely to defer to the man who occupies the same role. An additional reason for the "home consultation" is no more complex than the failure of her prior socialization to prepare her to function in social roles of command.

SECULAR CHANGES IN DUAL-CAREER PATTERNS

The experience of the men and women of our generation includes an important time differential. The usual pattern has been one in which the husband's career has advanced without interruption while his wife has deferred her own for child bearing and rearing. We do not suggest that this should be so but point out that it has been the case. Although we may have contributed more to child care than most fathers, it must be acknowledged that we have cooperated—perhaps even conspired—with our wives to give them the dominant role in home building, a role that corresponded with both our expectations and theirs about task division within marriage. Whatever the reason, the husbands in our cohort have had their careers moderately well established before their wives wholeheartedly pursued their own. It therefore required less personal sacrifice from a position of accomplishment to support a junior professional on her way up than to defer ambition, and not merely convenience, for the sake of another. We were also better able to provide guidance, when it was wanted, simply on the basis of our greater experience. The challenge to narcissism is far greater when coequal sacrifice is demanded at the beginning of marriage. In the past, women have borne the burden of delay disproportionately. They are becoming less willing to do so. They are right and men will have to change.

ROLE STRAINS FOR THE HUSBAND

For many professional men, it is an unusual and sometimes disquieting experience to attend, in the role of spouse rather than primary guest, the social gatherings that originate from work relationships. Depending upon his skills in small talk, the husband may stand around uncomfortably amidst strangers who politely

inquire, without any real interest, just who he is and what he does, quite a change from being "somebody" by definition. This experience provides at least a taste of what wives quite regularly experience, although it is a considerably muted one. No man in our sample can recall being asked about where he shops, his favorite dishes, the schools the children attend or being told how lovely he looks. And there is the assumption that we *do* work, an uncommon state of affairs for our wives outside academic circles. Indeed, we must acknowledge that the social role of husband as spouse is not really symmetrical with that of the wife. The following anecdote conveys its flavor:

> L's wife had been appointed to a senior position in a university, a role that made her the only woman on its governing council. The council had completed its year's work with a two-day weekend meeting followed by a dinner for "members and their wives" according to the invitation, with L as the only male "wife" (later changed to "spouse," with some embarrassment on the part of the secretariat). Soon after he arrived at the reception, L was called aside by the presiding officer and asked to be the unannounced "mystery after-dinner speaker." L, who is never at a loss for words (as his wife, when pressed, will testify), readily agreed. He was introduced amid general merriment, told a few more or less funny jokes (which by his report brought down the house) and then proceeded to admonish the group, in his self-appointed role as spouse-person, in protest against the invasion of "our" domestic weekends by university demands. All was in good spirit and taken seriously by no one, including the speaker; work patterns at the university remain unchanged, very much like the response to President Carter's entreaty to government executives on the importance of family life.

Clearly, "husband as spouse" is a special and jovial role, precisely because it remains so atypical.

Considerably less amusing is the attitude of some men who are smugly ensconced in traditional marriages. Having heard that one is married to a woman of some prominence, they respond with such comments as: "She must be quite a handful" or, more crudely, "How do you take not being in charge?" or (as a backhanded compliment) "You must be something to be able to manage her." Generally, the remarks are accompanied by a quizzical look if not a smirk. The assumptions clearly are that the husband is hardly a man if he is not in charge and quite a man if he is and that, however competent or independent the wife, what she "really" wants, like Katarina in *The Taming of the Shrew*, is a man to dominate, if not to spank, her till she exclaim, "Such a duty as the subject owes the prince, even such a woman oweth to her husband" (V, ii, 155–159).

THE DIVISION OF LABOR: WORKER, EXECUTIVE, OR PARTNER?

The commonplace chores of domestic life challenge the "liberated" husband's nominal commitment to an egalitarian marriage (9). Even those who accept task sharing as the norm usually view their role as accepting assignments, with more or less cheer, but expect executive responsibility to be carried by the wife. Being scrupulous about doing half the physical work, much of it mindless, is not the same as doing half the real work. Worrying about what needs doing and planning ahead for it exact important costs.

Family executive functions, in our clinical experience, demand a sizable reverberating neuronal circuit, thus preempting major pathways and consuming inordinate amounts of metabolic energy. How else can one explain the common complaint of the married woman physician: that she falls asleep evenings over journals and books when her husband still seems wide awake? The double load of family responsibility and professional responsibility results in wildcat strikes by overworked cortical neurons. As one woman professor said to her equally distinguished husband, "How often do you suddenly think, in the midst of lecturing to a class, 'My God, we have no toilet paper! I'd better stop on the way home and pick some up.' " That couple decided, not without some anguish, to contract on alternate months for one and then the other to take full responsibility for the household, including shopping for, and cooking, meals for guests. At follow-up, 3 years later, the plan is working remarkably well; the husband now boasts, to the considerable discomfort of his male colleagues, of his success in his new role.

After a national meeting a number of years ago (at the beginning of the women's movement) one member of our sample had a rude encounter with his own feelings. He had been almost completely ignored by a gathering of women who were all very interested in speaking with his wife. She stayed too long, he thought, and afterward, during a discussion of where to go for dinner, he felt she was too demanding. His response was anger, barely contained. Unable to control himself, he suddenly burst forth, castigating her character as controlling. Subsequently, he realized the source of his feeling in competitiveness and jealousy. Magnanimously (or so he thought), he gave her the gift of saying that he realized he had been acting out anger related to wounded pride and self-esteem. She said, "Yes, I know."

He had expected, he said, that she would soothe his feelings and (consonant with the model of behavior for men and women) give him support for the strength he displayed in admitting that he was jealous. She would say, in the expected scenario, that, after all, "you are strong and I was wrong." That had been demonstrated by his understanding of the angry feelings that he had renounced by identifying them. When she only acknowledged that he was obviously jealous, giving him no reward for his tour de force of insight, his anger redoubled. In later years he realized that moment to be a turning point (with no turning back) in the relationship with his wife. Her position was not that of being a wife, mother, and guardian of the hearth acting "as if" she were a professional like him. His anger had been

continuous because he felt a challenge that crystallized at the very moment because she only reiterated that he was, in fact, angry and she would not remove the reasons for it by reassuring him of his dominance and her submission. The change in him occurred slowly, reluctantly, and creakingly. Now, he regards his life as a rich one; in fact, he reports that it is hard for him to imagine what it would be like to have a resolutely domestic wife.

Many males in professional couples come only slowly to the understanding that their wives really "mean it," i.e., truly consider professional activities the major endeavor in their lives. Often the *female* partners arrive at this position with the help of other women; it is our impression that they arrive at it less often with help from their husbands.

Some men we have spoken to still indicate that there are times when they feel annoyance or anger engendered by a wife whose professional duties bend the household out of shape. Even those husbands who are beyond expecting a well-run, tidy household often are irritated when clothes are not available for their children for a special event. There is often an expectation that the wife should control all aspects of such events, as illustrated by the following incident reported by one of our subjects (after assurance of confidentiality):

> When I made a trip to the cleaners because I remembered that my son was to take his first look at a new school and had no clean clothes, I felt very virtuous and I wore my virtue as an indictment against my wife. It is only in retrospect that I realized that she had set up the appointment for him at the school and that really, logically, we shared the responsibility for the clean clothes equally. Happily I realize it now without her reminding me—that's too embarrassing. Maybe the encounter with such embarrassment in the past is helping to extinguish my annoyance.

However, such reactions extinguish slowly because of long-standing, reinforced attitudes regarding women's and men's roles.

What is essential to a workable arrangement is a "good wife" for both spouses—the luxury of a competent and caring housekeeper who not only does the household tasks but helps in the planning. Someone has to keep tabs on the necessities that need replacement and to deal with the small army of repair- and trades-people. That such a person is difficult to find hardly needs mention. Those who do find one feel blessed.[2]

THE THEFT OF TIME

Time, a scarce commodity in the dual-career family, becomes layered with multiple meanings in the psychological transactions between parents and children. To recognize that the development of a child is a function of the amount and kind of care the child receives is not to assert that the care must be provided solely by the parents. Even when surrogates provide the major part of the care, parents continue to possess far greater salience for children than substitute caretakers (10). Yet, despite what we, as professionals, know to be true, we remain prone to feelings of guilt. Many of us are too readily manipulated when our children use the tactic of measuring our performance by conventional standards.

> Larry had asked his mother to come to his school's Christmas play in which he was to be one of the Wise Men. None of her careful explanations of why she could not come during office hours (his father was going to be out of town) consoled him. Later that night, she became so uncomfortable at the vision of her poor son as the only child without a parent present that she cancelled her appointments for the following morning. When she came to school, she found herself the only parent present!

This vignette exemplifies something that is less of a problem for our children than for us as parents because of our fantasies of parental omnipresence. Real mischief results when the child's inevitable complaints about being shortchanged (complaints which occur in every family) strike a resonant chord in a father who shares his child's expectation that mother should aways be there and who resents being called upon to share the responsibility. Child and father can play upon mother's guilt about her "deviant" role and keep her in a chronic state of discontent. For most of us, to the contrary, our greater role in nurturance, beyond the usual didactic and disciplinary functions, has yielded a bouquet of satisfactions our own fathers were denied or denied themselves.

Given the demands of an active professional life, the physician-mother has, on average, fewer hours in the week than the housewife to be with her children. That statement does not translate into what is commonly taken to be its equivalent: that the child is cheated of care. How time is spent is more important than how much time is spent, within wide limits. We underestimate what we give our children.

> Allen's mother overheard him talking to a schoolmate about their parents' occupations. Allen proudly explained that both his parents were psychiatrists who make unhappy people feel better. His companion had no difficulty in describing his father's occupation as an orthopedist who fixed broken bones but he said his mother "didn't do anything." Allen was unable to understand this and kept insisting that she must "do something." His friend finally blurted out: "I know what she does! She yells and hollers at me and talks on the telephone all day!" After his friend had gone home, Allen ran to his mother in the kitchen, embraced her, and said: "Am I *glad* you're a doctor!"

Still, it is an inescapable consequence of the clock that there is more room and more need for fathering, a substantial benefit for us as well as for our children, if

[2]We acknowledge our profound debt to our housekeepers, Mrs. Soares and Ms. Lyons, who unsung and unknown help make our full lives possible.

we are capable of responding effectively. For the loss in absolute time, the children have offsetting gains in the quality of the relationships they can have with both parents. For the girls, wider career horizons are present as tangible possibilities; for the boys, there is exposure to more egalitarian marital roles. They interact daily with a woman as well as a man who is a full participant in the human activities of the workplace and the homeplace. There is less risk for the child of becoming the sole source of parental satisfaction, with its attendant infantilization and impediments to autonomy. Children are expected to contribute more to the functioning of the household since chores must be parceled out. We acknowledge that we speak of potentialities rather than guarantees; competence as a parent is not a necessary consequence of competence as a professional. What we emphasize is, other things being equal, the countervailing virtues within a dual-career marriage that more than offset the constraints imposed by the competing demands on time (11).

One of the most important costs of the dual-career marriage is *time lost* to companionship, a nonsexist cost borne equally by both partners, although even here equality is more apparent than real. Clock time spent apart is by definition the same for both partners, but experienced time may be quite different. To the extent that women are socialized into roles more expressive than instrumental and into greater dependence on affectionate feedback for maintenance of self-esteem, there may be differential pricing in its personal cost. However, we suggest that this is offset by the way men are socialized so that they are less ready to have personal needs for attention displaced by their wives' career demands. However the calculus works out in a given marriage, the fact is that professional work is not contained within a 9:00–5:00, five-day week schedule. Evening and weekend hours are often invaded, in part by emergencies, participation in local meetings and lectures or conferences out of town, and in part by keeping up with the literature and preparing papers for professional journals. Home work is less preemptive than away work because it is carried out in a common residence, can be interrupted for a bit of conversation or an affectionate gesture, and is followed by sharing a common bed. Sleeping together at the least provides comforting companionship, even if in a comatose state, and at its best can lead to something very much better. Time away leaves one partner homebound, beset with all of the household and child-care chores, and feeling lonely, perhaps even abandoned. The more professionally ambitious the spouses are, the more togetherness will be invaded this way. Yet an irreducible minimum follows inevitably from a serious commitment to professionalism as opposed to part-time doctoring. With two partners involved, the effects are psychologically multiplicative rather than additive. The very best efforts to coordinate schedules still result in her being out on evenings when he is home or him out when she is otherwise unengaged. Moreover, if there are young children to be cared for, scheduling may have to be deliberately staggered.

The net effect is a very considerable loss for the marriage, particularly for the good rather than the bad one. The husband and wife who enjoy each other ache at being apart; when the two do not get on well, being apart may partially resolve tensions between them. The couples we know speak sadly of "passing like ships in the night." The wife or the husband may be unavailable just at the very time when the partner needs support. Weekends take on an extraordinary importance as just about the only opportunity for sustained conversation. Social activity, other than that connected with the workplace, tends to atrophy just because time together alone becomes so highly valued and others, even close friends, can be resented for intruding upon it.

It is a problem without a full solution. What each spouse loves in the other is a configuration that would not be the same without the work commitment. But the work commitment diminishes availability. One has less of what one wants. The cost comes with the benefit. Even though the cost cannot be eliminated, we emphasize the importance of containing it. As is the case with medical care, without systems of cost control, time costs will consume the gross marital product.

THE JOYS OF TIME

Despite ourselves and our intent, we find that we have focused on problems and their resolution rather than joys and satisfactions. This may stem, in part, from a medical preoccupation with pathophysiology even amidst general health and in part from masculine embarrassment at public acknowledgment of tenderness and love. Be all that as it may, it remains to us to voice the cascade of pleasures that flow from a wife who is a complete human being in her own right, a "total woman" if we may rescue a phrase from its perversion. We have wives who are intellectual companions as well as lovers, and who are better lovers because we share so much more. It is high privilege, indeed, to live with another who is impassioned and compassionate, a sympathetic listener and a font of fresh insights and understandings, a distiller of wisdom.

We have had—and continue to have—a quite extraordinary experience in marriage, not one we were perceptive enough to seek when we fell in love nor even one we welcomed fully when its depth first became manifest but one with unique meaning for our own growth: the experience of living with a complete woman. We had been socialized to expect less and to settle for the creature comforts that came with that acceptance. Along the way, we may have shown some lack of grace in surrendering the customary conveniences; yet in return we have been granted a love the richer and fuller for having its roots in mutual respect and growing maturity. That, we acknowledge, we owe to our wives. Had it not been for their capacity to insist on respect for their integrity, it is doubtful we

would have achieved what we now enjoy. Of course it was not unilateral; we claim our due for what we have fashioned together; but we know it would not have happened without what they brought us: gentle courage, soft strength, selfless individuality.

Our wives are our best friends. No, it is not because, as the conventional wisdom currently has it, friendship between men is difficult, at least for us as particular men. We have men to whom we are close, whose friendship we treasure, with whom we can share our private thoughts, hopes, and fears. It is that our wives are all that, and more. They do not replace friends or make them unnecessary. We continue to need, and be needed by, friends, both men and women. But our wives, our "successful professional women," are our lovers, our friends, and our comrades in loves and lives of high adventure.

We consider ourselves the most fortunate of men.

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Asylums Revisited

BY ROGER PEELE, M.D., PAUL V. LUISADA, M.D., MARY JO LUCAS, DIANE RUDISELL, AND DEBRA TAYLOR

The authors revisited Erving Goffman's Asylum *(Saint Elizabeths Hospital) and found both the "institutionalization" and "secondary adjustment" phenomena he described 20 years ago. However, they question whether secondary adjustments necessarily follows institutionalization. They also express doubt that institutionalization should be condemned automatically and suggest that it may in fact be the approach of choice for some patients.*

TWENTY years ago, Erving Goffman did a year of field work at Saint Elizabeths Hospital. He said that his "immediate objective . . . was to try to learn about the social world of the hospital inmate, as this world is subjectively experienced by him." He continued,

> I started out in the role of an assistant to the athletic director, when pressed avowing to be a student of recreation and community life, and I passed the day with patients, avoiding sociable contact with the staff and the carrying of a key. I did not sleep in the wards, and the top hospital management knew what my aims were. (1, p. IX)

Parts of his study were published in the late 1950s. The book *Asylums*, which combined his observations of the mentally ill with social aspects of life in other types of institutions, was published in 1961.

Asylums has since become one of the most quoted books on the social aspects of mental illness. It not only spread the realization that institutions are inhumane when poorly funded but also nourished the idea that "institutionalism" or "institutionalitis" is a disease entity in itself that can be developed in the mental hospital and is, in fact, an intrinsic part of the institution, regardless of its staffing and accommodations. Goffman went one step further and suggested that medical and progressive institutions were more degrading than purely custodial ones (1, p. 150).

Other publications at about the same time (2–5), with different orientations, pointed out the social aspects of hospitals that might contribute to patients' behavior and problems in patient management. Together, these books helped create a climate of a need for change that had a tremendous impact on the thinking of the 1960s.

The need to improve the institutions that managed the mentally ill had been a recurrent theme in both public and professional domains for over a century; the call of the 1950s and 1960s was different in that it suggested abolishing the psychiatric hospital rather than improving it. Although the intent of Goffman's work was not to pronounce remedies, *Asylums* became a major resource for those who worked for reform and those who advocated abolition of institutions.

We will begin with the question of whether the setting Goffman visited has changed in the two decades since his stay and, if so, in what ways.

SAINT ELIZABETHS: A COMPARISON OF TWO PERIODS

Hospital statistics suggest that something is quite different at Saint Elizabeths now: the overall inpatient population has shrunk from over 7,000 to 2,700, and outpatient rolls have expanded from several hundred to 2,700. A third of the wards are sexually integrated, whereas men and women were completely segregated in 1956. All wards were closed wards then; now, one-fourth are open. At the time of Goffman's stay, all of the hospital was behind a wall or a fence; now, there is a major portion of the hospital that is not surrounded by a fence. Admissions have more than doubled and the number of deaths, which peaked in the year Goffman visited the hospital, is now less than a third of the 1956 figure. Although exact figures are not available, it is thought that admissions are younger now and the proportion admitted on a voluntary basis has moved from a few percent to about half. Median inpatient stays, which were then a number of months, are about three weeks. Still, none of these figures tell us whether the social phenomena Goffman saw have changed.

In an effort to examine the social phenomena from Goffman's point of view, three summer college students (M.J.L., D.R., D.T.) who were interested in

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The views presented herein do not reflect the opinions or policy of Saint Elizabeths Hospital, the U.S. Public Health Service, or George Washington University.

mental health studied Goffman's work in some detail and then visited some of the hospital wards to determine whether what he saw could still be seen.

A BRIEF REVIEW OF ASYLUMS

Goffman begins by describing a "total institution" as one "whose character is symbolized by the barrier to social intercourse with the outside and to departure that is often built right into the physical plant, such as locked doors, high walls," and so forth. Total institutions share the following characteristics:

> First, all aspects of life are conducted in the same place and under the same single authority. Second, each phase of the member's daily activity is carried on in the immediate company of a large batch of others [who are] required to do the same thing together. Third, all phases of the day's activities are tightly scheduled with . . . one leading . . . into the next. Finally, the various enforced activities are brought together into a single rational plan purportedly designed to fulfill the official aims of the institution. (1, pp 5–6)

Goffman next describes what he called the "moral career" of a mental patient, i.e., the process whereby a person with position, friends, and ties in the community is institutionalized and converted into an inmate whose social sphere is limited to his immediate hospital environment. The institutionalization process begins with a series of assaults on the recruit's self; these assaults strip the patient of his former identity. The patient is then inserted into the lowest rung of an all-embracing privilege system. Goffman asserted that although the stripping process and privilege system are rationalized as being in the patient's best interests on therapeutic grounds, the real purpose is to break his spirit and make him more manageable.

The process of stripping inmates of their identity involves such initiation rituals as trading personal clothes and belongings for hospital issue. The inmate undergoes mortifying procedures that consist of a series of assaults on his self-image. Private, personal activities go on public display; he must request permission for even the most minor activities that were purely volitional on the "outside," such as smoking, shaving, or going to the toilet.

With his prepatient identity in tatters, his outside community stripped away, and his life under constant surveillance, the inmate enters the privilege system, the bases of which are the house rules. Goffman observed that privileges were always reductions in the institution's degree of control over the patient's life; increased "freedom" was the reward for obeying the rules.

Goffman's major point concerned the phenomena he termed "secondary adjustments," which he saw as a direct result of institutionalization and as the hallmark of institutionalism. From the hospital's point of view, the stripping process and privilege system extricated the patient from his presumably pathological environment and introduced him to a therapeutic milieu. Goffman observed that rather than following this intended plan, patients acted as if their major concerns were to escape the hospital's pervasive control.

The inmate found a new identity in the ways he used to reduce the hospital's control over his life; these were the secondary adjustments, defined as "the habitual arrangements by which patients employ unauthorized means, or obtain unauthorized ends, or both." Patients varied in the types of secondary adjustments they made: some became professional errand-runners, using their time off the ward in the various "free places" on the grounds where there was no staff scrutiny. Others hired or were hired by fellow patients to perform services, forming quasi-business enterprises that assured an income independent of staff control. The major portion of the book is devoted to descriptions of the rich patient "underlife" at Saint Elizabeths Hospital—the aggregate of the secondary adjustments of the 7,000-plus inpatients.

Goffman finally considered that some secondary adjustments were overdetermined, i.e., were of no intrinsic value and were undertaken merely because they were forbidden. He saw secondary adjustments as placing a barrier between an individual and the social unit in which he is supposed to be participating, a "rejection of one's rejectors" that resulted from the patient's defensive attempts to keep something of himself from the clutches of the institution. Goffman concluded that some people outside mental institutions may derive a sense of being a person from joining a social unit, but that patients use secondary adjustments as a form of ritual insubordination whereby they obtained a sense of personhood by opposing in a thousand little ways the wishes of those in control of their social system.

Goffman's thesis is that the institutionalizing elements which were intended to produce model patients and a therapeutic milieu instead generated institutionalism, a counterproductive culture of passive resistance that rooted patients more firmly in the institution whose grasp they intended to escape.

THE REVISIT

Method

The years 1955 and 1956 marked a high tide of inpatients. The phenothiazine revolution, a growing negative attitude toward the value of hospitalization, milieu therapies, and the increased ability of patients to receive welfare were forces that have ensured a continuing ebb in the national inpatient census since then. In 1956, Saint Elizabeths' census peaked at more than 7,000; it has dropped to 2,700 today. From Goffman's point of view, how have these two decades of change affected his original asylum, Saint Elizabeths Hospital?

The two of us who are staff members were hardly in

a position to prowl the wards inconspicuously, sniffing out secondary adjustments and seeing the privilege system through the patient's eyes. To duplicate Goffman's vantage point, we undertook a study in which three students with little or no previous exposure to mental hospitals spent six weeks as observers on six inpatient wards. Their task was to observe and record the two phenomena described by Goffman: institutionalization and institutionalism. They were to look for evidence of the institutionalizing process by observing ward procedures and to assess institutionalism in patients by focusing on secondary adjustments.

Before going to the wards, the group developed a 24-item list of objectively verifiable characteristics from Goffman's book that was later consolidated into the following 10 categories:

1. Wanting to go to industrial therapy to get away from ward programs.
2. Doing favors for other patients.
3. No friends outside ward.
4. Bulging handbags, pockets, etc.
5. Repetitive "sad stories."
6. "Unauthorized" use of hospital objects.
7. Panhandling.
8. Employing or employed by another patient.
9. Doing favors for staff.
10. Territoriality.

These characteristics pertain to the specific secondary adjustments that Goffman felt were a result of patients' interactions with the asylum and were intended as a rough measure of institutionalism. Additional data included a description of each ward's daily activities. More subjective information was obtained during repeated visits to the same units over the six-week period as the observers developed relationships with patients. These data were then analyzed to produce the impressions in the Results section. The two authors on the staff then used the checklist to review each program and each patient's condition with the program staff. Institutionalization was measured by the number of structured hours per week, and institutionalism by the number of secondary adjustments in the patients in a given program.

In the 20 years since Goffman's visit, what was then the newest service in Saint Elizabeths Hospital has been converted from a traditional mental hospital inpatient service to a community mental health center. When the conversion occurred in 1969, the fence separating the building from the community was taken down. The building now houses four catchmented general psychiatric units, each consisting of a heterogeneous ward and outpatient team. There is a children's ward and separate inpatient treatment programs for alcoholics and drug addicts.

Results

On the three general psychiatric units, the patients fell into two categories: those whose average length of stay was about 15 days (the minority), and an older group, many of whom have been hospitalized on the ward for many years—a few as far back as Goffman's time.

TABLE 1
Ranking* of Programs in Terms of Institutionalization and Secondary Adjustments

Program	Institutionalization			Secondary Adjustments		
	N	Structured Hours/ Week	Rank	N	%	Rank
Addiction A	38	56	1	0	0	6
Alcoholism B	19	50	2	3	16	4
Alcoholism C	46	40	3	2	4	5
General Unit D	23	17–45**	4	11	48	3
General Unit E	20	16–43**	5	13	65	2
General Unit F	13	16–25**	6	11	84	1

*Ranking is from most to least on both measures.
**The vast majority of patients were at the lower end of these ranges.

In terms of institutionalization and institutionalism, we found sharp contrasts between the general psychiatric wards and those devoted to treating alcoholism and drug addiction. On the general wards there was some evidence of initiation rituals, the stripping process, and the privilege system, with minor differences from ward to ward. In fact, one ward granted no privileges at all to the new patients, reasoning that if a patient was well enough to leave the ward, he was well enough to become an outpatient. An underlife was found and there was ample evidence of secondary adjustments.

The programs for alcoholics and drug addicts contrasted sharply with the general units. Although patient stays were measured in months and ages were comparable to the short-term group on the general units, the stripping process and the privilege system were elaborated and formalized to a degree exceeding anything described by Goffman. For example, a recruit to the drug program who arrives for an admission appointment is seated in a chair facing one corner of a vacant room and asked to wait. If the patient protests that he was on time for the appointment, the wait is even longer, sometimes up to eight hours. When the patient enters the program, contact with the outside world ceases. House rules are elaborate and formalized. The privilege system contains multiple levels, and a large chart announces each patient's position and marks his progress. Another chart shows each patient's place, duties, and hours in the residential work system. Group activities are scheduled back-to-back from breakfast to bedtime, with the exception of an hour a day for "personal needs." The entire system is based on the philosophy that the patient must shed his former identity to be "reborn."

Our findings in the alcoholism programs were similar to those in the drug program, except that the institutionalization was less elaborate and formalized. However, despite this pervasive institutional milieu in both programs, we found much less evidence of secon-

TABLE 2
Diagnosis and Secondary Adjustment

Diagnosis	N	Secondary Adjustments	
		Number	Percent
Organic brain syndrome	15	11	73
Schizophrenia	20	16	80
Other psychoses	4	1	25
Neuroses	7	2	29
Alcoholism	68	7	10
Drug dependence	38	0	0
Miscellaneous	9	3	33
Total	159	40	25

TABLE 3
Length of Hospitalization in Relation to Secondary Adjustments

Length of Stay	N	Secondary Adjustments	
		Number	Percent
Less than 1 year	87	19	22
1–5 years	59	13	22
More than 5 years	13	8	62
Total	159	40	25

dary adjustments and institutionalism than Goffman described.

A more surprising finding emerged when we compared these six wards with each other: there appeared to be an inverse relationship between institutionalization procedures and patient institutionalism (see table 1). The most institutionalizing programs had the least institutionalism, which brings a number of questions to mind. Are secondary adjustments a function of diagnosis and length of hospitalization? Apparently this is the case (see tables 2 and 3), but there is also evidence that these behaviors are present in recent admissions and in patients with a variety of diagnoses.

DISCUSSION

Our results seem to indicate an inverse relationship between institutionalization and institutionalism, but such a conclusion is unwarranted for several reasons.

We think that a patient's condition makes a difference in whether he will be subject to institutionalism. Goffman did not differentiate patients by diagnosis or other characteristics. Table 2 shows a high correspondence between schizophrenic or organic diagnoses and secondary adjustments.

A second possibility would be to accept the generic concept of institutionalism, adding the thesis that institutionalism is just one step in the larger process of placing a patient in another structured setting out of the hospital, such as a half-way house, foster home, nursing home, or the patient's own home. Thus we may have missed many patients who were institutionalized but were no longer in the hospital, seeing instead an atypical group of patients whose institutionalism was not complete and who were not discharged for this reason. This argument assumes that the "completely institutionalized" patient had moved on to other settings and would help to account for our finding that the patients hospitalized for more than one year do not show secondary adjustments to a greater degree than is shown in table 3. This generic premise, however, is modified further by the argument below.

A third argument is that institutionalism is produced by institutionalization, but that Goffman's secondary adjustments are not true indicators of institutionalism. This argument would include the suggestion that the social breakdown syndrome described by Gruenberg and associates (6) or recent similar studies are more valid signs of institutionalism than secondary adjustments. We believe our most institutionalizing programs do produce homogeneous attitudes, behavior, and speech. Goffman's concept of secondary adjustments fails to capture these basic changes.

A fourth argument is that Goffman's secondary adjustments are secondary to some severe mental illnesses rather than to an institutionalizing environment. Do the "revolving door" marginal patients show so-called secondary adjustment behavior? Did the preinstitutional "village madman" of past centuries show the same behavior? Answers to these questions might help clarify the validity of this argument.

A fifth argument, somewhat suggested by our findings, is that secondary adjustments result not from institutional structure but from institutional neglect.

We believe that all five of these arguments have merit and, taken together, explain our findings. In revisiting the asylum we considered diagnosis, length of stay, and degrees of a program's institutionalism. Goffman did not consider any of these fully. We do not doubt that institutionalism takes place, but we now suspect that Goffman did not accurately describe its characteristics because he failed to differentiate programs that had a great deal of institutionalization from those which did not.

This revisit has also forced us to face the question of whether institutionalization is as bad for all patients as was thought two decades ago. The most publicly admired of the six programs we surveyed are those with the most institutionalization. Still, extreme forms of regimentation are as distasteful today as they were in Goffman's time. We would suggest tentatively that extreme forms of institutionalization be adopted as part of a mental health program only as the last resort for certain otherwise untreatable patients because of the psychological pain it causes those patients and its economic cost to the public. Our observations suggest that if an institutionalizing program must be adopted to fill the needs of some subcategories of patients, it should be done under the following conditions: 1) vol-

untary admissions, 2) homogeneity of programs, 3) blending of staff-patient roles, 4) planned prolonged hospitalization, 5) nonpsychotic primary diagnoses, 6) heavy use of psychological and social restraints and little or no use of physical restraints or medications, and 7) prompt discharge at the patient's request.

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Outpatient Treatment of Schizophrenia

BY NORRIS HANSELL, M.D., M.S.HYG., AND GRANT L. WILLIS, M.A.

The authors discuss the treatment of 575 schizophrenic patients over the past 15 years. They describe a shift the patients have experienced from reliance on inpatient treatment to heavy use of outpatient services. They have found that effective outpatient treatment for these patients involves a program of nearly continuous administration of neuroleptic medication combined with counseling and social and crisis services. A major component of the outpatient management of these schizophrenic patients is the education of the patients in how to regulate their own medication within a prescribed range. This educational component appears to add a measure of precision that enhances the safety and reliability of the treatment program.

WE ARE ENGAGED in the continuing study of 575 schizophrenic patients treated in a mental health center in an area of central Illinois that two decades ago had few local mental health services. In the past 20 years, local services have been developed to the point that the site of most care is near the patient's home. This shift to local services has occurred during the lifetime of some of our study patients and allows us to compare the appearance of their illness under both institutional and outpatient styles of treatment.

THE PATIENT GROUP

The median age of the 575 schizophrenic patients is 37 years; the youngest is 15 years old and the oldest is 88. Fifty-two percent of the patients are women. All but 18 have been hospitalized at least once, and most have been hospitalized two or more times. The diagnostic criteria have changed over the past decade but have remained conservative. The earlier criteria were presence of thought disorder and affect reduction, delusions, or hallucinations, all in the absence of gross brain or toxic disease. The more recent criteria also require a duration of episode of more than 6 months and the absence of intoxicated state or broad affect (1). Although the diagnoses were initially made by a number of different physicians, we established the diagnosis of all 575 patients in the study group. We regard this patient group as composed of seriously ill schizophrenic patients, either chronic remitting or chronic unremitting. We believe the group includes few mild, single-episode, or schizophreniform cases.

The study group represents a large percentage of the schizophrenic patients in our area who repeatedly undergo hospital or emergency care. For example, of all the schizophrenic patients admitted to local hospital care in 1975 from Champaign and Ford Counties, Ill. (population, 176,000), 82% are in our group of 575 patients. All but 7 of our study patients receive continuous or long-term/intermittent neuroleptic medication. Forty-one percent live alone, and an additional 11% live in group care, sheltered care, or nursing care facilities (most of these, but not all, are aged persons). Thirty-one percent of our 575 patients receive regular payments or pensions from welfare, Social Security, or veterans' agencies.

In 1975, 12% of our patients were hospitalized for at least 1 day, and 16% came to the hospital emergency room or the mental health center's crisis service. Two percent moved into distant hospital care after moving through local hospital care in 1975. Twenty-eight percent use day care and lodge-based social services to varying degrees.

We have made no attempt to discharge any of the 575 patients from continuing service, but we try to keep inpatient stays short. Counseling and social services are often suspended, sometimes for a prolonged period of time, after the resolution of life challenges. We attempt to find out whether neuroleptic medication is essential as well as to find the lowest effective dose of medication and the most convenient schedule of medication supervision that will work reliably. The average daily maintenance dose for our patients is equal to 2–5 mg of fluphenazine; the average daily dose during episodes is equal to 30–60 mg of fluphenazine.

The fact that using primarily outpatient services is a relatively recent phenomenon in central Illinois can be gathered by comparing two subgroups of our sample of 575 patients. An older cohort of 30 patients born in the 10 years following 1915 experienced an average of 72 days per person per year in state hospitals during the 3 years following 1962. The same cohort experienced an average of 5 days per person per year in state hospitals

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during the 3 years following 1971. The shift to new local services occurred in the interim. A younger cohort of 30 patients born in the 10 years following 1945 experienced a total of 77 admissions to a state facility in the 3 years following 1962 and a total of only 2 admissions in 1974 and 1975. The younger cohort continues to use local inpatient services occasionally (4 days per person per year). Thus it can be seen that in addition to a move to local inpatient services, there has been a major reduction in the length and total days of hospitalization in the past 15 years.

A major increase in the use of outpatient services has unfolded over the same interval. The older cohort made an average of 2 outpatient visits per person per year during the 3 years following 1962 and an average of 38 visits per year (including all contacts) in the 3 years following 1971. The contacts in 1963 were largely for counseling and sporadic chemotherapy. Ten years later the older cohort made contacts that involved an average of six types of service per year, including chemotherapy, counseling, day care, home visits, mutual-help groups, legal aid, court intervention, crisis services, and membership in the Lodge (a nonresidential, club-like setting for mutual-help groups and other services to assist in forming affiliations).

The younger cohort also made an average of more than 30 outpatient visits per year; these involved a similarly large variety of services. The younger cohort is not using as much inpatient care as the older group, but it is using more varied outpatient care more frequently. The comparisons within the older cohort and between the older and younger cohorts show that the interval between 1962 and 1975 has seen a distinct shift to outpatient care and to more varied types of care. However, there is a continuing use of inpatient care, and there is no sign that the total amount of care is decreasing.

RELIABILITY OF MEDICATION OVER DECADES

The accumulating evidence that some types of schizophrenia involve a biological disturbance with a lifelong pattern of risk, the clinical experience that neuroleptics decrease the likelihood of exacerbations in some schizophrenic patients (2–4), and the hazards of prolonged neuroleptic use (5) lead us to approach the proposal of chronic neuroleptic use carefully. If we determine that neuroleptics are essential for a given patient, we observe that patient to ascertain the lowest effective dose and propose arrangements for a long-term pattern of continuous or semicontinuous use of medication.

We deal with the problem of tardive dyskinesia by strictly assessing the necessity of neuroleptics, by using them intermittently if possible, by using the lowest effective dose, and by instructing the patient to call us if unusual mouth, trunk, or limb movements occur. More than four-fifths of our 575 patients omit their daily dose 1 or 2 days each week, except during episodes. Three of our patients have developed tardive dyskinesia in the past 2 years. All 3 have recovered after 1–12 weeks; 2 of these were hospitalized with psychosis when neuroleptics were withdrawn. These patients continue to present a problem; we have used reserpine during hospital episodes.

All of the individuals in our group of 575 schizophrenic patients have been counseled that their disease is likely lifelong, that it may involve altered brain biology, and that it is often substantially modifiable with chemical and social regulation. The counseling focuses on analogies to diabetes, on fluctuations in the course of the illness due to adaptational challenges, and on self-regulation of medication within a prescribed range. We tell our patients our opinion that lifelong care for their condition, if done with precision, does not result in an increase of sickness but in an increase of health and function. We also tell them that if their condition later warrants a less continuous mode of care we can shift our approach on the basis of strategic observations. We are concerned about the possibility that our informing the patients of our attitude of observation-based personal health planning might constrict a patient's overall situation, but we are not aware of any instance in which this has occurred.

Hogarty and associates (2) found that a high percentage of patients who were rehospitalized had experienced their exacerbations after they had stopped taking their medication. Similarly, in our group, not taking medication was highly correlated with a return of symptoms and with rehospitalization. It often takes 3–6 months or longer for such exacerbations to occur. Drug holidays lasting several days to several weeks usually do not accurately foreshadow longer-term consequences. Patients need to understand these time relationships because, after they learn that they likely have a lifelong program ahead of them, many show a keen interest in personally assessing the effects of their medication.

Our goal for these schizophrenic patients is the reliable administration of the lowest effective amount of medication over an interval of decades. Therefore, we focus the clinical relationship on teaching patients enough about their disease, the drugs needed to control it, and their reactions to the drugs so that their full energies can be applied to the purpose. If a patient wants to stop taking medication when he or she is doing well clinically or if a patient voices an intention to assess the consequences of stopping medication, we encourage him or her to do so but only under close supervision.

Patients are instructed in a method for looking for the earliest signs of deregulation, i.e., the symptom cluster of disturbance of the sleeping-waking cycle, floating affects (sadness, panic, suspicion, perplexity), difficulty concentrating, preoccupation with a narrow range of thoughts, and loss of pleasure in social contacts. In the past 2 years, 38 or our 575 patients have assessed the effects of stopping medication; 36 of these

patients experienced a return of the early-warning symptom cluster or frank psychosis and returned to the use of medication.

Two other reasons for stopping taking medication are side effects, broadly considered, and cost. Early in treatment, we ask our patients to work closely with us if side effects occur. We have concluded that patients who experience side effects and do not tell us about them or work with us on their control eventually stop taking the offending drug. Early in treatment we encourage our patients to discuss side effects fully, including the subtler forms of akathisia (6). More than two-thirds of our 575 patients experienced some side effects, most of which were manageable with persistent attention. Twenty-two percent are taking an atropinic material for control of motor side effects. Forty-six percent have done so occasionally. If cost becomes a controlling factor in whether or not a patient continues to take medication, the center assumes or defrays the cost.

Of equal importance in establishing a reliable pattern of medication administration over decade-length intervals of time is helping the patients achieve a substantial degree of self-regulation of medication within a prescribed range. Most of our patients do best when taking the lowest dose that will control the sleep disturbances and floating affects. At the same time, the patients are also instructed in a method for increasing their medication during episodes of life challenge. Teaching patients to regulate themselves within a range offers major gains in reduction of side effects, reduction of overall amount of medication taken and thus of the chance of developing tardive dyskinesia, and enhancement of reliability.

We counsel patients to use some of the following experiences to signal that they are taking too low a dose: a breakup in the orderliness of the sleeping-waking cycle, diminished concentration or preoccupation with a few ideas, floating affects, ideas of influence or reference, and such psychosomatic symptoms as headache or paresthesia. In raising the dose, we instruct patients to increase it by a half and wait 3 days to assess the effect. They may repeat this maneuver several times.

Similarly, we counsel patients that if they experience forms of dissociative phenomena or sleeping-on-inactivity they might be taking too high a dose. In this event we instruct patients to reduce their dose by a third and wait 3 days to assess the effect. We estimate that more than 400 of the 575 patients in our study group are systematically increasing and decreasing their own doses successfully. The rest of the patients, most of whom are more than 50 years old, prefer to be given an unvarying dose routine and to make deviations from it only on direct instruction from a physician.

Of the total group, 28 receive all or part of their medication in the form of depot fluphenazine. Seven of these are given their injections by a visiting nurse; the rest are given the drug in the emergency room of a local general hospital. Most of these patients were not successfully managed in a program of self-regulation and also experienced repeated hospitalizations when they stopped taking medication. Patients in this group must give their consent and continuing cooperation for any depot fluphenazine administration, so education and counseling are still important for them. We regularly lower the dose and stretch the interval between injections to reestablish the minimum effective amount.

There are two main principles in gaining the cooperation of patients for decade-long programs of medication. First, we recommend the use of no more or less medication than is necessary and take steps to gain information on this point. Second, the actual consequences of any decision to stop or change medication deserve a close watch; we will assist the patient at any time to make such a decision.

THE SELF-REGULATION GROUP

Many of our patients who eventually became quite successful in self-regulation of their medication seemed to have learned such skills most effectively in a group context. The self-regulation group, which was organized for this purpose, meets weekly under the direction of a psychiatric nurse. The group members discuss and react to each other's problems with medication for control of psychiatric symptoms. The composition of the group is constantly changing. Some patients use it 4–16 times, mainly during the induction phase; a few attend almost indefinitely. The size of the group ranges from 8–22 members. Of our 575 patients, about 55 are attending or have attended the group.

The usefulness of the group resides in the fact that it provides a setting for an individual to learn what he or she may need to know to make decisions about medication use and for the more skilled to teach the less skilled in self-regulation. Learning occurs through the reports each patient gives on the state of control of his or her target symptoms, together with reports on side effects and defining objectives for improving the state of self-regulation. Members take individual responsibility to contact their physician to discuss any changes or actions they wish to take regarding medication. The most useful features of the group are its ability to convert reluctant or careless medication users into skilled self-regulators and its role in raising the average level of self-regulation to close to the best level represented in the group.

ADAPTATIONAL EPISODES

For well-controlled schizophrenic patients the onset of an adaptational episode is typically not first signaled by the presentation of psychotic behavior or hallucinations. Difficulty sleeping, difficulty concentrating, lowered work endurance and task focus, floating affects

(including sadness, panic, perplexity, anger, suspicion, and certitude), and, for a smaller group, frank ideas of reference or preoccupation with a few ideas usually signal the onset of an episode. Coincident with these early signals of decompensation are the loss of enjoyment in being with people and a loss of a feeling of attachment to friends and kin.

We try to teach schizophrenic patients to be more effective managers of their adaptational episodes. During such crises most of our patients increase the dose of medication that is usually effective in controlling their symptoms; this increase averages a doubling or tripling of their ordinary dose and lasts from several weeks to several months.

At the beginning of an adaptational episode we encourage patients to increase their dose of medication and to watch their sleeping-waking and resting-working rhythms as markers of the completeness of their regulation. We then encourage them to feel for a comfortable pace in entering and moving through the situation. We encourage them to get rest, food, water, and relaxation during the period of challenge, remembering that the problem-solving effort may extend over many weeks. We ask them not only to try to formulate the current problem but also to become aware of what they want for the future; we also ask them to describe the past in such a way as to define the current situation in the context of a distinct past and an envisionable future. We encourage patients to name their helpers and to keep in regular reporting contact with them. The regulation of adaptive work seems to proceed best when all parties in the process have an opportunity to form a task group. Above all, we ask our patients to avoid getting exhausted and to avoid isolation. We encourage them to stay hooked to others, to a path, and to a pace.

OTHER CHEMICALS

The outpatient care of schizophrenic patients presents a continuing need to be aware of the interaction between the disease and the medications used for its control and alcohol, sedatives, minor tranquilizers, anorectics, and decongestants. The clinical problems arising from these interactions are not uncommon. Sixty-three of our 575 patients had one or more problems involving such interactions in 1975.

The problem we find with alcohol is that patients sometimes attempt to use it to control schizophrenic symptoms, particularly the sleep disturbances and floating affects. Not only is alcohol ineffective in the control of these symptoms in schizophrenia, but time is lost and attention is diverted from more precise methods for regulation of symptoms. The clinical problem arising from the use of minor tranquilizers, sedatives, and soporifics is the same as the problem with alcohol. These materials are much less efficient than neuroleptics in regulating the sleeping-waking cycle and the floating affects of schizophrenia. In addition, it has been our experience that the duration of action of minor tranquilizers can be measured in hours, in contrast to the much slower, enduring effects of neuroleptics. The too-brief duration of action of minor tranquilizers yields inherently destabilizing aftereffects for both patient and clinician.

Of a different nature are problems with amphetamines, ephedrine, and congeners. Amphetamine psychosis has long been known to mimic schizophrenia; it is not so widely recognized that continuing use of small amounts of sympathomimetics has a deregulating effect on chronic schizophrenia. Their use often yields subtle or dramatic exacerbations in the course of the illness, the origin of which is not apparent without precise inquiry. In our group of 575 patients, 26 have shown such interactions; this number is likely an underestimate because we might not have recognized many of the events due to the fact that we came to appreciate this matter only recently.

It is not mainly sympathomimetics prescribed by physicians that constitute the bulk of this problem but the presence of these materials in over-the-counter pharmaceuticals, particularly diet pills and items intended as cold pills and respiratory decongestants. For example, we have found three diet pill items marketed locally that contain phenylpropanolamine, namely Diet-Trim, Hungrex, and Slender-X. The list of proprietary decongestants containing phenylephrine, phenylpropanolamine, ephedrine, and congeners is long. For example, it includes Allerest, Benzedrex, Contac, Coricidin "D," Dristan, Mistol, Neo-Synephrine, Novahistine, Ornex, Romilar, Sinarest, Super-Anahist, Vicks-Sinex, and 4-Way Cold Tablets. We ask our patients to avoid the use of decongestants altogether; there seems to be a difference in the number of exacerbations due to this problem during the winter season of respiratory infections.

SUMMARY

In central Illinois the period since the early 1960s has seen a shift in the management of chronic schizophrenia from heavy use of inpatient treatment to heavy use of outpatient treatment. For schizophrenic patients who might otherwise be repeating users of hospital service, effective outpatient service appears to involve a program of continuous, or nearly continuous, neuroleptic medication combined with counseling, social, and crisis services. A substantial amount of education focused on the patient's role in self-regulation of medications within a prescribed range appears to allow useful improvements in safety, precision, and reliability.

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Change in ABPN Part II Examination Format

Beginning with the October 1977 American Board of Psychiatry and Neurology Part II examination, the one hour of oral examination in Neurology previously administered to Psychiatry candidates will be replaced by a written examination. The written examination is based on a series of audio-visual vignettes of neurological patients. Each vignette will last approximately three to four minutes and will be followed by written, multiple-choice questions pertinent to the brief patient presentation. There will be no examiner involvement or patient contact in this section.

Thus, all new Psychiatry candidates will take three sections during the Part II examination:
1 one-hour examination in Clinical Psychiatry (live patient)
1 one-hour examination in Clinical Psychiatry (audio-visual)
1 one-hour examination in Clinical Neurology (audio-visual)

The Medical Basis of Psychiatry

BY ARNOLD M. LUDWIG, M.D., AND EKKEHARD OTHMER, M.D.

The authors state that the medical model represents the most useful and appropriate model available for the practice of psychiatry. They discuss the implications for psychiatry of the three essential features of the medical model: the concept of disease and diagnosis, the concept of etiology and treatment, and the nature of the doctor-patient relationship. They conclude that the adoption of the medical model for psychiatry would have profound implications for residency training, continuing education, delivery of care, standards of ethical and professional practice, dealings with third-party payers, and conflicts with other professions.

THE OBJECTIVE of this paper is to clarify the essential features of the medical model for the practice of psychiatry. The entire thesis derives from the basic premise that the primary identity of the psychiatrist is as a physician (1, 2).

Because of the "model muddlement" that has beset psychiatry (3), comment is necessary about models in general. Models are neither real nor sacrosanct; they are abstractions invented by the human mind to place facts, events, and theories within an orderly framework (3). Just as a grid or wheelspokes design may determine the layout of a city, models provide conceptual templates for the organization of information. Because models are neither true nor false, they can be judged only by their relative usefulness or uselessness. A model that is not sufficiently comprehensive, that excludes contradictory or incongruous data, or that requires frequent restructuring to accommodate new information ceases to be either intellectually gratifying or of practical value. It is our contention that the medical model represents the most useful and appropriate model available for the practice of psychiatry.

THE MEDICAL MODEL

There are three essential features of the medical model: the concept of disease and diagnosis, the concept of etiology and treatment, and the nature of the doctor-patient relationship. We will discuss the implications of each of these features for psychiatry.

The Concept of Disease and Diagnosis

The primary signs and symptoms of disease are often characterized by five major features: 1) with the exception of genetic-congenital conditions, they indicate a change from a previous healthy state; 2) they represent deviations from an appropriate reference group for the individual; 3) they display varying probabilities of association (e.g., certain signs occur almost always with certain symptoms and vice versa); 4) their cross-sectional patterns of association may differ from their patterns over time; and 5) they contribute to or are associated with impairment in psychological and social functioning (i.e., secondary signs and symptoms).

When different patterns of signs and symptoms tend to be associated with different patterns of morbidity and/or mortality, different diagnostic labels drawn from an accepted classificatory scheme are employed to differentiate among these patterns, each of which is presumed to be produced by natural causes. The diagnosis of disease, then, represents an attempt at categorization of information and a probability statement regarding the expected association among certain signs and symptoms with their course over time, their underlying pathological basis, and their possible etiological sources. Because of modern detection and screening methods, the early stages of some diseases may be established by "silent" signs before the appearance of any symptoms.

There are four main types of disease diagnoses used in medicine. The degree of reliability and precision in diagnosis is directly related to the degree of knowledge of primary cause of the particular pattern of symptoms and signs. If the specific etiology of this pattern can be determined, either in vivo or post mortem, a causative diagnosis can be made (e.g., bacterial infection). If only tissue or organ changes can be demonstrated (without knowledge of their cause), a pathological diagnosis can be made (e.g., cancer). If only the syndromatic association of symptoms and signs and their natural history are known, a descriptive-prognostic diagnosis can be made (e.g., Ménière's disease). If isolated symptoms are not associated with any signs, then only a symptomatic diagnosis can be made (e.g., tension headache).

Critics who invoke the imprecision and poor reliability of many psychiatric diagnoses as an argument

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TABLE 1
Categorization of Selected Affective-Cognitive-Behavioral Disorders

Disorders with Biological Dysfunction			Disorders with Social-Psychological Dysfunction		
Neuropsychiatry	Functional Psychiatry	Medicopsychiatry	Social Work	Social Psychology	Clinical Psychology
Epilepsy	Schizophrenia	Hypothyroidism	Problems of living	Criminology; forensic issues	Problems of living
Headache	Bipolar affective illness	Hyperthyroidism	Minority problems	Social deviance	Learning disabilities
Dementia; organic brain syndrome	Unipolar affective illness	Alcoholism	Poverty	Anomie syndrome	Marital difficulties
CNS syphilis	Episodic dyscontrol syndrome	Obesity	Family troubles	Dependency syndrome	Existential depressions
Huntington's chorea	Sociopathy	Hypochondriasis	Coordination of community services		Adjustment reactions
Narcolepsy	Anxiety; phobic reactions	Drug dependence			Personality disorders
Delirium	Conversion hysteria	Sexual dysfunction			Coping deficits
Parkinsonism	Hyperkinetic syndrome	Psychosomatic problems			
Mental retardation	Borderline syndrome	Anorexia nervosa			

against their nonmedical nature fail to realize that a similar situation pertains to many nonpsychiatric medical diagnoses (4). This relative unreliability in diagnosis does not indicate that diagnosis should be abandoned but that more precise criteria should be developed.

On the basis of the above considerations, psychiatric disease would pertain to any affective-cognitive-behavioral disturbances that impair a person's adaptive and adjustment responses and in which some known, suggestive, or presumed biological abnormalities or dysfunctions (mainly genetic, biochemical, or physiological in nature) play a significant role. In certain instances, this biological dysfunction represents the necessary but not the sufficient condition for the appearance of the disease (e.g., schizophrenia); in other instances, both the necessary and the sufficient condition (e.g., toxic psychosis); and in still other instances, the sufficient condition alone (e.g., anxiety attack). In all instances, this biological dysfunction will be associated with altered forms of brain activity, reflected in some impairment in reception of information or sensory stimuli, cognitive or central processing of signals, and general behavior outputs. Concern for the biological components of disease does not preclude an understanding of environmental factors.

This concept of disease and nondisease has profound implications for psychiatry. Psychiatry previously has relinquished responsibility for the care and management of diseases with known physical etiologies (5), such as general paresis, mental retardation, pellagra, and epilepsy. To a certain extent, this trend is continuing in the areas of alcoholism and drug dependence. Psychiatry now assumes primary responsibility for diseases of unknown etiology, such as schizophrenia and manic-depressive psychosis, and for a multitude of social-psychological problems presumably caused by metapsychological conflicts, faulty learning, poor interpersonal communications, and social inequities. On the basis of a medical definition of psychiatric disease, the proper purview of psychiatry should be modified to include certain borderline conditions that overlap with neurology and medicine and to exclude those clearly in the domain of the social sciences.

Psychiatric diseases may be classified as either neuropsychiatric, medicopsychiatric, or functional psychiatric. The designation "functional" indicates that, either because of inadequate theories or limited technology, the presumed biological dysfunction has not yet been established. Excluded from the disease concept are so-called problems of living, social adjustment reactions, maladaptive learning patterns, situational depressions, and various social deviance syndromes because it is assumed that sustained biological dysfunctions are neither necessary nor sufficient for their appearance and that somatic therapies are not indicated for their treatment. Personality disorders, existential depression, and certain learning disabilities would be more appropriately placed in a gray zone in terms of presumed biological dysfunction. Only the accumulation of more scientific knowledge can determine whether these disorders constitute psychiatric disease or not.

Table 1 presents a categorization of some selected disorders to illustrate the presumed areas of special expertise of the psychiatric physician with respect to

TABLE 2
Selected Types of Disease Theories

Type of Theory	Etiological Requirements	Example of Treatment	Level of Treatment
Infectious (e.g., bacterial and viral infections)	Koch's postulates; presence of specific antibodies	Germicidal agents	Curative
		Increase in immune response	Compensatory
		Suppressant agents	Syndromatic
		Symptomatic agents	Symptomatic
Deficiency state (e.g., rickets, scurvy, dehydration)	Decrease in necessary nutrients, minerals, or hormones; reversal of pathology with replacement	Replenishment	Curative
Excess state (e.g., Cushing's syndrome, acromegaly, atherosclerosis, hypercalcemia)	Increase in necessary nutrients, minerals, or hormones; reversal of pathology with depletion	Dietary control	Curative
		Counteracting agents, radiation, surgery	Compensatory
Imbalance (e.g., acid-base or electrolyte imbalance, cardiac arrhythmia)	Hyperplasia or hyperfunction versus hypoplasia or hypofunction of organ, tissue, or system	Agonist or antagonist	Compensatory
		Corrective surgery	Curative, compensatory
Toxicity (e.g., heavy metal poisoning, silicosis, radiation sickness)	Presence of toxin, poison, noxious agent, or abnormal metabolite	Decrease in toxin by binding agents, antidotes, antisera	Curative, compensatory
		Symptomatic agents	Symptomatic
Immune-autoimmune (e.g., asthma, hives, collagen disease, rheumatoid arthritis)	Antigen-antibody response with local or general reaction	Desensitization	Curative, compensatory
		Antiallergic agents	Syndromatic, symptomatic
Continuum (e.g., essential hypertension)	Deviation from "normal" range	Corrective agents	Compensatory, syndromatic, symptomatic
Traumatic stress (e.g., traumatic arthritis, fracture, noise deafness)	Anatomic and/or functional pathology secondary to trauma	Surgical repair	Compensatory
		Symptomatic agents	Symptomatic
Degenerative-destructive (e.g., cataract, aseptic necrosis, tumor)	Anatomic and/or functional pathology without external trauma	Prosthesis	Syndromatic
		Surgical repair	Compensatory, syndromatic
Genetic (e.g., Huntington's chorea, phenylketonuria, idiopathic epilepsy, sickle-cell anemia)	Positive family history; identification of abnormal functions or structures	Supplemental agents	Compensatory
		Prosthesis	Syndromatic
		Surgery	Compensatory, syndromatic
		Symptomatic agents	Symptomatic
Congenital (e.g., cleft palate, mongolism)	Negative family history; identification of abnormal functions or structures resulting from intrauterine or birth factors	Prosthesis	Syndromatic
		Surgery	Compensatory, syndromatic
		Symptomatic agents	Symptomatic
Secondary disease (e.g., Addisonian crisis, radiation burn, dumping syndrome)	Side effects or complications of treatment for primary disease	Substitutive agents	Curative, compensatory
		Symptomatic agents	Symptomatic

differential diagnosis and primary responsibility for treatment. Since not all patients presenting at mental hygiene clinics, emergency rooms, or physicians' offices have a psychiatric disease, the psychiatrist should be proficient in distinguishing among those disorders potentially associated with a biological dysfunction and those with a social-psychological dysfunction. Once this determination is made, the psychiatrist should assume primary therapeutic responsibility for the former disorders; nonmedical professionals might assume primary responsibility for the latter. This does not mean that the psychiatrist should focus exclusively on disease and the nonmedical professional on problems of living. The issue is one of emphasis and expertise. If the physician chooses to work with some "clients," which must always remain his or her prerogative, then he or she cannot claim any special expertise over his or her nonmedical colleagues, especially since their training in this area may be superior.

The Concept of Etiology and Treatment

In general, levels of treatment tend to correspond directly to the degree of knowledge available about etiology of disease. We may designate these levels of treatment as curative, compensatory, syndromatic, and symptomatic. Curative treatments pertain to elimination of causative agents or factors with consequent reversibility of damage and reduction of the need for further treatment. Compensatory treatments pertain to the minimizing of further dysfunction and the maximizing of existing function. Syndromatic treatments pertain to the suppression of major manifestations of

TABLE 3
Etiological and Treatment Aspects of Psychiatric Diseases

Item	Type of Etiological Theory	Example of Treatment	Level of Treatment
Functional psychiatry			
Schizophrenia	Deficiency state, excess state, imbalance, toxicity, immune-autoimmune, and genetic	Megavitamins	Compensatory
		Major tranquilizers	Compensatory, syndromatic
		Anticholinergic agents (for drug side effects)	Symptomatic
Bipolar affective illness	Deficiency state, excess state, imbalance, and genetic	Lithium	Curative*, compensatory, syndromatic
		Major tranquilizers	Compensatory, syndromatic
		ECT	Syndromatic*, symptomatic
Unipolar affective illness	Deficiency state, excess state, and genetic	Tricyclics and monoamine oxidase inhibitors	Compensatory*, syndromatic
		ECT	Syndromatic
Sociopathy	Deficiency state, imbalance, immune-autoimmune, genetic, and congenital	Antidepressants	Compensatory*
		Symptomatic drugs	Symptomatic
Anxiety; phobic reactions	Excess state and imbalance	Minor tranquilizers and sedatives	Symptomatic
		Systematic desensitization	Curative, compensatory, syndromatic
Conversion hysteria	Excess state (e.g., corticofugal inhibition)	Narcosynthesis	Compensatory, syndromatic
		Hypnosis	Compensatory*, syndromatic
Medicopsychiatry			
Alcoholism	Deficiency state, excess state, toxicity, continuum, genetic, and secondary disease	Disulfiram	Curative*, syndromatic
		Sedatives and minor tranquilizers	Symptomatic
		Vitamins	Compensatory
		Aversive conditioning techniques	Syndromatic, symptomatic
Drug dependence	Deficiency state, excess state, immune-autoimmune, toxicity, and secondary disease	Methadone	Compensatory, syndromatic, symptomatic
		Antagonist agents for overdose or prevention	Compensatory, syndromatic
Obesity (bulimia)	Excess state, imbalance, continuum, genetic, and secondary disease	Diet control	Curative
		Amphetamines	Compensatory, syndromatic
		Behavioral techniques	Curative, syndromatic
		Surgical bypass	Syndromatic, symptomatic
Primary hypochondriasis	Imbalance and secondary disease	Suggestion (placebo)	Symptomatic
		Symptomatic agents	Symptomatic
Hypothyroidism	Deficiency state and secondary disease	Replacement	Compensatory
Neuropsychiatry			
Idiopathic epilepsy (temporal lobe type)	Excess state, genetic, and congenital	Anticonvulsants	Syndromatic
		Surgery	Compensatory
Huntington's chorea	Imbalance, degenerative-destructive, and genetic	Haloperidol	Symptomatic
		Symptomatic agents	Symptomatic
Presenile dementia (Alzheimer's and Pick's disease)	Toxicity, degenerative-destructive, and genetic	Symptomatic agents	Symptomatic
Parkinsonism	Infectious, deficiency state, and degenerative-destructive	L-dopa	Compensatory
		Anticholinergic agents	Syndromatic
		Amantadine	Syndromatic
Narcolepsy	Imbalance, genetic, and congenital	Stimulant agents	Syndromatic
		Antidepressants	Syndromatic
Mental retardation	Infectious, deficiency state, excess state, toxicity, immune-autoimmune, continuum, degenerative-destructive, genetic, and congenital	Diet (phenylalanine)	Compensatory
		Symptomatic agents	Symptomatic
		Special learning techniques	Compensatory

*We are not sure of these levels of treatment.

the illness but do not ensure correction of the underlying dysfunction or elimination of all residual effects. Symptomatic treatments pertain to the suppression of selective manifestations of the illness without elimination of underlying causes or residual effects.

It should also be noted that the reliance on scientific

method to assess efficacy of treatment becomes increasingly important as less is known about the actual etiology of a particular disease. Single case studies may be sufficient to evaluate the effectiveness of treatment intervention for such causative diagnoses as inguinal hernia or pellagra, but double-blind controlled treatment studies are essential to evaluate the relative efficacy of treatment for such syndromatic diagnoses as migraine headache or bipolar depressive illness.

As we have indicated, there are many etiological theories of disease. Table 2 provides a list of 12 major types of medical disease theories, examples of specific diseases, the requirements to test the theories' validity, examples of treatments used, and current levels of treatment approaches. This categorization is arbitrary in that there are several etiological theories under investigation for any particular disease. In general, the more theories that are advocated for a particular disease and the more a unitary theory is proposed for all diseases, the less is known about actual etiology.

Table 3 shows that similar theories are operative in the biological investigation of most psychiatric diseases,[1] although inadequate conceptualization and technology prevent the gathering of objective laboratory and other diagnostic tests. Just as there are numerous etiological disease theories under investigation in cancer research (e.g., infectious, toxic, imbalance, degenerative, and autoimmune), a similar situation pertains to such psychiatric diseases as schizophrenia. Despite unconfirmed etiologies, these theories of disease provide many avenues for experimental investigation and the development of specific, more effective therapies.

There has been sufficient research on stress and altered levels of sensory stimulation in psychiatry to indicate the profound effect of many environmental variables or situations on the biological functioning of individuals. Acknowledged environmental influences are not incompatible with a disease concept of etiology, but their often vague conceptualizations are. However, even if environmental variables can be specified, it is essential to presuppose or demonstrate the existence of a biological diathesis preceding or a biological dysfunction following the introduction of environmental variables as a necessary or sufficient condition for the appearance of disease. When no sustained biological dysfunction can be demonstrated or presumed, no disease can be said to exist.

The Doctor-Patient Relationship

The physician is granted awesome responsibility, aptly termed "Aesculapian authority," for the care and welfare of the patient (6, 7). The physician makes life and death decisions about patients; violates their bodily integrity and privacy through physical examination, diagnostic procedures, and surgery; and gains access to their innermost thoughts. This responsibility is held in check by equally awesome ethical obligations to act in the best interests of patients, to maintain confidentiality, and to provide quality care.

In his or her assessment of patients, the physician may judge the patients to be sick, thus granting a socially sanctioned decrease in general responsibility and implying that the illness cannot be due to or cured by volition alone (3, 7). However, the sickness does not need to correspond to the particular sick role adopted by certain patients (8). This general viewpoint stands in contrast to most nonmedical models, in which a patient may be regarded as not sick (i.e., as a client or an analysand) and therefore not granted the sanctioned decrease in responsibility. The patient's problem may be viewed as bad or deviant and caused by his or her witting or unwitting decisions or learning.

Since the special responsibilities and prerogatives of physicians have at all times pertained only to the treatment and prevention of sickness or disease, it could legitimately be questioned whether psychiatrists abrogate their authority as physicians when they treat only "nonsick" individuals by means of individual counseling, family and group therapy, or sensitivity sessions.

THE PRACTICE OF PSYCHIATRY

The relevance of the medical model for psychiatry must start with the premise that the psychiatrist is a physician. As a prerequisite for actual diagnosis and treatment, the psychiatric physician must obtain an adequate and comprehensive data base. This includes detailed and skillful interviewing of the patient or other informants and careful physical, neurological, and mental status examinations.

Having elicited special symptoms and signs, the psychiatric physician then resorts to the process of differential diagnosis in an attempt to account for the various symptoms and signs according to the probability of their association with certain diseases or nondisease reactions. After the formulation of a differential diagnosis, a specific diagnosis is needed. For this process, the psychiatric physician uses a variety of laboratory tests and specific diagnostic procedures to rule out certain diagnoses and to confirm others. For the functional psychiatric diseases, for which there may not be specific laboratory tests, syndromatic diagnoses may be made on the basis of standardized diagnostic criteria (9).

No diagnosis of disease should be made that does not carry with it implicit information about likely age of onset, course and duration, prognosis, and type of treatment available. The diagnosis of psychiatric disease should not be made by exclusion but on the basis of positive biological or behavioral criteria.

As to the administration of therapy, the psychiatrist should possess particular competence in and knowledge of the use, side effects, and complications of all appropriate somatic therapies; certain short-term procedures, such as hypnosis, behavior modification, and

[1]Because of space limitations, general references are not provided; they will be furnished by Dr. Ludwig on request.

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biofeedback techniques to treat conditions for which somatic therapies are not as yet very effective (e.g., anxiety and phobic neuroses and hysterical conversion reactions); and skill in the selective administration of supportive, insight-oriented, and problem-solving techniques to resolve problems confounding, contributing to, or complicating the primary disease.

Group therapy and ward management programs are entirely compatible with the medical model, provided they meet the three following criteria: 1) they should not overlook the biological component of disease; 2) they should be designed to increase treatment compliance; and 3) they should promote optimal functioning of patients. In his or her role as physician, the psychiatrist must view the patient as something more than his or her primary disease; the patient may display many maladaptive attitudes and behaviors existing independent of the disease that influence recovery and rehabilitation. In order to facilitate recovery and optimal functioning in patients, the psychiatrist may use psychosocial interventions as the only means available to modify harmful attitudes and behaviors.

With medicopsychiatric and neuropsychiatric diseases, the psychiatric physician should be sufficiently competent to undertake the primary treatment of most of these patients, in consultation, if necessary, with appropriate specialists. It represents fragmented patient care, for example, to refer a depressed person to a neurologist for treatment of headaches and to an internist for management of obesity and mild hypertension.

BEYOND DISEASE

It is essential to emphasize that management of the sick patient entails far more than the diagnosis and treatment of disease; the patient's illness must be addressed as well. According to Barondess (10), a disease represents a biological event of a pathogenic nature that can be understood mainly in scientific terms (i.e., objective, quantitative, and reproducible). An illness, on the other hand, is a human event and represents "a grouping of discomforts, dysfunctions and resultant personal and social dislocations occurring in a person, and representing the interaction of that person with a disease" (p. 154). Illness must be understood as an important life event, associated with many others and embedded within a matrix of concerns, hopes, fears, and responsibilities experienced by the patient. A physician whose role transcends that of scientist is obligated to address not only the diseased condition but also the person who is ill and his or her immediate family, who may be distressed and confused by the situation.

As long as the medical model pertains to the care of patients, its dependence on science will never be complete; there will always be room for the art of medicine. In the practice of this art, the psychiatric physician has the opportunity to bring all of his or her interviewing skills, perceptivity, and empathy to bear, much in the manner of any adept physician (1).

Those who discount the medical model because of its presumed emphasis on organ pathology and disease process rather than on the whole individual not only possess a limited concept of disease but also ignore the entire idealistic tradition of medicine. Humanism and a belief in the basic dignity of man are not the monopoly of psychiatry. They are implicit in the oath of Hippocrates, accepted by all physicians. These values can be just as easily imparted during a brief physical examination with the administration of diagnostic procedures as during an insight-oriented psychotherapy session. The extent to which these values are not imparted to patients is not so much a failing of the medical model as a failing in the practicing physician. There is no inherent contradiction between the treatment of disease and care and concern for the ill individual.

Some have impugned the medical model as too reductionistic or restrictive to deal with the complexities of human nature. Such criticisms are more pejorative than legitimate. If there is reductionism in the medical model, it pertains only to an emphasis on specificity and the elucidation of causality. If there are restrictions in the medical model, they pertain to its emphasis on objectivity and scholarship, its application of contributions from science, and its commitment to distinguishing fact from fiction in all aspects of psychiatry.

The adoption of the medical model for psychiatry would have profound implications for residency training, continuing education of practitioners, revisions of standards for Board certification, the delivery of psychiatric care, education of the public about the proper role of the psychiatrist, the establishment of standards of ethical and professional practice that discourage radical schools of thought, negotiations with third-party insurance payers, and territorial conflicts with other professions. This indeed will represent a formidable challenge for psychiatry. Such changes can represent only a beginning that reaffirms the foundation of psychiatry in medicine.

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A Comparison of Videotaped and Live Patient Interview Examinations and Written Examinations in Psychiatry

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The authors scored the examination performance of 28 psychiatrists who were tested after conducting an interview with a real patient and after seeing a videotaped interview with an actor playing the role of either a psychotic or a neurotic patient. They also scored the performance of 15 of the same examinees who completed a written examination. Eighty-five percent of the examinees reported that the videotaped interview examination was a useful learning experience, and 81% said that it was a useful testing experience. The examiners' feelings varied regarding the use of the standardized videotaped format for certification and recertification of psychiatrists.

THE AMERICAN BOARD of Psychiatry and Neurology (ABPN) initiated the use of a videotape of an actual patient interview during Part II of the Board examination in March 1975 (1). A systematic report of the effects of this new method of assessment is not yet available. Voluntary anecdotal reactions of candidates and examiners have been mixed. If the Board increases its current responsibilities by broadening its scope from certification to recertification as well, a videotaped interview of a real patient or an actor or actress is a potential means of developing a more standardized, generalizable, and economical assessment method of a psychiatrist's competence.

The College of Family Physicians of Canada (2) and the American Board of Pediatrics (3) have introduced simulation methods in their certification examinations with reported success. Although some studies support correlations between a clinician's performance with patients and his or her ability to be examined by means of a real or videotaped physician-patient interaction (4–6), a literature search we conducted revealed no such reports involving psychiatrists specifically. We also found no comparisons of performance in any field between situations in which the clinician knew the interview was a simulation and those in which he or she did not know it was a simulation. Furthermore, unpublished studies by the ABPN suggest that correlations exist between high written performance scores and high oral examination scores, but supportive data for such statements are not available and have been questioned in published studies (7–11). In an effort to address some of these concerns, we undertook a study with the following questions in mind:

1. What are the similarities and differences and advantages and disadvantages of assessing a psychiatrist's skills by means of his or her conducting an observed clinical interview and then being questioned by examiners as compared with his or her observing a videotaped clinical interview and responding to a set of standardized questions asked by examiners?
2. Do the two methods of assessment differ as to examinee performance ratings and as a learning technique in the opinions of both examinees and examiners?
3. What is the relationship between each of these assessment methods and a written objective examination?
4. Does it make a difference in performance scores on the videotaped interview examination if the patient is neurotic or psychotic or if the examinee is or is not informed that the patient being interviewed is an actress or actor?

METHOD

Examinees

A group of 28 psychiatrists who recently completed an intensive review of psychiatry and neurology course in preparation for Part I or II of the ABPN examination volunteered for the study. Each interviewed a patient and observed a videotaped interview as part of the course. Each was randomly assigned to the order in which the two examinations were taken and randomly assigned to examiners so that each examinee was tested by two different examiners. Each test situation lasted approximately one hour and was scheduled a week apart. Candidates were informed that the examination using the live patient interview format

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At the time this work was done, Dr. Naftulin was Associate Professor of Psychiatry, Dr. Wolkon was Associate Professor of Psychiatry (Psychology), Mr. Donnelly was Instructor in Psychiatry (Psychology), Dr. Burgoyne was Assistant Professor of Psychiatry, Dr. Kline was Associate Professor of Psychiatry, and Dr. Hansen was Associate Professor of Pediatrics and Psychiatry (Child), University of Southern California, Los Angeles, Calif. Dr. Naftulin is now in private practice at 9911 W. Pico Blvd., Suite 1270, Los Angeles, Calif. 90035.

would be conducted according to ABPN methods and scored using ABPN criteria. The examiner met the patient and had his or her chart available for study before the examinee interviewed the patient. Examinees were also informed that because ABPN was considering a videotaped interview format as a possible means of conducting future examinations, such an examination procedure would be included in the course.

Examiners

Four full-time academic psychiatrists served as examiners. All had examined psychiatrists on Part II of the ABPN examination at least once before and had conducted simulated oral examinations for residents and colleagues using the ABPN format. In our study two examiners conducted 12 interviews, one conducted 13, and one conducted 19. On one day the examiner tested the candidates after they viewed videotapes; on another day the examiner tested different candidates after they interviewed a patient.

Examiners used the ABPN pass, condition, or fail criteria in scoring both the videotaped and the actual patient interview. They were provided with an additional evaluation form for each test condition. An abbreviated version of the American Board of Orthopedic Surgery's 4-item interpersonal skills instrument (12) was used in the patient interview situation, and an item checklist of 10 topics on which the candidate was assessed was developed for the videotaped test situation. Examiners viewed the videotapes ahead of time, agreed generally on the working diagnosis of the "patients," agreed on answers to the questions asked, but disagreed about the importance given to some topics covered by the instrument. Nevertheless, all agreed to use it. At the completion of the study each examiner was asked specific questions in order to compare his attitude about the two examination formats, and each examiner completed a subjective narrative of his experience with the study.

Patients

All of the patients who agreed to be interviewed by the examinees were hospitalized with psychosis, thought and/or affective disorders, or brain syndromes.

Videotaped Interviews

The videotaped presentations consisted of a real psychiatrist interviewing actors playing the role of a psychotic or a neurotic patient.[1] Two bearded young men in their late 20s played both the psychotic and neurotic roles.[2] The order of actor and the order of role were randomly assigned to the examinees. For example, each examinee observed a 10–12 minute interview of one actor playing the role of a psychotic or neurotic patient and was examined on what he or she observed. The examinee then viewed the other actor playing the second diagnostic role and was examined after that interview. To determine whether the examinee's performance was affected by his or her perception of the videotaped patient as a real patient or an actor, a random selection of 14 of the 28 examinees were told that the videotaped patients were actors. The other 14 examinees were not specifically told but led to believe that the interviews were of actual patients. At the conclusion of the videotaped interview examination, all examinees were told that the videotaped patients were actors.

Written Examination

Eight weeks after the videotaped interview examination, 15 of the 28 examinees responded to an 80-item multiple-choice instrument designed like APA's Psychiatric Knowledge and Self-Assessment Program and Part I of the ABPN examination. Most items were selected by means of an item analysis from a longer test administered to a similar group of psychiatrists the previous year.

FINDINGS

Videotaped Interview Examinations

The scores earned by the candidates on the videotaped interview examination did not significantly differ (according to two-tailed t test) as a function of the order in which the examinees observed the neurotic or psychotic patient roles ($t=1.23$), which actor played which role (neurotic, $t=1.22$; psychotic, $t=0.59$), or the order in which the examinees took the videotaped or live patient interview examination (neurotic, $t=0.31$; psychotic, $t=1.25$). The examinees' scores for the psychotic role interview were highly correlated with their scores for the neurotic one ($r=.85$). This result suggests that testing for both categories of patients is not necessary for this type of examination.

The performance scores of the examinees did not differ significantly as a function of whether they were or were not told that the patients were actors (neurotic, $t=1.12$; psychotic, $t=0.08$). Each examinee was asked if it would have made a difference in his or her performance had he or she been told that the patient was an actor and not an actual patient or vice versa. All examinees in both test conditions reported that believing the patient was either an actor or an actual patient would not have affected their performance. These findings indicate that it makes no difference in examination performance whether examinees are told that the patient in the videotape is real or an actor or actress.

Live Interview Examinations

After interviewing a real patient, each examinee was rated on an 8-point scale (12) by the observer-examiner in four areas: 1) ability to obtain a history, 2) ability to relate well to the patient, 3) ability to apply factual

[1]We are grateful to Warner Johnson, M.D., for his participation.

[2]We are grateful to Eric Berk and David Reynolds, Ph.D.

TABLE 1
Mean Scores Given by Examiners in the Real Patient Interviews

Criterion	Mean Score Given by Examiner				F	Significance
	1	2	3	4		
Ability to obtain a history*	4.75	4.63	5.50	5.86	2.32	n.s.
Ability to relate well to the patient*	5.63	5.00	5.17	5.86	1.04	n.s.
Ability to apply factual information in a clinical setting*	3.75	4.13	4.83	5.57	3.22	$p < .05$
Overall final rating*	3.13	4.25	4.67	5.86	8.32	$p < .001$
ABPN numerical score**	72.88	73.50	73.16	82.86	5.48	$p < .01$

*Rated on an 8-point scale: 1 = inadequate; 8 = outstanding.
**75 and above = pass; 74–70 = condition; 69 and below = fail.

information in a clinical setting, and 4) an overall final rating. Each examinee was also given an overall numerical score according to ABPN criteria (see table 1). The examiners did not differ on the ratings they gave in areas 1 and 2 listed above. However, examiners significantly differed on their ratings of areas 3 and 4 and on the ABPN ratings. Application of Duncan's multiple range test (13) on each of the three significant items indicated that one examiner (examiner 1) contributed most to the statistical difference (see table 1).

Videotaped Interview Examination Scores Compared with Others

The overall mean performance scores for the live interview and videotaped interview examinations did not differ significantly ($t=0.91$). However, the score an examinee earned on one of the examinations was of no help in predicting his or her score on the other examination. The correlation was not significantly different from zero ($r=.08$). Even when the numerical scores were categorized into pass, condition, and fail, we found no statistically significant relationship between the two examinations, despite 65% examiner agreement in categories of the examinations ($\chi^2=.23$).

The relationship among the test scores of the 15 psychiatrists who took the written as well as the two oral examinations was also not significant. The written and the live interview examination correlated ($r=.39$). The written and the videotaped interview examination correlated ($r=.09$).

Oral Examinations

After the videotaped interview examination, each examinee was asked whether the procedure was a useful learning experience and a useful testing experience. Twenty-four of the examinees (85%) said that it was a useful learning experience, and 23 (81%) said that it was a useful testing experience.

The examiners were asked to write comparative evaluations of the two interview examination methods. They said that the videotaped interview examination did not allow them to estimate the examinee's ability to establish rapport, relate to a patient, demonstrate interpersonal skills, and elicit relevant information. On the other hand, they also said that the standardization inherent in the videotaped interviews was obviously lacking in the actual patient interview.

The issue of examinee anxiety was raised in terms of both formats. In addition to expected test anxiety, the examinees suggested that psychiatrists who were not accustomed to being observed while interviewing might have undue anxiety in the patient interview format; they also suggested a number of obvious test preparation procedures. The examinees reported that the videotape format created specific anxieties because of its unique artificiality and because it did not allow the examinee the flexibility and discussion inherent in the traditional ABPN format.

One of the examiners suggested combining the videotape format with a written examination. This would have the advantage of being more standardized and less expensive than the traditional examination format. The potential advantages of the videotape format were spontaneously suggested by the examinees in this study. Their suggestions included systematically varying the complexity and ambiguity of the symptoms of the "patient" in the videotaped interview as well as matching types of patient to the subspecialty emphasis of the examinee.

Specific technical points raised by the examiners will not be mentioned here. The administrative confusion that can occur in the actual interview format, such as the patient's arriving late or not arriving at all and not being able to find an office for the interview, are eliminated or greatly reduced by using the videotape. One issue raised by the examiners was the relatively greater amount of preparation they needed in the standardized videotape format compared with what they perceived as more spontaneity and less preparation in the traditional ABPN format. The examiners also pointed out a related issue: examining with the videotape was more tedious and monotonous.

One examiner totally rejected the videotape format as a useful way to examine because of the above reasons. Another examiner, although not rejecting the videotape format outright, stressed the importance of the candidates being examined on the basis of patient encounter. A third examiner felt that both methods were fair and both were adequate tests for certification. He further stated he would prefer to examine in

the traditional manner but would prefer to be examined in the more standardized manner. The fourth examiner recommended the use of both formats in the total certification and recertification process.

DISCUSSION

Contrary to our predictions, the patient-centered and videotape-centered scores of examinees did not correlate positively. Further, contrary to ABPN claims (unsupported by published data) that written scores in the basic sciences not only effectively screen candidates but correlate positively with oral examination scores, our findings demonstrate no such correlation. In addition, neither the oral patient-centered examination scores nor the videotape-centered scores correlated with the written scores. These findings indicate that approximately the same performance scores are earned with a neurotic or psychotic patient after a videotaped interview and that examinees reported it would make no difference if the live or videotaped patient were real or an actor.

The lack of relationship between the videotaped interview examination and the traditional oral examination may be explained in terms of examiner variables and patient variables. Using real patients means that some examinees will interview "easy" patients and others will interview "difficult" patients, creating a major potential source of error in assessing the performance of a psychiatrist expected to show a minimal level of competence. This error could be corrected by standardizing the clinical test situation by means of the videotaped interview.

These data indicate that there are significant differences among examiners in their assessments of the candidates in the real patient interview examinations but that none existed in the videotaped interview examinations. Apparently, the experience the examiners had in actual and simulated ABPN examinations did not successfully establish standardized criteria for evaluating psychiatric performance. The low agreement among examiners about the importance of questions in the videotaped interview examinations may have led to a small amount of inconsistency. Thus in both types of examinations, but especially in the ones involving real patients, there is a need for improved standardized scoring procedures with experienced examiners well-trained in such procedures.

The examiners reported that it was easier to compare examinees using the videotape format than the live patient format because the standardization of the former reduced differences of actual patient selection and assignment. In addition, the standard list of questions reduced the possibility of gross error by the examiners. A list of standardized questions may also be useful in the real patient examination, but its appropriateness would be influenced by controlled patient variables if patients are selected by means of the current ABPN method.

IMPLICATIONS FOR RECERTIFICATION

Among medicine's responses to public concerns over accountability is an increasing effort to assure the clinical quality of its practitioners (14, 15). Recent efforts to assure clinical competence in psychiatry have included appeals for continuing education, certification, and recertification (16, 17). The small number of certified psychiatrists in comparison with other specialists has been sufficiently documented (18). Third-party payers, limited licensing proposals, salary differentials, and the inevitability of a national health insurance program will increase the rate with which psychiatrists seek certification and probably recertification. The ABPN joined 22 members of the American Board of Medical Specialties in a resolution to adopt recertification as a goal (19); it has also solicited APA component groups for positions on recertification. Although concerns about psychiatry's certifying procedures as well as droll personal experiences are well documented (16, 20–25), ABPN is to be commended for its openness in providing at least limited feedback to candidates concerning their performance and eliciting feedback from candidates as to how they perceived their "rite of passage." Since at least one study documents the perceptions of patients examined by candidates (26), it is hoped that future studies will systematically reflect candidates' perceptions of their examination experiences and that such perceptions will be compared with candidate performance.

Board-eligible and Board-certified psychiatrists—satisfied, dissatisfied, or apathetic with ABPN procedures—deserve an informed method of assessing the ability of their assessors to assess them. In an era of increasing concern over accountability, the Board must increase its accountability to colleagues eligible for certification and to those diplomates soon to be recertified. Clinical competence levels must be made explicit, examination procedures standardized, and examiners better trained to examine for designated skills and knowledge among colleagues. Correlations must be established among different test methods (written examinations, videotaped interview examinations with real patients or actors, and actual patient interview examinations) before one method can sufficiently replace or complement another in a meaningful way. Findings from this study indicate that such correlations are not significant.

Recertification provides one path psychiatry can take in its efforts to assure quality care for patients. However, findings from this study suggest that the means by which certification and recertification are to be achieved remain a persistent challenge for psychiatry in the United States.

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Platelet MAO Activity in Schizophrenia and Other Psychiatric Illnesses

BY JOHN SULLIVAN, M.D., CHARLES N. STANFIELD, AND CHARLES DACKIS, M.D.

Previous reports have indicated that platelet MAO activity is low in some schizophrenic and other psychiatric patients. However, other studies have not been able to replicate this phenomenon. This study supports the previous observations of low enzyme activity in some chronic schizophrenic patients. The authors discuss the thesis that low platelet MAO activity may be a biochemical marker for vulnerability to psychiatric illness.

THE RELATIONSHIP between low platelet MAO activity and psychiatric illness in general and schizophrenia in particular continues to interest many behavioral scientists and clinicians. Since Murphy and Wyatt's 1972 report (1) of lower platelet MAO activity in 33 chronic schizophrenics than in 22 controls, 14 follow-up studies have appeared in the literature. Seven of these confirmed Murphy and Wyatt's observation of low platelet MAO activity in some schizophrenic patients, whereas the other 7 studies failed to demonstrate a significant difference in enzyme activity in schizophrenic patients.

The first follow-up study (2) to confirm the findings of the initial report showed that the mean MAO activity of both schizophrenic and nonschizophrenic twins in 13 monozygotic pairs was significantly lower than that of 23 nonpsychiatric controls. Because drugs, diet, and environmental factors at the time of blood sampling could not readily explain the decreased MAO activity in twins discordant for schizophrenia and since platelet MAO activity is under genetic influence (3), Wyatt and colleagues (2) concluded that low platelet MAO activity might provide a genetic marker for vulnerability to schizophrenia. Meltzer and Stahl (4) subsequently reported that platelet MAO activity in 12 chronic schizophrenic and 10 acute schizophrenic patients was significantly lower than that of 15 normal subjects. Nies and associates (5) also showed that platelet MAO activity was significantly lower in 12 schizophrenic patients than in 12 normal subjects. In addition, these authors, like Meltzer and Stahl, noted that the choice of substrate was critical to the demonstration of a difference in enzyme activity.

In 1975 Zeller and associates (6) reported low platelet MAO activity in a large group of schizophrenic patients compared with nonpsychiatric controls. On the basis of different substrate affinities between the MAO from the schizophrenic patients and controls, Zeller and his group suggested that the platelet enzyme was structurally different in the schizophrenics. Two additional reports of low platelet MAO activity appeared in this journal. Domino and Khanna (7) studied 13 male chronic schizophrenic patients who had not received psychoactive medication for at least six months before evaluation. Schildkraut and associates (8) identified a subgroup of schizophrenic patients characterized by low platelet MAO activity, auditory hallucinations, and paranoia. In a recently published study demonstrating reduced platelet MAO activity in schizophrenic patients, Murphy and associates (9) could not show parallel reductions in the activities of other platelet enzymes (including succinate dehydrogenase, cytochrome C reductase, and lactate dehydrogenase), thus suggesting that the reduced MAO activity could not be explained by nonspecific platelet changes.

To our knowledge, the first report that failed to confirm Murphy and Wyatt's observation of reduced platelet MAO activity in schizophrenic patients appeared in this journal in 1974 (10). Although Friedman and colleagues did not find a difference in platelet MAO activity in this study of 26 schizophrenic patients and 23 nonpsychiatric control subjects, they did discover a tendency toward lower MAO activity in the schizophrenic group; this tendency was more marked in the women. Similarly, Shaskan and Becker (11) were unable to demonstrate reduced platelet MAO activity in 24 schizophrenic patients compared with 15 controls. These investigators found a bimodal distribution of MAO activity in the nonpsychiatric subjects and alcoholics used as controls as well as in the schizophrenic patients. A more recent study (12) of 680 nonpsychiatric subjects demonstrated a unimodal frequency distribution of platelet MAO activity, and 2 other recent studies (13, 14) have also shown

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low platelet MAO activity in patients with alcoholism. Therefore, as Murphy and Wyatt have suggested, sampling and technical difficulties may have influenced the findings of Shaskan and Becker (11).

A study by Carpenter and colleagues (15) also failed to demonstrate a difference in enzyme activity between 44 acute schizophrenic patients and 131 nonpsychiatric controls. However, it should be noted that studies of adopted-away children of schizophrenic patients (16) suggest that acute and chronic schizophrenia are different illnesses. Takahashi and associates (17) reported similar platelet MAO activity in "33 schizophrenic patients . . . newly admitted . . . who had never been treated with psychoactive drugs" and in "50 normal control subjects" with lower platelet MAO activity in "43 . . . chronically ill schizophrenic patients . . . on . . . various phenothiazines." Three additional studies failed to show significantly lower platelet MAO activity in chronic schizophrenic patients. In two of these studies (18, 19) the schizophrenic patients were receiving conventional doses of neuroleptic medication; in the third study (20, 21) they had not been treated with antipsychotic medication for at least six months.

Thus the present literature on platelet MAO activity is interesting and suggestive of a link with some psychiatric illnesses, but it leaves many questions unanswered. We will therefore present additional data and theoretical inferences that may prove useful in delineating the relationship between platelet MAO activity, schizophrenia, and other psychiatric illnesses.

METHOD

The control group consisted of 53 men between 21 and 59 years of age (mean = 38.8, SEM = ± 2.0) who were selected from surgical patients hospitalized at the Durham Veterans Administration Hospital for herniorrhaphy or hemorrhoidectomy (N=39), Veterans Administration personnel (N=6), and Duke University students (N=8). None of these individuals gave a history of past medical or psychiatric illness. Some of the surgical patients were taking a benzodiazepine for sleep. Otherwise, there was no history of drug use in this group.

The 21 schizophrenic patients in the study were male veterans, between the ages of 21 and 51 years (mean = 31.1, SEM = 1.8), who were hospitalized on the psychiatry service with a diagnosis of chronic undifferentiated schizophrenia or paranoid schizophrenia over the same time period as were the surgical patients. All of the psychiatric patients were evaluated by a first-year psychiatric resident and by a Board-certified senior staff psychiatrist. Additional information was obtained on the psychiatric patients from social workers and ward psychiatric nurses. All of the schizophrenic patients admitted to the study met the *DSM-II* criteria (22) for chronic undifferentiated schizophrenia or paranoid schizophrenia as well as Feighner and associates' criteria for schizophrenia (23). Each of these patients had been hospitalized from 3 to 18 times over a period of 3–26 years. Some of them were also taking a benzodiazepine for sleep. However, there was no past or present history of alcoholism, drug abuse, organic brain syndrome, or chronic medical illness. All of the patients gave a history of taking neuroleptic medication in a dose range equivalent to 400–1700 mg of chlorpromazine. Informed consent was obtained from all of the subjects after the nature of the study had been fully explained.

None of the control subjects or schizophrenic subjects demonstrated abnormalities on peripheral blood smear, red blood cell count, hematocrit, hemoglobin, mean corpuscular volume, mean corpuscular hemoglobin, or mean corpuscular hemoglobin concentration. Physical examination revealed no evidence of dermatitis or peripheral neuropathy. These criteria were included in the protocol because iron-deficiency anemia (24) and riboflavin deficiency (25) have been associated with low platelet MAO activity.

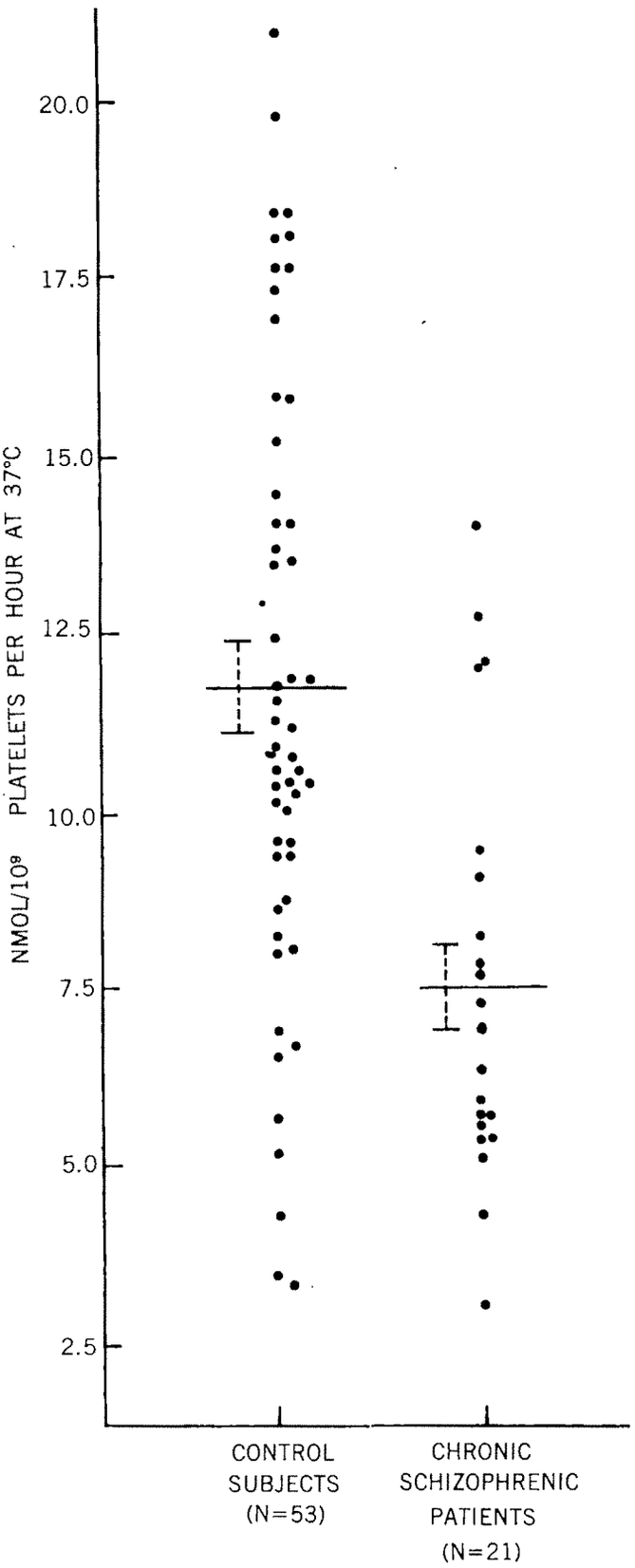
Venous blood samples (18 ml) were collected for platelet MAO activity determinations, and platelet pellets were prepared by sequential centrifugations of platelet rich plasma (PRP) samples obtained during the first week of hospitalization. Some of the platelet pellets were frozen from several days to a week at −80°C so that enzyme activities from subjects of both the control and schizophrenic groups could be measured at the same time. Previous work in our laboratory has demonstrated that freezing the pellets from several days to six weeks results in negligible loss of enzyme activity. Platelet counts were determined on PRP aliquots using a standard electronic particle counting procedure (Coulter Electronics Thrombocounter, Curtin Matheson, Atlanta, Ga.).

Washed platelet pellets were homogenized in a Thomas grinding vessel and distributed as .05 ml aliquots containing .2–.5 mg of protein. The aliquots were incubated with tryptamine, prepared from tryptamine bisuccinate, side chain $-2-^{14}C$ (New England Nuclear), and tryptamine HCl, at a final concentration of 7×10^{-5} M in .08 M phosphate buffer, pH 7.2. The reaction was terminated by addition of HCl, and the product was extracted into washed toluene. Product formation was assessed by liquid scintillation spectrometry (Intertechnique Model SL 30) using external standards. Phosphate buffer was used instead of protein for the blanks. The reaction was linear for both time and platelet count within the dimensions of the assay. Method and sample variation (calculated as the difference between paired samples divided by their mean) was ±4% for duplicate samples from the same specimen. Additional details of the assay procedure have been published elsewhere (26).

RESULTS

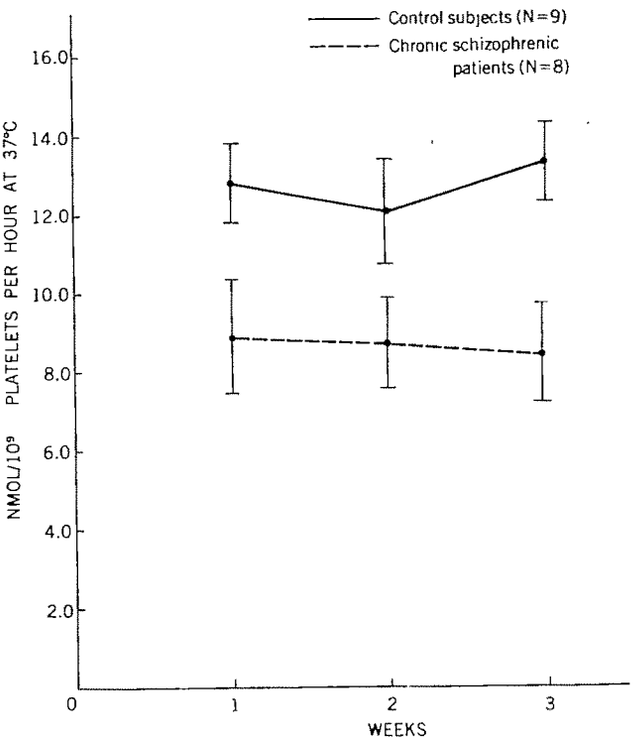
As demonstrated in figure 1, mean platelet MAO activity for the chronic schizophrenic group was 7.7±.7

FIGURE 1
Platelet MAO Activity in Chronic Schizophrenic Patients and Control Subjects*



*Mean activity was significantly lower in the schizophrenic patients ($p<.001$).

FIGURE 2
Mean Platelet MAO Activity in Chronic Schizophrenic Patients and Control Subjects over a Two-Week Period



nmol/10^9 platelets per hour (mean±SEM) and 11.7±.6 nmol/10^9 platelets per hour for the controls. Thus mean platelet MAO activity was 34% lower in the schizophrenic group ($p<.001$, two-tailed t test). The platelet count for the schizophrenic group was $2.85\times 10^8\pm.15$ (mean±SEM) and was not significantly different from the platelet count for the control group ($2.90\times 10^8\pm.18$). Figure 2 shows that the mean platelet MAO activity values were relatively stable over a two-week period for both the schizophrenic and control groups. (Due to technical limitations in the laboratory and attrition, only 9 control subjects and 8 schizophrenic patients were followed.) The mean intrasubject coefficients of variation for the schizophrenic subjects (17.1%±3.3%) and the control subjects (16.0%±2.6%) were within experimental error.

DISCUSSION

The low platelet MAO activity observed in the chronic schizophrenic patients in this study is consistent with the seven previous reports of low enzyme activity in some schizophrenic patients and lends additional support to the validity of Murphy and Wyatt's original observation (1). The results of our study cannot be explained by differences in race or sex, and age does not appear to be a significant variable. Although the schizophrenic subjects were significantly younger

than the control subjects (3.1±1.8 years versus 38.8±2.0 years) ($p<.05$, two-tailed t test), the study by Robinson and associates (27) on the effects of aging on platelet MAO activity showed a gradual increase in platelet monoamine oxidase activity in men after the age of 45.

Two subsequent studies (12, 21) failed to show a correlation between platelet MAO activity and age. Diet, activity, and the effects of hospitalization also do not appear to be significant variables in that the mean enzyme activity of the schizophrenic subjects was still significantly less than that of the 39 surgical patients (7.7±.7 versus 12.0±.7, $p<.001$, two-tailed t test). Drug effect is also an unlikely explanation for the low platelet MAO activity observed in the schizophrenic subjects. Although there is some evidence that neuroleptic medication has a slight inhibitory effect on MAO activity in vitro and in vivo (17, 28), other investigators have demonstrated increased MAO activity after treatment with neuroleptics (21). There is also a report in the literature (9) that 10-fold to 1,000-fold higher drug concentrations are required for in vitro MAO inhibition than are obtained in plasma during treatment with neuroleptic medication. There is ample additional evidence to suggest that the low platelet MAO activity observed in some schizophrenics is not merely drug effect (2, 7, 29).

These observations are particularly interesting in light of reports that some patients with bipolar affective illnesses (30, 31) and alcoholism (13, 14) demonstrate low platelet MAO activity. Severely disturbed manic-depressive subjects are often clinically indistinguishable from schizophrenic subjects, particularly paranoid schizophrenics (32, 33), and alcoholism has been associated with both schizophrenia (34) and manic-depressive illness (35). Thus low platelet MAO activity may be associated with several forms of psychiatric illness. Buchsbaum and colleagues (36) have provided further support for a relationship between low platelet MAO activity and psychiatric illness. They found that probands with low enzyme activity in a population of 375 college student volunteers had 8 times the incidence of suicide or suicide attempts in their families as well as twice as many psychiatric problems as did subjects with high enzyme activity. These authors therefore suggested that low platelet MAO may predict vulnerability to psychiatric disorder.

Although it is possible that low platelet MAO activity reflects low MAO activity within the central nervous system, the relationship between platelet MAO and brain MAO remains problematic (37). As with reports on the platelet enzyme, reports on regional brain MAO activity in schizophrenia are contradictory. There are two reports in the literature of low regional brain MAO activity in schizophrenics (38, 39) and four statements of normal regional brain enzyme activity in schizophrenics (40–43).

Another important issue is the role of genetics in the low platelet MAO activity observed in some psychiatric patients. As previously discussed, Wyatt and associates proposed, on the basis of their twin study (2), that platelet MAO activity may provide a genetic marker for vulnerability to schizophrenia. Zeller and associates' data (6) suggested that the platelet MAO of the schizophrenic subjects they studied was different structurally from that of their nonpsychiatric controls. Our finding of stable platelet MAO activity over 2 weeks in both the controls and the schizophrenics is consistent with Murphy and associates' observations (12) of controls and psychiatric patients over 8–10 weeks and implies that platelet MAO activity is a relatively stable characteristic of the individual. In addition, previous work (3) has shown that platelet MAO activity is under genetic control. Therefore, low platelet MAO activity may provide a genetic marker for vulnerability to psychiatric illness. The clinical expression of the mental illness could result from the interplay between the degree of penetrance of the genetic lesion and psychosocial factors.

However, the substrate and enzyme kinetic studies of other investigators (9) argue against an alteration in the active site of the enzyme, and another group of researchers (44) could not demonstrate a difference in electrophoretic patterns of platelet MAO in chronic schizophrenic subjects, manic-depressive subjects, and nonpsychiatric controls. Additional physicochemical, animal, and clinical studies are necessary to delineate whether the low platelet MAO activity observed in some patients with psychiatric illness reflects primarily a genetically determined structural abnormality of the enzyme or another factor such as a more fundamental biochemical alteration, changes in the lipid environment of the enzyme (45), enzymatic inhibition, changes in the synthesis or degradation rates of MAO (46), psychosocial factors, or chronicity of the illness.

Another promising approach suggests that the low platelet MAO activity in schizophrenics and other patients with psychiatric illness results from immunological factors. For example, some studies have demonstrated in vitro morphological transformations of lymphocytes from schizophrenic patients, suggesting antibody production (47). Other investigators have reported antibodies to brain protein and lymphocytes in schizophrenics (48). There are also reports of low activity of leukocyte oxidative enzymes (including MAO) isolated from schizophrenic subjects, and supplementary animal experiments suggest that these low enzyme activities result from immunological changes (49, 50). We have also recently typed lymphocyte MAO, using multiple substrates and inhibitors, and found it identical to platelet MAO. In addition, we have demonstrated a decrease in lymphocyte MAO activity in the same schizophrenic subjects who demonstrate low platelet MAO activity (unpublished data, from Sullivan and Stanfield). Thus the low platelet MAO activity observed in some patients with psychiatric illness may result from immunopathological factors.

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Foundations' Fund Prize for Research in Psychiatry

The Board of Directors of the Foundations' Fund for Research in Psychiatry has made a grant of $30,000 to the American Psychiatric Association for the establishment of an annual Foundations' Fund Prize for Research in Psychiatry.

Candidates for the prize must be citizens of the United States or Canada and must be nominated by a sponsor. Sponsors must be members of the American Psychiatric Association. Members of the prize board are excluded from submitting nominations.

The sponsor should submit a supporting letter justifying in detail the nomination and summarizing the research accomplishments of the nominee.

The nominee should submit the following:

1. an up-to-date curriculum vitae
2. an up-to-date bibliography
3. a group of representative and thematically linked papers or books published (or accepted for publication) that are in English and dated within 10 years of the deadline for submission
4. a summary statement written by the nominee that emphasizes the principal theme (or themes) of the work and its internal cohesiveness and consistency

Six copies of each entry should be sent to Joel J. Elkes, M.D., Chairperson, Foundations' Fund Prize for Research in Psychiatry Board, American Psychiatric Association, 1700 Eighteenth Street, N.W., Washington, D.C. 20009. Entries will be acknowledged but cannot be returned. The deadline for submission is December 1, 1977. Any entry received after that date will be automatically entered for consideration for the 1979 award unless withdrawn by the nominator or candidate.

A Psychodynamically Oriented Group Training Program for Early Childhood Care Givers

BY STANLEY I. GREENSPAN, M.D., BARBARA SILVER, PH.D., AND MARTIN G. ALLEN, M.D.

A training program for enhancing the care-giving qualities of child-care workers was developed and implemented through semistructured groups for family day-care mothers. The authors describe the theoretical orientation, program goals, and the outcome for trainees and trainers. Most impressive was the group process and content, which indicated the effectiveness of a psychodynamically oriented program for care givers—people who ordinarily would not be expected to integrate such an experience successfully.

THIS PAPER describes a unique training program for family day-care mothers—a group of women who are becoming an increasingly important source of care givers for children. These women take care of a small group of other people's children, especially the children of working mothers. Of the six million children under six years of age who have working mothers (1), 31% are cared for in someone else's home (2).

Preliminary investigations have led to optimism about the quality of care in family day-care arrangements and the potential for further development of this resource (3–6). However, there is no direct means of learning about the quality of this care or the usefulness of training day-care mothers to facilitate the total development of the children because each care giver, in a sense, conducts a private business in her own home.

An experimental training program that employed a dynamic developmental perspective was conducted in an attempt to learn more about family day care and to enhance the capacities of family day-care workers. The purpose of this program was 1) to learn more about family day-care mothers through their participation in small, semistructured groups, and 2) to enrich their understanding of children, thereby perhaps changing some of their routine patterns of child care.

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The main goal of the training itself was to develop the capacity of the family day-care worker to facilitate the emotional growth of the children in her care. Specifically, the purpose of the training was to enhance the day-care mother's capacity to 1) perceive what the child is communicating (accurate perception of what the child is communicating will facilitate the development of his reality testing), 2) understand empathetically, but not overidentify with, the child's emotional experience (an adult's balanced understanding of what the child is feeling, as well as the child's own emotional experiences, enhances the child's sense of trust and security), 3) respond temporally in an effective way to the child's behavior (feedback to the child about his behavior is important in helping him establish a sense of inner control and internal boundaries), and 4) respond flexibly to the child's behaviors (flexible and sensitive differential responses to a child help develop a sensitive and differential repertoire of coping mechanisms).

A second goal of the program was to learn whether a relatively untrained person in the mental health field could, under professional supervision, conduct the training experiences for the family day-care workers. If so, the training program would have the potential for broader use and would be economically feasible.

Two groups were formed; each was led by a relatively untrained social service worker under supervision. Whereas training programs for such groups have usually been oriented toward didactic presentations, the present program was more psychodynamic in orientation. The focus was to be on understanding and openness in communication, discussion of child-care problems shared in common, and the expression of feelings. This method approaches the model of group therapy with day-care mothers verbalizing, sharing, and delineating the specific emotions and concerns that are involved in their understanding of and response to the children. The group leaders focused on both the content and the process of the group. The content provided useful examples of adult-child situations for discussion. The group process enabled the day-care mothers to explore together their mutual and individual concerns. The leaders were to highlight feelings, conflicts, defensive patterns, and alternate means of coping.

THE TRAINING GROUPS

The group members selected were the first 16 women who responded to a letter of invitation sent out by the local department of social services. (There were 35 responses to 200 invitations.) The letter stated only that there would be an opportunity to meet with other family day-care mothers for seven 1½-hour sessions to discuss their common concerns. Each group originally consisted of 8 women with socioeconomic status designations of lower middle-class and below. There were both black and white day-care mothers, and they ranged in age from 23 to 50 years. At the first session 1 group lost 2 women; 1 never came and 1 dropped out.

TRAINING AND SUPERVISION OF GROUP LEADERS

In the supervisory sessions the 2 leaders were instructed to 1) attempt to sensitively understand what was being said, 2) facilitate discussion of themes by rewording discussions, 3) pose questions that would further develop a theme, and 4) highlight the theme when it seemed to emerge only in fragments. In general, the leaders were to encourage members to speak out about what was on their minds and to promote development of these ideas. The leaders were not to direct or suggest issues for discussion.

In order to facilitate group discussion, a number of techniques were stressed. The leaders were instructed to move between process and content, look for affect within the group, stress the differences between stereotyped and flexible ways of dealing with stressful situations, move between the adult and child world, and move between relationships within and outside the group.

In addition to learning the principles of group leadership, both leaders were helped to understand and cope with their own emotional struggles. The main issues were their initial feelings of suspicion and helplessness, tendencies to deny or cover up their own emotions as stimulated by the groups, and to use certain defenses, the most prominent of which was boredom. A parallel was drawn between the feelings they experienced in relation to the group and the feelings the family day-care workers experienced in relation to the children they cared for. The supervisors held the view that this experience would be productive for all concerned.

One of the leaders began her experience with mixed feelings; she was suspicious of any approach that focused so substantially on emotions, denigrated the day-care workers, and felt bored. Yet, by the end of the program she felt a part of the group, looked up to some of the women as mother figures, and manifested a sincere, warm regard for them. The second leader initially had warm, positive feelings toward the people with whom she was to work. However, due to her personality, she was less able to tolerate expressions of emotion, attempted to keep the group focused on concrete issues, and often felt overwhelmed.

DEVELOPMENTAL SEQUENCE OF THE GROUP SESSIONS

Without the motivation of psychic pain or symptoms, a number of day-care mothers were able to form two training groups that developed features similar to those of short-term treatment groups; that is, from a period of initial reserve there evolved cohesion, identification, open expression and examination of feelings, capacity to deal with anger, and finally, the experience of the separation process. For the sake of brevity, we will provide only a combined summary of both groups' thematic progression.

The initial sessions dealt with a number of issues generally common to beginning groups, e.g., factual data regarding members' identities, what the tasks ought to be, and inquiries about the role of the group leaders. This exploratory phase quickly yielded to more specific discussions about the responsibilities of real versus day-care mothers, what characterizes a "bad" parent, and how one deals with "problem" children. The issue of problem children dominated the early sessions, with the day-care mothers citing many examples.

For instance, one woman described her problem with a passive, withdrawn, negativistic boy who clung to her and would not sleep at naptime. She was aware that the boy's father frequently beat him at bedtime, yet she did not know how to use this information to help the child. Instead, she found herself torn between being either overindulgent or minimally nurturing. At times she felt sorry for him and at other times angry because he was "such a pest." The group wisely advised her to provide the boy with alternative activities when he was demanding and to give him her full attention when she felt relaxed and could spare the time. Another day-care worker's problem with twin brothers who would not separate from each other and whose behavior made her "anxious" led to a discussion of how one stimulates a sense of trust in children when there appears to be little or none. At one session the issue of trust was discussed at such length as to imply that what was actually being dealt with indirectly was how to develop trust among the members of this new group.

What came through clearly from almost the first session was the annoyance the women experienced when their routines were inevitably interrupted. It was apparent that they were at times rigid and inflexible in many "problem" situations.

In the first two sessions the quality of interaction was one of polite acceptance of each other, with the expected initial emotional reserve. Surprisingly, there was also an acceptance of the leader's relatively restrained role of facilitator, rather than active intervenor. By the third and fourth sessions the facilitator

noted the growth of a melding process, which became more apparent at each session. As the members of both groups became more emotionally open, the depth of the content began to match this growing emotional accessibility. Talk focused on problem children, as well as the problems created for those who care for other people's children. For instance, many of the day-care mothers worried about what to do when a child was injured and how to deal with the natural mother. Should the caretaker act immediately without first contacting the parents and thus risk being sued, or should she delay until they could be notified? This very real problem highlighted an important affective concern for substitute mothers, that is, their relationships and feelings toward the natural mothers. Hints of competition and rivalry between care givers and real mothers emerged, as well as reaction formations and guilt feelings against these feelings.

During the first and last of the seven sessions, both groups completed a projective test that contained a number of items dealing with the vicissitudes of sexuality and aggression in children. Discussions about these items in the middle sessions led to some interesting exchanges, parts of which could only have been elicited from groups whose members were becoming more open and supportive toward each other. For example, they were able to talk about what constituted masturbation and to question its appropriateness in young children. When menstruation was discussed, the members felt able to express themselves freely enough to state beliefs such as that of one strong-minded woman who claimed that "male nosebleeds are the counterpart to menstruation and could lead to sterility." Another topic was the appropriate time for toilet training and some of the problems encountered during this process. A major concern centered on the discontinuity encountered between the care giver's own procedures and beliefs in dealing with children and those of the natural parents, and the effect on the children.

In one revealing and emotional session, a mother described a 5-year-old boy in her care as having "effeminine [sic] likes" and "wanting to pull down the pants of my young son." When she asked him what he was doing, he claimed to "want to put a stick in his rectum." With this disclosure the group's anxiety rose to a level that precluded much response from the other members. However, there was notable continuity between this meeting and the next, when the woman who at an earlier session had related her beliefs on nosebleeds and menstruation now offered her views on identifying the homosexual child. She stated emphatically that "a child should be turned upside down and if the child has a vagina, rectum, and penis, the child is a homosexual." This time the women were able to respond actively, despite the high level of anxiety that surfaced. Some agreed with the "theory" presented, while others found it amusing, although their amusement had a nervous quality to it. The ensuing discussion permitted an exploration of both the facts regarding children's sexuality and the feelings that children's sexuality generates in adults. The spectrum of adult defenses, from denial to exaggerated interest, was exposed.

There was another interesting development at the same time. The women who had related some of their experiences with problem children in earlier sessions said that these children now seemed to be less difficult. For example, the twins were playing with other children, and the clinging, withdrawn, negativistic boy was less tense, more outgoing, and more flexible. However, the women were unsure as to why the children were doing better. There were also references to their own decreased anxiety and the helpfulness of the groups' suggestions.

Although the groups were racially mixed, the issue of race did not surface until the middle session. A black woman, in reporting that her son's jacket had been stolen in school, implied that the theft had racial connotations. The women then shared their personal experiences with racism, although at no time was the racial composition of the groups mentioned directly. Most of these women took care of a racially mixed group of children, and they talked about the stresses and rivalries among the children, but also some of the benefits to themselves and the children in a changing society.

At one point during these same sessions a woman became openly irrational, voicing suspicions about the social service system, her inability to trust a neighbor, and her belief that at times people followed her and were "after her." She attempted to dominate the group by yelling at some of the other members. The women responded to all of this reassuringly, for the most part minimizing the woman's irrationality and at times even ignoring it. It is important to note that there was a sense of comfort and security in the group as a whole and that the group members did not become fragmented by this disruptive behavior. The following week the woman appeared more intact, and she spoke about her concerns in a more reasonable fashion. There were no signs of the suspiciousness or open hostility and anger of the previous session. In talking about herself, she claimed that for some reason she was feeling more relaxed than she had the week before.

With only two sessions remaining, separation concerns emerged in one group. One of the women told of a natural mother who did not want to separate from her daughter when the child was brought to the care giver's home. A discussion ensued about the effects on children of separation from their families and the effects of separating a child from his or her mother. The women talked about becoming attached to the children in their care and their feelings at having to separate from them. This exchange appeared to result in the development of a feeling of empathy for the natural mothers, as well as a useful exploration of the women's own feelings of attachment, dependency, and separation.

Issues of separation and termination were dealt with

formally in the final session. The members of both groups expressed their high regard for the leaders, and they agreed that the experience had been very useful. There were also covert indications of anger toward authority figures and talk about doctors who are not always helpful. Both groups showed resistance to completing the projective test again, although most finally agreed to do so. However, two women staunchly refused to comply, and the groups supported their continued resistance. Many of the participants indicated their wish to meet on their own, and they talked about sharing equipment and joining together for trips. Much care was taken to exchange telephone numbers, and there were many promises to keep in touch with each other.

ASSESSMENT OF THE GROUP EXPERIENCE

The following four measures were used to assess the groups' functioning: analysis of the groups by the supervisors, subjective assessment of the group members by the group leaders, self-assessment by the day-care mothers, and assessment by the use of a projective test developed specifically for this project (7).

When the two group leaders were asked to rate the women on a scale from 0–5 in relation to the degree of positive change, they gave 9 of the 14 women scores higher than 3.

The self-ratings of the group members were positive. They said that the group experience resulted in the "improvement of problem kids," "the exchange of ideas," and "new ways to help kids," and they referred to a sense of group identity. While their positive reports were subjective, they were interesting since traditionally this population responds to "training" with apathy.

Recorded in terms of pregroup and postgroup experience, the individual response pairs on the projective test items were rated equal 57% of the time (that is, there were no qualitative differences in the response before and after the group sessions). When the pairs were rated unequal, the postgroup responses were rated better in a ratio of 2 to 1.

DISCUSSION

Three findings need to be emphasized. The first was the day-care mothers' wish for help and their reports that they had benefited from the group and that there were positive changes in some of the children for whom they cared. Often, however, it was not clear what aspect of the group experience had been helpful. It was felt that the day-care mothers' guilt, anger, or feelings of helplessness about a child's negative behavior inadvertently reinforced the behavior. This inadvertent reinforcement occurred because feelings of anxiety or helplessness led the care giver to respond in a stereotypic or insensitive manner, which, in turn, further increased the child's frustration, provided attention for his maladaptive behavior, or set up expectations for the continuation of the behavior. Verbalizing her feelings and being in a setting where her concerns could be aired decreased the day-care mother's anxiety and permitted her to think and respond more flexibly and autonomously. Although there were times when specific recommendations were helpful, these incidents did not appear to be as impressive as the nonspecific group effect.

The second finding related to the day-care mothers' openness and ability to use a semistructured setting. Discussions of anger, dependency, sexuality, and separation highlighted the usefulness of a semistructured, psychodynamically oriented group setting. It is questionable whether such material would have emerged and been worked with in a more structured setting. Verbalization of this material permitted factual correction when appropriate. More important, shared concerns and anxiety could be usefully explored and a general tolerance for "feelings" and capacity for balanced empathy reinforced.

The third finding was the groups' cohesiveness. Wishes for communal trips, shared equipment, and so forth indicated a deep level of a sense of belonging that cannot be underestimated in jobs where adult-adult contact is minimal. In this context it is important to note the "process" that occurred in the groups; some initial anxiety and distrust gave way to a series of sessions in which primitive and affectively laden material was worked on. Subsequently, there was an indication of some aspects of a termination process.

The high degree of motivation of the family day-care mothers was an impressive finding. Usually, when there is no obvious extrinsic motivation (e.g., financial gain), voluntary participation by members of lower and lower middle class socioeconomic groups is poor. The importance of mobilizing interest and curiosity rather than passivity in any type of training program cannot be overemphasized.

An additional outcome was the indication that under supervision, people relatively untrained in mental health can lead such groups. The use of group leaders who had not had formal mental health training has important financial implications in making such programs feasible. However, it is necessary to be selective about the characteristics that group leaders should possess, e.g., openness, warmth, and a reasonable level of psychological maturity.

Some liabilities of this type of group experience should also be considered. In a relatively unstructured group setting there is the possibility that a high degree of stress may be created in the group leaders and members. In these training programs one group leader who was under much personal pressure experienced an added strain in leading a group.

Although none of the day-care mothers in the present study appeared unduly stressed, the effects of verbal attacks or regressive emotional experiences must be considered. Members' ego strengths, skills of the

group facilitators, and the quality of supervision all play a part in determining to what extent certain group experiences will have deleterious or positive results.

In summary, this report has been an attempt to develop a picture of what these groups were like for the family day-care mothers and the group leaders and of how the training experience was used. It is hoped that additional investigations will continue to explore this area.

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Anger and Fear

BY HOSSAIN B. DANESH, M.D.

The author proposes that fear and anger are innate defensive mechanisms mobilized by perceived threats to the individual. Both mechanisms are accompanied by anxiety and an urge to escape or attack. The individual's lifestyle, which is the result of interaction between his innate qualities and life experiences, determines whether he will attempt to deal with threats, his fear and/or anger, and the resulting anxiety in an objective or pathological manner. This formulation underscores the need to learn methods of coping with anger and fear from early childhood, so that they do not develop into pathological behavior patterns.

WE ALL get angry, but our understanding of the nature of anger differs considerably. The argument about the causes and effects of anger ironically finds its expression in the psychiatric literature through an avoidance of the term itself. Anger usually appears in camouflage under definitions such as aggression, violence, and hostility, or else is referred to almost casually and in passing, as though it is too simplistic for consideration. In a world in which "aggression" has become common property, in which "violence" is now a cliché, and in which "hostility" has been "cosmeticized" in entertainment and institutionalized through sports, it might be useful to approach the concept of anger and its influence upon us.

While we have all experienced the simple state of anger, it is clear from our clinical observations in psychiatry that the condition, Janus-like, contains a paradox. For although psychiatrists avoid the term itself in the psychiatric literature, they have succeeded in unearthing a cluster of avoidance techniques by which the phenomenon of anger manifests itself in the form of psychosomatic disorders, depression, destructive behavior including suicide and homicide, and interpersonal as well as family difficulties. Therefore if anger is not expressed and dealt with, it undergoes a subtle change and can subvert the creative functions of a human being not only through violence and hostility but through self-destructive and self-limiting conditions as well.

In order to review the interrelationship between these polarities and thereby reach a fresh definition of the phenomenon of anger, in this paper I will consider the major prevailing theories of aggression and will attempt to arrive at a pattern within which the fluctuating manifestations of anger, fear, anxiety, and aggression can be understood.

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BACKGROUND THEORIES

Aggression and Violence

In the early decades of this century (whose wars have made anything experienced by previous decades seem insignificant), Freud (1) postulated that man was a creature at the mercy of his instincts, which were essentially of two kinds: life and death, love and hate, the sexual and the aggressive instincts. The former he named "eros" and the latter he described as "the aggressive instinct whose aim is destruction." Elaborating on this theory, he wrote that human instincts are "only of two kinds: those which seek to preserve and unite . . . and those which seek to destroy and kill."

The application of this theory finds itself aptly reflected in a scrap of significant correspondence between Freud and Einstein (2). The latter wrote to Freud on July 30, 1932, upon the invitation of the League of Nations and its International Institute of Intellectual Co-operation in Paris. His hope was for a "frank exchange of views" on the question that he considered to be "the most insistent of all the problems civilization has to face" and which was simply, "Is there any way of delivering mankind from the menace of war?"

Freud's reply was characteristic. He expostulated against the unscientific nature of the question; he said that he had expected

> a problem on the frontiers of what is knowable today, a problem to which each of us, a physicist and a psychologist, might have our own particular angle of approach. . . . [But] You have taken me by surprise . . . for the question such as it is can only have one possible answer. It is a general principle [he stated, thereby dealing a death blow to the League of Nations and many a failing mind since then] that conflicts of interest between men are settled by the use of violence. (2, pp. 203–204)

The discouraging view of aggression that Freud cast upon psychological thought is still less pessimistic than that of Lorenz and other ethologists. Gorney (3), in a sensitive paper, compared the theories put forward by Freud and Lorenz and stated that the former

"allowed for the possibility that if one could shut off aggression (and violence) at the source, love would still exist." The latter forced the logic to its most bitter conclusion and "carried the instinctual conception of aggression a dismal step further toward hopelessness. Because love was to him [Lorenz] nothing but made-over aggression, elimination of aggression would also eradicate love" (p. 437).

The subtle and dangerous appeal of these theories, which allow man to justify his violence, his lust for power, and his crimes and prejudices, has created a richly divergent atmosphere for discussion. A number of studies now provide convincing evidence that aggression is in fact a learned defensive behavior against threats. In *The Anatomy of Human Destructiveness* (4) Fromm reviewed the evidence against the instinctivist thesis and reached the conclusion that data from the neurosciences, animal behavior studies, and the fields of paleontology and anthropology indicate that the instinctivistic interpretation of human destructiveness is not tenable. Fromm stated that

> while we find in all cultures that men defend themselves against vital threats by fighting (or by fleeing), destructiveness and cruelty are minimal in so many societies that these great differences could not be explained if we were dealing with an "innate" passion. Furthermore, the fact that the least civilized societies like the hunter-gatherers and early agriculturalists show less destructiveness than the more developed ones, speaks against the idea that destructiveness is part of human "nature." (4, pp. 204–205)

Eron and associates (5) have determined, through a 10-year longitudinal study on the relationship between learning conditions in early childhood and aggressive behavior among adolescents, that such behavior is "shaped by learning through socialization practices and varies by sex." They further stated that their findings detract from "the theories of the ethologists who argue that aggression in man is innately determined."

In reviewing the literature one soon becomes aware that when different investigators and authors use the term "aggression," they are not speaking about the same phenomenon. In fact, all of the theories suffer from lack of a clear definition of "aggression" and its distinction from "anger." Furthermore, even though the role of fear and anxiety in this complex of behavior and feelings is acknowledged, the nature of this interrelationship is by no means defined.

Anxiety and Fear

The feeling of anxiety is a familiar one. We experience anxiety under varied circumstances and conditions. At times we are able to locate the source of our anxiety, while at other times we find it very difficult to do so. Fear is an experience similar to that of anxiety. However, in the psychiatric literature a distinction is made between fear and anxiety. Whereas the former seizes upon us simultaneously with an apprehension of known danger and is a direct result of our knowledge of its source, the latter occurs with less clearly conceived synchronization and in the absence of the focus of the danger's source. In an analysis of this distinction Lief (6) wrote,

> Anxiety is a diffuse, highly unpleasant, often vague feeling of apprehension, accompanied by one or more bodily sensations that characteristically recur for the same person. . . . [It] is an alerting signal; it warns of impending danger and enables the person to take measures to deal with a threat. Fear, a similar alerting signal, is ordinarily differentiated from anxiety on the basis of whether the threat is known or unknown, external or internal, immediate or future, definite or vague, nonconflictual or conflictual in origin. (p. 857)

The realities that underlie these distinctions have become part of the popular psychological mythology of our century. The stresses of modern urban life, the pace of industrialization and rapid change, and the increasingly alienated and declining position of the individual in this complexity all combine in the shape of threats, responsibilities, and actual dangers that engulf him; they have all been mentioned at one time or another as causes of the anxiety of modern man. This view is similar to psychoanalytic thought, which postulates that anxiety is a reflection of the existence or anticipation of danger within the human being, which can severely paralyze his proper functioning, lead him to the point of inactivity, and reduce him to a state of near helplessness.

On the other hand, however, some degree of anxiety is essential for the individual's safety and growth. Without anxiety there is little motivation for protecting oneself or improving one's condition in life. At this point we reach the final link in psychiatric thought between anger and anxiety. Rothenberg (7), in his studies on this subject, arrived at the conclusion that anxiety and anger are closely related, since both are the organism's response to threat and danger. He stated

> On the basis of all that is currently known about anger and anxiety, both clinically and experimentally, it seems likely that both of these phenomena are aspects of a diffuse alerted and aroused state. Anger becomes the predominant manifestation of this state when the motoric arousal begins to be directed at the source of threat or obstruction or at an imagined source. Anxiety is the predominant manifestation when the motoric arousal is undirected or is directed toward avoidance or escape. (p. 458)

While these observations are all insightful, they are nevertheless subject to the same dilemma and often seem to be diffuse and directed toward an avoidance of the definition of anger. Rothenberg's most definitive statements, that "anger . . . is always accompanied by anxiety" and that "neither of these manifestations ever seems to occur exclusive of the other" (p. 458) seems at best to invite the mind to further exploration of the relationship between the two. We have yet to arbitrate between normal and abnormal anxiety and to assess the role of anxiety in defending the organism. We need to clarify the relationship among anxiety,

fear, and aggression and to understand the position that anger holds in this equation.

UNDERSTANDING THE PATTERN OF ANGER

The overall pattern with which one must begin the close analysis of anger resides in the primary assumption that life, characteristically and continuously, exposes the individual to threats and opportunities.

Threats can be either actual or assumed. They can originate from the external environment and assume the form of physical or social threat, or they can rise like mist from within and take on the shape of psychological or spiritual crisis. The individual can meet these dangers head-on and with full consciousness, or they can creep up on him gradually and in a disguised form. Some perceive these threats in a realistic manner and respond to them accordingly. The alternative is either to underestimate the intensity of the danger and subject oneself to injuries and afflictions, or to go overboard and respond to the slightest challenge as though it were, indeed, a fearful and destructive danger. Our perception of the danger situation is closely related to our upbringing and our endowments. Once the threat is felt, two major defensive mechanisms are put into motion. They are immediate and automatic—they are innate defensive mechanisms. However, depending on the nature of the individual's life experiences and endowments, one or the other takes a more predominant position.

These defensive mechanisms are fear and anger. A close analysis of these twin mechanisms shows that they both consist of three stages, the first two of which are identical in both. The first component is the alert signal, which informs the individual of the presence or possibility of threat. The second is a condition of anxiety that mobilizes one's needed defensive energy. The third, in the case of fear, consists of an urge for escape or withdrawal, and in the case of anger, involves a desire to attack and thereby eliminate the source of threat. The presence of anxiety, in conjunction with fear and anger, has been observed in both research and clinical work, as Gellhorn (8) has attested. According to Gellhorn, "Chronic anxiety . . . is due to the simultaneous activation of the ergotropic and trophotropic systems at a high level of arousal or psychologically speaking to fear and aggression." The use of the term "aggression" in this context is unfortunate, because Gellhorn was speaking of two emotions, fear and anger. Therefore, when the individual is threatened, defensive mechanisms of fear and anger are mobilized, and both are accompanied by anxiety and an urge to escape or fight.

During infancy and early childhood it is difficult to distinguish between the two responses of fear and anger, since the individual responds with both indiscriminately. As he matures, accumulates experience, and learns from models in his life, one or the other of these defensive mechanisms becomes the primary mode of his self-defense. However, this does not automatically exclude the other defensive response, and indeed, throughout life we respond to threats, to a lesser or greater degree, with both fear and anger. Besides, whenever we respond with either fear or anger the other response is also present, although in a less obvious manner.

These two defensive responses are in the realm of emotions. In this phase the individual has not yet committed himself to any action (behavior).

Following this phase comes the phase of solution. Here the individual has two basic choices. The first is the "objective-creative" solution, which consists of a realistic evaluation of the threat and a unique response to it. During this process the individual assesses the intensity and relative significance of a threat and estimates his own capacity to deal with it. Consequently, he comes to a decision either to carry out his urge to escape or to stand in his tracks and attack the source of danger. In either case, within this "objective" framework, both the fear and anger responses are utilized for the purpose of self-preservation, and as such can be construed as constructive acts.

The other impressive characteristic of the objective-creative solution lies in its essential creativity. An individual, by approaching the threat in a state of full consciousness, draws upon all of his resources, the accumulated force of his inherited endowments and the vast reservoir of his particular life experiences. The final result is necessarily unique and explains why human beings are capable of a multitude of reactions to threats and dangers, all of which are healthy although differing widely in expression. The existence of this creative impulse and its role in the development of an individual's life expression was identified by Adler (9), who stressed, "Do not forget the most important fact that not heredity, and not environment are determining factors—both are giving only the frame and influences which are answered by the individual in regard to his styled creative powers."

The second, or "pathological," solution has its roots in the faulty perception of ourselves and our world. Our personalities, turning on the axis of heredity and environment, set into motion a predictable ebb and flow of anger and fear, with the anxiety that accompanies them. If our models and experiences have adopted the mechanism of anger in preference to fear, the pathological solution manifests itself in the form of aggressive behavior. The divergence between anger, which is not an act, and aggression, which is a learned behavior, becomes clear at this point. Whereas anger is an emotional defense that rises to protect the individual's integrity and does not involve a destructive element, aggression is a negative implementation of that defense which is shaped by specific experiences of the individual and is a learned behavior quite distinct from his integrity.

In the case of fear the individual's pathological behavior manifests itself in avoidance techniques or apathy. Avoidance techniques include all such behav-

iors that allow the individual to underestimate his own capacities, overestimate the intensity and extent of the threats, or both. Individuals with such a lifestyle are fearful, uncertain, apologetic, sickly, and shy. They avoid opportunities and live a life of isolation, avoidance, and emptiness.

Apathy is still another pathological solution used by many in the face of threats. Here the individual, for all practical purposes, avoids solution, abandons hope, and denies possessing inherent capacities and qualities that are obvious to the objective observer.

CONCLUSIONS

The formulation presented here has identified three phases in the development of our response to challenges of life—the threat phase, the innate defense response, and the third phase, which consists of the learned behavior used by the individual as a solution to the threats.

The threats are either from within or without and can be actual or assumed. The response to these threats is in the form of fear and anger, with one or the other taking prominence. Both of these responses are accompanied by chronic or acute anxiety and the urge for escape or attack. The individual's lifestyle, which is the result of interaction between his innate qualities and his life experiences, determines whether he will attempt to deal with these threats, his fear and/or anger, and the resulting anxiety in an objective and creative manner or in a pathological form.

By attempting this juxtaposition between the objective-creative and the pathological solutions to the forces of anger and fear (which arise from the invariable threats that an individual faces during his lifetime), a number of salient points emerge. Among these are the number and types of threats that produce the variations of anger and fear and the necessary mechanisms to decrease and, if possible, eradicate these threats. Furthermore, by differentiating between emotions and behavior, the innate and life-preserving qualities of normal fear, anger, and anxiety are sharply defined and separated from the pathological, life-threatening, and learned behavior of violence and apathy.

Finally, this formulation underscores the need for learning methods of coping with anger and fear from early childhood, so that they do not turn into pathological behavior patterns. In order to arrive at such objectivity, one must first approach the definition of "anger" with an uncluttered mind. Until then, the pathological solutions are inherent in one's interpretation of the word itself.

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Episodic Dyscontrol and Family Dynamics

BY HENRY T. HARBIN, M.D.

The author describes characteristics of patients, primarily adolescents, who suffer from episodic violent behavior and their relations with their families. Typical family patterns include overly close alliances by the adolescent with one or both parents and transmission by parents of inconsistent values regarding aggression. Family therapy is the preferred treatment approach; treatment should be active, should initially focus on ways of handling dyscontrol episodes, and should emphasize that the patient is responsible for his or her actions.

THIS PAPER will discuss clinical conceptions of and treatment techniques for patients and families whose predominant symptom is periodic violent outbursts. The sociological and psychological literature documents the high incidence of violence among family members or between intimates. Goode found that at least 50% of homicides were committed by family members or lovers (1). The finding that one-fifth of all police fatalities occur while the officer is intervening in a family argument also points to the need for increased understanding of intrafamilial violence (1). Another statistic that points out the disturbing amount of violence in the family is the increasing incidence of child abuse: some authors have estimated that there are 500,000 to 2.5 million cases per year (2). In addition, juvenile violence is increasing in frequency every year on a national basis. It has been noted that more individuals who commit the four major violent crimes (homicide, rape, assault, and robbery) fall into the age groups 15–17 and 18–24 than into any other age group (3). Several researchers and reviewers of the literature have pointed out the paucity of systematic research into the family interactions of violent individuals (2, 4). These statistics and findings highlight the need to clarify more fully the familial dynamics that may foster repetitive violent episodes.

This paper will present characteristics of patients with episodic dyscontrol who were seen at the University of Maryland Institute of Psychiatry and Human Behavior. The majority of these patients were adolescents treated with family therapy. In addition to violent outbursts, most of these patients had episodes of suicidal behavior. Most of the identified patients had EEG abnormalities (temporal lobe spikes and 6-14/second spikes), and some showed evidence of soft neurological signs. Some patients took anticonvulsant medication or a tranquilizer. (It should be pointed out that, although most of the patients in this sample had some organic involvement, most patients with problems of violence do not.)

Violent behavior consisted of aggression directed at inanimate objects such as windows or furniture, aggression toward other people, sometimes with intent to kill, dangerous misuse of automobiles, and repetitive violence when drinking alcohol. Suicidal behavior sometimes occurred in conjunction with aggressive outbursts. For example, one patient put his hand through a window in an attempt to cut his wrist while he was angrily breaking up furniture.

A family therapy approach to violent behavior emphasizes the communicational meaning of the assaultive behavior and attempts to change the interactional patterns that maintain this dysfunctional symptom. Organic instability increases the vulnerability of the adolescent or young person to a multiplicity of family and developmental pressures, some of which may be of sufficient magnitude to lead to a dyscontrol episode. However, once the cycle of violence is set in motion, these episodes may take on an adaptive function for the family. The dyscontrol episodes may serve as the final common pathway for other family members' stresses, may be an attention-seeking mechanism for an individual, or may serve a protective function by shifting the focus away from covert marital problems. It is important to evaluate and weigh the overall mixture of intrapsychic, interpersonal, and organic variables that influence dyscontrol episodes before deciding on a particular treatment approach.

TYPICAL FAMILY PATTERNS

Overly Close Alliances

In reviewing cases treated in our hospital as well as families that were only evaluated, I noticed certain repetitive family interaction patterns. A violent adolescent or young adult frequently has a close, almost symbiotic, emotional bonding with one parent; the other parent is usually only indirectly or ineffectually involved. Covert and overt marital disturbances are almost always present with this constellation. Of course, one of the major developmental tasks for the symptomatic young person is separation from the family and increasing autonomy from parents. The parents them-

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selves are usually struggling with middle-age life crises, declining health, and identity confusion; these issues are intertwined with the difficulties of the adolescent. The natural ambivalence of the young person while separating will be exaggerated due to the overly close relationship with one parent and will swing from intense hostility to equally intense guilt and shame. The adolescent's attempts at separation invariably cause stress for the parents and siblings as their dependency on the adolescent is disrupted.

The following case example shows the symbiotic relationship between a violent male adolescent and his mother. Therapy with this family continued over a period of 6 months; at the end of this time the therapeutic goals of cessation of violent outbursts, increased autonomy for the adolescent, and reinvolvement of the father with the family had been successfully reached.

A, a 17-year-old male, was referred because of several episodes of violent behavior. His mother and father participated in outpatient therapy. Both parents were frightened and concerned about what was happening with their son. The mother was the dominant figure in the sessions, and her questions and actions relating to her son were very intrusive. The father was somewhat passive and distant and seemed too timid to intervene in child-rearing matters, thereby abdicating his role. A was particularly confused about his life goals and would swing from wanting to live at home to wanting to elope with his girl friend. His first violent outburst occurred while he was with his girl friend and was fantasizing about how much money he owed his mother and how he would never be able to pay off his debts to her. He became extremely anxious and began to smash chairs, threatened to hit his girl friend, and finally broke a window with his hands. It required several people to control A, and he was hospitalized. He had only a sketchy memory and awareness of the events during his rage reaction.

It was subsequently determined that A had 6-14/second EEG spikes and he was placed on diphenylhydantoin. He was certainly in a situation in which his loyalties were split between his girl friend, who was as domineering as his mother, and his mother, to whom he owed a much greater emotional debt. He seemed to be torn between the alternatives of either destroying himself or someone else as a response to this dilemma.

The following case also shows an overly close relationship between parent and child, but this time both parents are involved.

In this family the symptomatic person was the 19-year-old son, B, who had periodic outbursts of violence followed by suicidal behavior such as head banging and attempts at hanging himself while under the influence of alcohol. B also drove his automobile very recklessly while drinking and had had several accidents. He was involved in a very hostile, dependent relationship with his mother and a more positive but symbiotic relationship with his father. The father was extremely dependent on his son and had great difficulty in setting limits for fear of hurting him or driving him away. The mother, however, was extremely hostile and rejecting at times. The marital relationship was chaotic, and there was a great deal of emotional distance between the spouses. B was unable to separate from home even though he had stated often that this was his only wish. Whenever he became angry with his parents for setting limits or disagreeing with him he would leave the house and get drunk; sometimes he became involved in provocative or assaultive actions with police when they attempted to arrest him or stop him for misusing his car. B had some evidence of brain dysfunction on EEG tracings.

In these examples and others, one sees the adolescent's alternations between extreme hostility toward self and others and extreme (mutual) dependency with the parents. Steirling points out that guilt over leaving a home that is overly close can cause an adolescent to be overcome by self-destructiveness (5). The psychopathology of the parents, the inability of the parents to negotiate their marital problems, the young person's confusion and fear about separating from home, the instability of the young person's central nervous system, and the poor self-esteem of both parents and child all add together to create a milieu in which episodes of behavioral dyscontrol can take place.

Distortions in Value Transmission

Another frequently repeated pattern in these families is inconsistency in the values and limits concerning aggression transmitted by the parents to the adolescent or young adult. The parents may react very angrily to violent behavior at one time and then disqualify this limit by ignoring the same misbehavior another time. One parent may punish a certain behavior while the other parent subtly encourages it so that the child is caught in a covert conflict between the spouses. Other parents react to extremely violent behavior with a bland affect and seem afraid even to discuss punishments for these difficulties. This inconsistency in limit setting has been described in other families with delinquent adolescents; however, when delinquent behavior is violent, this inability to set limits can be an extremely grave and dangerous problem. The following clinical example demonstrates the problem of parental inconsistency and how it can affect the offspring.

A 17-year-old girl and her family were referred because she had set two fires in an attempt to kill both her family and herself. During the first interview the parents seemed unable to hold their daughter accountable for her extremely destructive behavior. They seemed fearful of confronting her, as if they could not bring themselves to face the fact that their daughter had tried to murder them. Even when the therapist attempted to interject some of the missing affect and dangerousness of the situation, the mother responded that perhaps they should forget about the past and try to start over again. The 15-year-old brother of this adolescent girl also had frequent violent "accidents"; once he had cut his younger sister's lip. Even when he slugged his sister in the stomach at a family party and knocked her down, the parents did not see the need to punish him. This lack of parental follow-through of punishment was mixed with frequent verbal diatribes about the evils of disrespect and hostility. This inconsistency between words and behavior made it difficult for the children in this family to know where the limits were for their own drives, particularly their aggressive urges.

Many parents of children who are violent seem almost immobilized by guilt over their own seeming failure as parents. They feel that they are at fault for not having prevented the symptom through better parenting in the past. They feel that they have no right to be the authority in the present because of their past failures. The adolescent or young adult may exploit this shame by accusing the parents of past lapses in proper limit setting. The therapist may also unwittingly exacerbate this guilt by asking questions about the past and either clarifying or bringing into awareness mistakes that the parents made.

Another element that contributes to inconsistency in the transmission of values is the parents' fear of the young person's violence. This fearful reaction and consequent withdrawal by the parents from confronting the symptomatic behavior is most evident in one-parent families. The following examples show how this fear affected two patients whose fathers were not in the home and who lived with their mothers.

A rather large, heavyset 27-year-old man was living with his mother and two sisters. He had had several episodes of intrafamilial violence while under the influence of alcohol. He had hit and kicked both his mother and sisters over a 2- to 3-year period. The family sessions revealed that they had never even confronted this man with his behavior, largely because of their fear of him. In fact, the patient had not known about the extent of his violent actions because he had been drunk and his family had never told him. He responded with a great deal of shame to this confrontation and renewed his efforts to control his drinking.

A 19-year-old boy had repeatedly attacked his mother and sisters in the home. Twice the mother had to go to the hospital for treatment of injuries inflicted by the patient. However, she had never sought outside help in controlling her violent son. In fact, during one incident the patient himself called the police to come arrest him.

PSYCHOTHERAPEUTIC CONSIDERATIONS

Before deciding on a treatment approach, it is necessary for the therapist to thoroughly evaluate the extent and nature of the violent actions and the degree of dangerousness, as well as to consider the necessity of a neurological workup. It is important to evaluate the family dynamics as well as the individual's problems. In my experience, a family therapeutic approach provides much more leverage than individual therapy because it makes the patient's most important interpersonal environment accessible to the therapist. Family therapy is particularly indicated when the aggressive act is directed toward another family member, unless, of course, the violent actions are of such magnitude that the person must be hospitalized or jailed.

In general, the therapist must be active and directive in attempts to change the violent symptoms. Haley (6) and Minuchin (7) have described detailed strategies for actively changing behavioral symptoms via a shift in the family structure. One of the most immediate and primary interventions that a therapist should make with these families is to clarify and systematize the steps to be taken if another violent episode occurs. This therapeutic action will accomplish several goals: 1) it will decrease the parents' or other family members' fear because they will have a more clear and definite plan of action; 2) it will push the parents to confront a young person or adolescent who is acting out aggressive urges, thereby encouraging more open communication between them; 3) it will push the parents to be more consistent and specific in their limits with the adolescent, thereby leading to a clarification of the usually blurred generational boundaries; and 4) it will decrease the symptomatic person's fears of losing control by showing him that he can be managed by others in the immediate environment.

The parents need to be told that they should call the police, if necessary, and that the young person can be taken to an emergency room or even to a correctional institution if he or she is completely out of control. Certain available members of the extended family or neighbors may be identified as persons who can be called upon to intervene in an emergency. The parents need the support of the therapist as an authority in order to help them reestablish their own authority over their offspring.

These actions by the therapist should be carried out during the first one or two sessions, particularly if the violence is life threatening. If a dyscontrol episode occurs during therapy the family's handling of the matter should be examined in great detail, and any inconsistency or confusion should be remedied. After clarification of behavioral limits has been accomplished, the therapist can work on other underlying problems in the family, such as overly close alliances between parents and children, marital dysfunctions, or covert depression and suicidal thoughts in family members. It is important to keep in mind that whenever a sensitive problem for the family is being worked through, the potential for violence from the identified patient will increase.

When the symptomatic adolescent is diagnosed as having a seizure disorder and is placed on medication, family psychotherapy becomes more complicated. The diagnosis may intensify an already-existing belief on the part of the parents that the adolescent is not accountable for violent behavior because he is physically "ill" and, therefore, not responsible for anything that happens. This may make it even harder for the young person to disentangle himself from the family. The therapist can help by pointing out that seizures of any kind are triggered by emotional conflicts and stress and that everyone in the family can work on coping with this stress in ways that will decrease the likelihood of a violent episode. A further explanation is also necessary; the adolescent or young adult must be told that he is responsible for whatever physical or personal damage is done during a dyscontrol episode, regardless of whether there is a partial organic etiol-

ogy. These dynamics were evident in the case of A, mentioned above.

During family therapy A had several more dyscontrol episodes. In one of these the parents called the police and neighbors to control their son and had him taken to the emergency room. The father then cleaned up the mess and repaired the broken furniture for his son. The therapist asked the parents not to do this in the future and had them clarify with the son that he was responsible for his behavior. At first A became angry and said that he had a medical problem and could not be responsible for his behavior. The therapist replied that it was true that he had a medical problem but that there was no one else to hold responsible for his behavior but himself and that this is what would happen. The next weekend A had one more violent episode, but this time he had to repair all the furniture and pay for the damage out of his own pocket. He did not like this, but he did not have any further dyscontrol episodes.

Another common problem in treating these families is the emotional reaction of the therapist. Violent individuals can stir up fear or intense anger in the therapist that can lead to countertherapeutic actions. Lion and Pasternak (8) described in some detail the countertransference reactions involved in treating violent patients in individual therapy. One of the greatest dangers for the family therapist is that his emotional reactions are likely to be the same as those of the family members; this may lead to an intensification of conflict on both sides. The family therapist is not only reacting emotionally to the identified violent patient but also to the anxiety or denial of anxiety of the other family members. The therapist may withdraw and become paralyzed by fear that he is going to be attacked, just as some parents do, and thus be unable to effect positive action. He may react by scapegoating the symptomatic member and ignoring family pathology, thereby further isolating the violent person. It is important to have colleagues to consult with when treating these families in order to maintain a sense of objectivity and neutrality, which can be easily lost in the face of such disturbing behavior.

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Plasma Testosterone in Homosexual and Heterosexual Women

BY NANETTE K. GARTRELL, M.D., D. LYNN LORIAUX, M.D., PH.D., AND THOMAS N. CHASE, M.D.

The authors measured plasma testosterone levels in 21 homosexual women and 19 age-matched heterosexual women. Although values for the two groups overlapped considerably, testosterone concentrations averaged 38% higher in homosexual than in heterosexual subjects. A statistically significant inverse correlation between age and testosterone level was found for heterosexual but not for homosexual women.

THE DEVELOPMENT of improved techniques to measure hormone levels in plasma has stimulated a broad reevaluation of the possible endocrinologic basis of human sexual function. For example, recent studies have shown that plasma testosterone concentrations in men correlate with sexual activity, although mean circulating levels of the hormone in practicing homosexual and heterosexual men are no different (1–7). The possible influence of testosterone on female sexual behavior has yet to receive as much investigative attention; however, previous observations of urinary excretion patterns suggest that testosterone concentrations in homosexual women may exceed those in heterosexual women (8–10). In this study, we report that plasma testosterone levels in homosexual women, measured by radioimmunoassay (11), appear substantially higher than those of heterosexual women of the same age.

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METHOD

Twenty-one homosexual women (referred by local homophile organizations) were age matched with 19 heterosexual women; all had volunteered to participate in this study after full discussion of its purposes, procedures, and potential risks. The age range of the homosexual women was 21–35 years (mean=26.8±0.80) and that of the heterosexual women was 21–33 years (mean=26.0±0.74). Sexual practices of study subjects during the preceding year involved only individuals of the same sex (for homosexuals) or opposite sex (for heterosexuals). None of the individuals studied were taking birth control pills or other steroid preparations or using marijuana frequently. All were found to be in good general health, nulliparous, and free of menstrual irregularities, hirsutism, or abnormalities of the external genitalia. Each underwent venipuncture at 8 a.m. on the first, second, and third days of her menstrual period. Day one was defined as commencing on that 8 a.m. which occurred within 24 hours of menstrual onset. Plasma separated from the resulting samples was stored at 0°C until testosterone assays were carried out. Plasma testosterone was measured by a modification of the method of Nieschlag and associates (11) with LH-20 Sephadex as the chromatographic medium (12). Reported testosterone values are average levels for the three-day specimens, corrected for percent recovery, which ranged from 60% to 90%.

RESULTS

As shown in figure 1, testosterone values for the heterosexual women ranged from 16–39 ng/100 ml (mean=26±7 ng/100 ml), and values for the homosexual women ranged from 24–56 ng/100 ml (mean=36±11 ng/100 ml). Thus, although ranges for

FIGURE 1
Plasma Testosterone Levels in Homosexual and Heterosexual Women

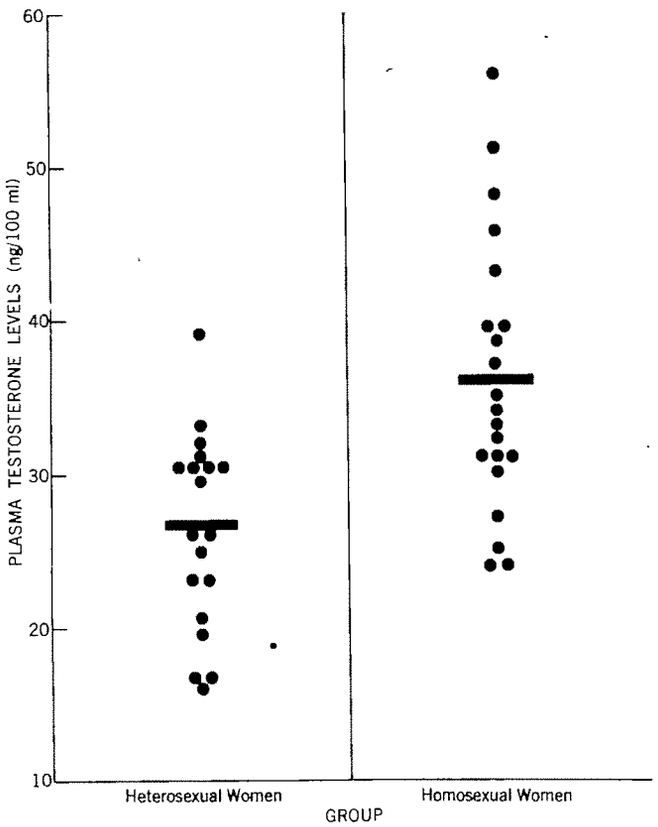
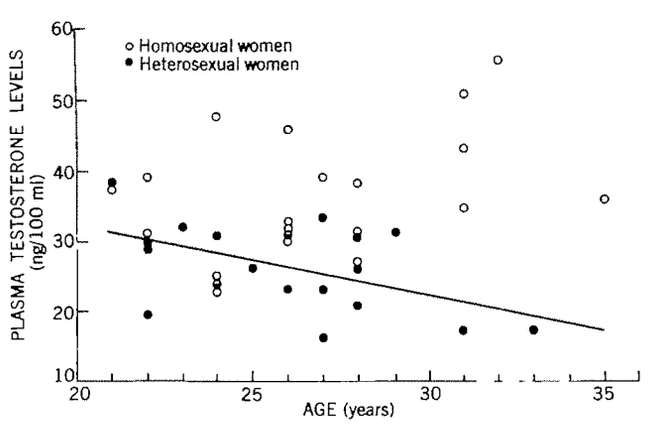


FIGURE 2
Plasma Testosterone and Age in Homosexual and Heterosexual Women



the two groups overlapped considerably, circulating testosterone values in homosexual subjects averaged 38% higher than those in the heterosexual group ($p<.001$).

It is unlikely that the observed differences in testosterone levels reflect age differences between the two groups studied, since the mean age of the homosexual women did not differ statistically from that of the heterosexual women. Moreover, in the heterosexual group, a statistically significant inverse correlation ($r=-.518$; $p<.02$) existed between age and testosterone concentration (figure 2). In the homosexual group, a slight tendency in the opposite direction was observed ($r=.359$; $p>.05$).

DISCUSSION

Results of this investigation support and extend earlier conclusions derived from analyses of testosterone in urine (8–10), even though such excretion studies are complicated by the collection of samples without regard to phase of menstrual cycle (8, 13, 14), by the low urinary levels of unmetabolized testosterone, as well as by the absence of any unique testosterone metabolite (15–17).

Available information precludes drawing any behavioral conclusions about the apparent association of elevated plasma testosterone levels and female homosexuality. Such factors as sleep, stress (5), mood or lifestyle might, for example, affect circulating levels of this hormone. Moreover, there is currently no basis for speculating whether the observed endocrinologic differences might reflect a primary mechanism for or a secondary response to a particular sexual orientation.

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A Study of 100 Physician Psychiatric Inpatients

BY ROBERT ERWIN JONES, M.D.

The author reviewed the case records of 100 physician inpatients in a private psychiatric hospital. He found that these physicians were more likely to have diagnoses of affective disorder and drug abuse than the general psychiatric hospital population, that their peak susceptibility occurred in their late 40s, that general practitioners and psychiatrists were overrepresented, and that female physicians were almost four times more vulnerable to suicide than male physicians. He describes some critical aspects of engaging the disabled physician in psychiatric hospitalization.

ALTHOUGH THE EMOTIONAL MORBIDITY of physicians is becoming better documented (1–8), there are still surprisingly few studies of psychiatric illness among physicians, except in the area of drug addiction. Reports on physician psychiatric inpatients consist of those by Pearson and Strecker (9), who studied 20 physician inpatients at the Institute of Pennsylvania Hospital; Duffy and Litin (10), who studied 93 at the Mayo Clinic; A'Brook and associates (11), who studied 114 at two English hospitals; Simon and Lumry (12), who studied 36 at the Minneapolis Veterans Administration Hospital; and Pond (7), who studied 29 physician inpatients at the Priory Hospital and 23 physician inpatients and outpatients at the Maudsley Hospital.

To augment this small but growing body of literature about psychiatric illness among physician inpatients, in this paper I will review data from the hospital case records of 100 physicians admitted to the Institute of Pennsylvania Hospital, a private psychiatric hospital, from 1967 to 1975. I will take special note of diagnosis, precipitating factors, alcohol and drug abuse, suicide, admissions of female physicians, and leaving against medical advice.

The 100 physician inpatients in this study were distributed in age at first admission as follows: in the 10–19-year-old group there was 1; 20–29, 5; 30–39, 22; 40–49, 38; 50–59, 23; and 60–69, 10; 1 of the patients was 89 years old. The average age was 45. The age distribution indicates a peak in the 40s. One interesting patient was first treated in this hospital at age 13 for more than a year for an illness described as catatonic schizophrenia. After he completed medical training, he returned three times to the hospital for treatment of manic-depressive illness with lithium carbonate; meanwhile, he has continued to work in medical research.

The 100 inpatients had a total of 200 admissions; 59 of these were single admissions. There were 5 1-day admissions. One nearly 5-year admission was that of a senile physician who died. Fourteen patients had 2 or 3 admissions in 1 year, indicating a cluster of admissions for one illness and possibly indicating that the first admission should have been longer. Eleven patients had 5 to 10 admissions to other hospitals. The average length of stay for the 100 inpatients was 36.5 days. The average length of stay for the drug- and/or alcohol-abusing patients was 29 days. Hospital stays of addicted physicians were only 10 days or less in 16 cases, indicating that detoxification rather than psychotherapy was the goal.

DIAGNOSIS

Table 1 indicates the primary diagnoses of the 100 physicians admitted in 1967–1975. The table also shows the diagnoses of 9,567 patients discharged from the hospital in the same period (13).

In the physician inpatient group, affective disorders

Revised version of a paper presented at the 129th annual meeting of the American Psychiatric Association, Miami Beach, Fla., May 10–14, 1976.

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TABLE 1
Diagnoses of 100 Physician Inpatients and 9,567 Inpatients Discharged from the Hospital During 1967–1975, in Percents

Diagnosis	Physician Inpatients	Discharged Patients
Psychoses associated with organic brain syndrome	1.0	2.6
Psychoses		
Schizophrenia	6.0	20.8
Involutional melancholia	2.0	2.6
Manic-depressive illness	19.0	10.1
Psychotic depressive reaction	3.0	2.9
Total	30.0	36.4
Neuroses		
Anxiety neurosis	8.0	—
Obsessive-compulsive neurosis	1.0	—
Depressive neurosis	43.0	—
Total	52.0	43.2
Personality disorders		
Passive-aggressive personality	1.0	—
Obsessive-compulsive personality	1.0	—
Emotionally unstable personality	1.0	—
Other	—	2.1
Alcoholism	5.0	4.5
Drug dependence	7.0	—
Total	15.0	6.6
Transient situational disturbances		
Adjustment reaction of adolescence	0.0	4.6
Adjustment reaction of adult life	1.0	1.0
Depressive adjustment reaction of late life	1.0	—
Total	2.0	5.6
Other	0.0	5.6

TABLE 2
Medical Specialties of 100 Physician Inpatients and All Physicians Practicing in the United States in 1970*, in Percents

Specialty	Physician Inpatients	All Physicians
General practice	28.0	18.6
Anesthesiology	5.0	3.5
Internal medicine	13.0	13.5
Dermatology	1.0	1.3
Neurology	1.0	1.0
Pathology	4.0	3.4
Pediatrics	4.0	6.1
Psychiatry	11.0	7.5
Radiology	3.0	3.4
Surgical specialties	16.0	27.5
Intern	2.0	—
Resident	6.0	—
Not recorded	5.0	—
Other	1.0	14.2

*According to the U.S. Department of Health, Education, and Welfare (14).

(depressive neurosis, manic-depressive illness, psychotic depressive reaction, involutional melancholia, and depressive adjustment reaction of late life) accounted for 68% of all conditions. A greater proportion of the physician inpatients had a depressive diagnosis than the hospital population as a whole. Simon and Lumry (12) found that depression was the complaint of 86% of the 36 physicians they studied and that 41% of their group were preoccupied with suicidal ideas.

The percentage of the 100 physician inpatients in our hospital in 1967–1975 who were drug addicted and/or alcoholic was about triple the percentage in the total hospital population over the same period, but schizophrenia was diagnosed much less frequently in these physicians.

The accuracy of psychiatric diagnosis of physicians is open to question because psychiatrists have a propensity to protect their medical colleagues from such potentially damaging labels as alcoholic, drug addict, and schizophrenic. Especially in the area of drug and alcohol abuse the statistics may be inaccurate. For example, for the 100 physician inpatients in our hospital, alcoholism was the primary diagnosis in 5 cases and the secondary diagnosis in 7; however, the case histories revealed noteworthy alcohol abuse in 12 additional cases. Drug addiction was the primary diagnosis in 7 cases and the secondary diagnosis in 6; however, drug abuse was recorded in an additional 9 case histories. Combined alcohol and drug abuse was described in 6 more case histories that did not carry the diagnosis of either type of dependency. Thus, although only 12 of 100 patients carried the primary diagnosis of alcohol and/or drug abuse, according to the case records more than half (N=52) actually had a substantial drug and/or alcohol problem.

The most common diagnosis used in undiagnosed alcohol and/or drug abuse is depressive neurosis; this fact helps to explain the high proportion of affective disorders in this series of physician inpatients. The reasons for such concealment of the facts are related to insurance benefits, lack of knowledge about diagnosis and treatment, and countertransference factors such as overprotection of the patient.

A comparison by specialty of these hospitalized physicians with the medical population of the United States indicates that general practitioners and psychiatrists were overrepresented but surgical specialists were underrepresented among these hospitalized physicians (see table 2). I can only speculate on the reasons for this disparity. The premorbid personality types of the different specialists is probably important. Also, general practitioners and psychiatrists tend to practice alone in their offices, but surgeons practice as members of teams and therefore receive the daily support as well as the scrutiny of their peers.

PRECIPITATING FACTORS

The psychiatrists who recorded the case histories studied were impressed with the compulsive, hard-working nature of the physician inpatients and their

drive for accomplishment and achievement. Some descriptions of the premorbid patients are as follows: "perfectionistic," "achiever," "overworked, then builds up resentment about extra duties," "has a large, successful practice," "driven to be successful," "driving hard," "has grandiose ideas about his practice," "could not refuse to accommodate patients," "complete preoccupation with work." A suicidal pathologist said, "The perfect doctor spends a great deal of time and energy in his role as a physician." The records have repeated references to the patient's inability to relax, such as "no vacation in five years." One general practitioner had not taken a vacation in 15 years! An alcoholic physician whose hospital privileges had been revoked because of his drinking had worked 7 days a week, seeing more than 100 patients daily. One amphetamine user had overworked at two hospitals, putting in 80–90 hours a week. The records give the impression that it was not so much the overload that created a burden for the physician as it was the overzealousness of the physician who attempted to handle the workload. In other words, the personality of the physician may have been more important than the occupational stress.

Although pressure of work was the most commonly given reason for hospitalization by the recording physicians, marital discord was also frequently cited. One record described a physician whose "practice was so busy it interfered with marriage." Interestingly, the records are noticeably inadequate in sexual histories, except for sporadic mention of poor sexual adjustment. This might reflect a reluctance on the part of psychiatrists to question their colleagues about such matters or to record the information. Concern about other family members and financial worries were the next most common reasons for hospitalization reported.

ALCOHOL AND DRUG ABUSE

In the drug- and/or alcohol-abusing group, toxicity and its consequences often precipitated admission. Four physicians had suffered falls and 2 had had automobile accidents after drinking. Eleven physicians had had staff privileges revoked by their peers, and 2 had had their medical licenses revoked. In only one instance was it recorded that a patient had complained that the physician was intoxicated, and there is no mention in these records of malpractrice that might be attributed to mental illness or addiction.

Even if pressured to enter the hospital by colleagues or family, most of these physicians (N=95) were admitted as voluntary patients. Four physician inpatients (2 schizophrenic, 1 manic, and 1 acutely intoxicated) required emergency detention, and 1 senile patient was committed by the court. The reluctance of physicians to seek treatment for either physical or emotional illness has been cited by many authors (9). Seventeen of the 100 physicians in our hospital had sought outpatient therapy before hospitalization.

TABLE 3
Specialties and Age Groups of 16 Suicidal Physician Inpatients

Item	Number of Physician Inpatients
Specialties	
Intern	1
Resident	2
General practice	5
Anesthesiology	1
Internal medicine	1
Obstetrics-gynecology	1
Psychiatry	2
Surgery	3
Age groups	
20–29	3
30–39	3
40–49	4
50–59	4
60–69	2

SUICIDE

Fifteen of the 100 physicians attempted suicide before admission, and 1 did so immediately afterward. Four others had attempted suicide on previous occasions. Seventeen more described suicidal thoughts that prompted their admission. Fourteen of the 16 suicide attempts were by overdose, 1 by shooting in the chest, and 1 by laceration of the neck. Thirteen of the 16 entered the hospital with a diagnosis of depression. Seven of the 16 had a history of drug or alcohol abuse. The specialties and age groups of the suicidal patients are shown in table 3.

In light of the fact that 37% of the hospitalized physicians had a history of suicidal thoughts or attempts, the prevention of suicide must be uppermost in the minds of the treating physicians. Two physician inpatients in this series made serious suicide attempts after admission to the hospital. Strenuous efforts at prevention of suicide must continue after a patient enters the hospital.

It is interesting to note that none of the 15 patients who made suicide attempts just before admission was treated with ECT (although 3 others in the total group received ECT). I do not have follow-up information about this suicidal group. The rate of unsuccessful suicide attempts in this series (16%) is comparable to the reported suicide rate among physician psychiatric patients (5%–19%) (4).

FEMALE PHYSICIANS

All of the 11 female physicians in this study group entered the hospital voluntarily. Eight of them admitted themselves for treatment of depression (5 neurotic, 2 manic-depressive, and 1 depressive adjustment reaction of late life). Six were general practitioners. Five had attempted suicide by means of overdose before admission, and 1 of these again attempted suicide by

laceration after admission. Four of the 5 who attempted suicide were Roman Catholic, and 3 were unmarried. This high percentage of suicide attempts (45%) contrasts markedly with the lower percentage among male physician admissions (11 of 89, or 12%). The average age of the suicidal women was 40, while that of the men was 49, indicating that younger unmarried female physicians are vulnerable to suicide, while the peak period of vulnerability for men is middle age.

DISCHARGE AGAINST MEDICAL ADVICE

Nine physician inpatients left the hospital against medical advice; 8 of these were drug and/or alcohol abusers. This rate is substantially higher than the overall rate of such discharges from our hospital, which was 1.1% over the 2-year period 1971–1973 (15).

COMMENTS ON TECHNIQUES OF CARE

The responsibility of caring for emotionally ill physicians rests with physicians. This unusual situation arouses denial, avoidance, resistance, hostility, and other defenses in both patient and doctor. Moreover, the family of the physician very often does not know how to care for the sick physician and turns to his or her colleagues for assistance. These, of course, are the very people from whom the potential patient may wish to hide the illness. The moment when the sick physician's family seeks expertise is critical because his or her colleagues' attitude and conduct can greatly influence future events for the patient, especially the patient's own attitude toward his or her illness. The colleague must be careful not to minimize the condition, not to avoid the situation, not to retreat and do nothing, but to respond with constructive guidance and compassion.

More effective and supportive mechanisms can be developed to care for sick hospital staff members, such as the Committee on Staff Health of the Institute of the Pennsylvania Hospital, a group of experts who offer confidential, nondisciplinary help. Physicians or their families may consult this group about a staff member's disturbed behavior. The group recommends proper care while safeguarding the potential patient's staff position, thus preventing rejection or humiliation by fellow staff members.

Physicians should not be afraid to use peer pressure. On the basis of our records, I would say that peer leverage—discontinuance of admitting and operating privileges, for example—is an effective and safe mechanism for prompting the hospital admission of the disturbed physician. The timely and compassionate use of this mechanism is much better than legally revoking a license later. A critical moment comes after the medical staff has decided to compel a colleague to obtain treatment. The staff must keep in mind that even if he or she denies it, which is likely, the potential patient is most likely depressed (there is at least a 60% chance), is very likely abusing alcohol or drugs (a more than 50% chance), may very likely have marital or family difficulties, and, faced with loss of income during hospitalization as well as with loss of prestige because of losing staff privileges, may develop suicidal thoughts (a more than 30% chance) or make a suicide attempt (a 15% chance). After the medical staff has decided to recommend hospitalization, it is essential that they exercise maximum care of the patient until he or she is safely in professional hands.

Engaging any alcoholic patient in treatment requires special technique; the alcoholic physician is no exception. One technique that is sometimes of value is direct confrontation of the patient by concerned members of the family. These family members must be carefully prepared ahead of time about the method and goals of the confrontation. Follow-up care is of utmost importance. A medical Alcoholics Anonymous may be available. The initial phase of hospital care is fraught with danger. Therefore, I would not hesitate to assign private duty nurses to physician inpatients for the first days of hospitalization until treatment is well under way.

I do not have accurate reports of follow-up care or results, but other authors have commented on the reluctance of physicians to remain in outpatient care. In my experience, the physician who seeks hospital care in a crisis quickly reasserts his or her driving, overachieving mechanisms and is not motivated to explore his or her personality, often giving lack of time and the pressures of medical practice as the reasons. Patients with chronic or recurrent illness require special consideration and are well advised to work in structured supervised settings where others can monitor their condition. Physicians who have multiple psychiatric hospitalizations need not fear that they cannot continue to practice medicine. Our records show that some physicians with repeated hospitalizations have maintained successful careers.

One way in which the psychiatrist might err in treating physicians is in assuming that all physicians are knowledgeable about psychiatry or psychotherapy. It is important to be instructive about psychiatric terminology, the goals of treatment, the mechanisms of drug actions, and the process of psychotherapy, just as with any other, nonmedical patient.

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On the Safety of Long-Term Treatment with Lithium

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Thirteen patients with bipolar affective illness who had received lithium therapy for 1–5 years were tested retrospectively for evidence of cortical dysfunction. Data on patients younger than 55 show no abnormalities on the Halstead-Reitan Neuropsychological Battery and suggest that chronic lithium therapy is relatively safe. Significantly elevated Halstead Impairment indexes were observed among elderly patients, but these data are difficult to interpret.

IT IS WELL known that lithium toxicity can result in a variety of central nervous system (CNS) abnormalities. Schou and coworkers (1) originally described 5 toxic patients exhibiting confusion, convulsions, or coma who made full recoveries within 6 weeks. Other investigators have also reported full recovery from lithium toxicity after discontinuation of the drug (2–4). A few reports have challenged the assumption that a patient who recovers from lithium intoxication will be free from persistent neurological sequelae. Von Hartitzsch and associates (5) described 2 patients in whom prolonged lithium intoxication was followed by permanent damage to basal ganglia and cerebellum. Permanent brain damage has likewise been reported by Juul-Jensen and Schou (6), although the contribution of diphenylhydantoin must be considered in 1 case, and by Cohen and Cohen (7) in 4 patients treated with lithium and haloperidol concurrently.

Much more disturbing are reports of adverse reactions among patients receiving therapeutic levels of lithium carbonate. Confusion, convulsions, dysarthria, cogwheel rigidity, and coma (8–13) have been observed in patients whose serum lithium levels did not exceed 1.5 mEq/liter. Alterations in EEG, consisting of diffuse high-voltage slow waves that disappear after discontinuation of medication, have also been reported at therapeutic levels (14–16). There are also a few reports of persistent focal EEG changes (16–18). Finally, Small and coworkers (19) have demonstrated significant neuropsychological impairment among patients maintained on therapeutic doses of lithium for only 3 weeks.

We became interested in this problem after observing 2 manic-depressive patients who developed persistent cortical dysfunction following acute lithium intoxication. We wondered whether manic-depressive patients maintained on chronic lithium prophylaxis for several years would exhibit any evidence of cortical dysfunction. Therefore we interviewed 13 patients with bipolar affective illness who had not experienced lithium intoxication and assessed cortical dysfunction by both EEG and the Halstead-Reitan battery.

METHOD

The 13 subjects were patients at the Dartmouth-Hitchcock Mental Health Center, Hanover, N.H. All

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had manic-depressive illness and had been included in a previous investigation on manic-depressive illness in families (20). Criteria for the diagnosis of bipolar affective illness included a history of at least one episode of illness lasting 3 or more weeks in which two of the three following manic signs or symptoms were evident: elated mood, flight of ideas, and hyperactivity. In addition, at least four of the following were required: decreased need for sleep, grandiosity, intense irritability, excessive plans, distractibility, increased sexual drive, pressure of speech, or extravagance. Individuals with a history of alcoholism or thought disorder were excluded. Thirteen of the original 39 patients studied by Goetzl and associates (20) had remained on lithium carbonate therapy and agreed to participate in the present study.

The CNS status of each patient was assessed by EEG and by Reitan's modification and elaboration of the Halstead neuropsychological test battery (21). The Halstead-Reitan battery is a sensitive indicator of the integrity of the cerebral hemispheres (22). It consists of the Wechsler Adult Intelligence Scale (WAIS), the Trails Test, the dynamometer, the Category Test, the Tactual Performance Test, finger tapping, the Speech Sounds Perception Test, the Seashore Rhythm Test, and a sensory and an aphasia examination. The impairment index represents a general approximation of the degree of cognitive deficit. According to Reitan, an impairment index of 0.5 or less is generally within normal limits. We also interviewed each patient and reviewed medical records to obtain data regarding lithium dosage, duration of therapy, serum lithium levels, use of other medications, and the patient's view of his or her illness before and after lithium maintenance therapy was initiated. It should be noted that before lithium therapy was initiated all patients had received a thorough neurological examination; no neurological abnormalities were detected in any patient at that time.

RESULTS

All patients were clinically euthymic at the time of this assessment and all were receiving lithium carbonate in maintenance doses of 600–1500 mg/day. The duration of maintenance therapy ranged from 1.2 to 5 years, with a mean of 3.2 years.

Every patient reported that lithium had changed his or her life. Before beginning lithium therapy each patient had been hospitalized at least once. Subsequent to treatment 9 patients reported no further hospitalizations, 3 reported one relapse, and 1 patient had required hospitalization twice. Table 1 shows individual patient data related to duration of illness, duration of lithium therapy, daily dose, and serum lithium level at the time the testing was conducted.

Twelve of the 13 patients had normal EEGs. The single exception, patient 3, was a 25-year-old woman who had received lithium carbonate for 3 years and whose serum lithium levels had remained within the therapeutic range. Her EEG was symmetrical with paroxysmal variations in voltage and rate throughout the record, which was interpreted as "an abnormal record suggestive of a seizure disorder." Neurological findings were within normal limits and there was no history of seizures or symptoms suggestive of a seizure disorder.

Among the 8 younger patients (aged 18–53) given the full Halstead-Reitan battery of neuropsychological tests (patients 1–8 in table 1), 7 showed no evidence of cognitive impairment or cortical dysfunction. All had impairment indexes of 0.1–0.3 and in none of their test results was there evidence of even mild focal cortical dysfunction or mild delirium. One patient had a testing record that was questionably borderline abnormal (patient 6 in table 1). He was a 41-year-old high school graduate with a WAIS full-scale IQ of 100 and an impairment index of 0.6. There was no evidence of focal cortical dysfunction but four of his seven Halstead battery indexes were in the lower end of the impaired range (Category Test, 72 errors; Tactual Performance Test, total time=18 minutes, 39 seconds; Tactual Performance Test (location), 2 correct; and Seashore Rhythm Test, 27 correct). We would judge his test results to be essentially normal or to indicate at most a very mild diffuse cortical dysfunction suggestive of a delirium.

Five older patients aged 59–68 were also tested (patients 9–13 in table 1). One of these, patient 12, had a record within normal limits. The other 4 patients all had elevated impairment indexes (0.7–0.9). While older subjects commonly have some elevation in impairment indexes (23), it was our judgment that these 4 patients manifested mild cognitive impairment beyond that which could be explained by their age alone. In none of their records was there consistent evidence for focal or asymmetric cortical dysfunction; rather, they seemed to have mild diffuse cortical dysfunction of a type often seen in mild states of delirium.

The trend toward elevated impairment indexes among the 5 oldest patients is highly significant in comparison with the 8 younger patients ($p<.005$, two-tailed Mann-Whitney U test). A comparison of the mean data for the two groups (table 1) shows that there are no differences in daily dose, serum lithium level, duration of lithium therapy, education level, or WAIS full-scale IQ. Thus the exposure of both groups to lithium carbonate is comparable. In addition to the age difference and the aforementioned difference in scores on the Halstead impairment index, the older patients have suffered from manic-depressive illness for almost 20 more years.

DISCUSSION

Many psychiatric investigations using the Halstead battery are restricted to subjects 55 or 60 years of age because older subjects sometimes exhibit cognitive impairment or cortical dysfunction secondary to the

TABLE 1
Comparison of 13 Manic-Depressive Patients Treated with Lithium for Several Years

Patient	Age	Sex	Education (years)	Illness Duration (years)	Lithium			Halstead Index	WAIS
					Therapy Duration (years)	Daily Dose (mg)	Serum Level (mEq/liter)		
Under 55 age group									
1	18	M	12	2	2	1200	0.8	0.3	123
2	24	M	14	5	5	1200	1.0	0.3	111
3	25	F	12	12	3	1200	0.8	0.1	119
4	29	F	12	10	4	600	0.8	0.1	108
5	37	F	16	19	1	900	0.7	0.1	113
6	41	M	12	10	2	900	0.5	0.6	100
7	51	M	19	23	5	1500	0.9	0.3	130
8	53	M	16	10	4	900	0.6	0.1	114
Mean	34.8		14.1	11.6	3.3	1050	0.76	0.26	116.1
Over 55 age group									
9	59	M	15	13	5	900	0.6	0.9	107
10	62	M	16	30	3	1200	0.7	0.7	122
11	65	F	12	25	3	600	1.0	0.9	107
12	66	M	13	46	4	900	0.9	0.4	124
13	68	F	15	37	3	600	0.4	0.7	110
Mean	62.4		14.2	30.2	3.6	840	0.72	0.72*	114.0

*Significantly higher than under 55 age group, $p < .005$, two-tailed Mann-Whitney U test.

aging process or to various systemic medical diseases. On the other hand, the Halstead index is a fairly sensitive indicator of neuropsychological function for nonelderly subjects.

Among 8 patients younger than 55 maintained on lithium therapy for several years, 7 exhibited no evidence of impaired cortical function on the Halstead battery. The eighth patient was essentially normal, although his performance on four Halstead subtests was very slightly abnormal. To our knowledge, no other retrospective studies of possible long-term effects of lithium carbonate on cortical function have been reported. Like all retrospective studies, this one lacks baseline data that would permit pre- and posttreatment comparisons. In this regard, it is impossible to evaluate the significance of the abnormal EEG observed in 1 patient because a baseline EEG was not obtained before treatment was initiated. The data strongly suggest, however, that patients maintained on lithium carbonate do not exhibit cortical dysfunction after years of treatment.

Results for the 5 patients older than 55 are much more difficult to interpret. It is impossible to judge whether their elevated Halstead indexes were secondary to lithium or some other cause. Older patients are well-known to be more susceptible to the deliriogenic effects of many agents (e.g., general anesthetics, benzodiazepines, anticholinergics), and this may be the case with lithium as well. It might follow that older patients would also be more susceptible to structural CNS changes secondary to lithium toxicity, although we know of no data on this issue. Finally, it may be that subtle cognitive impairment is a natural consequence that follows decades of manic-depressive illness with or without lithium therapy. We know of no data that address this interesting question. Whatever the reason, lithium is no exception to the axiom that therapeutic agents require careful monitoring in older patients.

Although we realize that this report may be added to the growing list of published warnings about the dangers of lithium, we have not submitted these results as an admonition. Rather, we are impressed by the lack of CNS consequences observed among the 8 younger patients after years of lithium therapy. Questions raised by data such as these must remain unanswered, however, until prospective studies are launched to study the relative safety of long-term treatment with lithium carbonate.

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Focused Psychiatric Curriculum Selection: Student, Psychiatrist, and Nonpsychiatrist Physician Expectations

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The authors obtained curriculum needs analysis data from second-year medical students and compared these with data obtained from nonpsychiatrist physicians and psychiatrists. All groups felt that interviewing skills, the doctor-patient relationship, managing the dying patient, and evaluation of suicidal patients were important topics. All gave psychological testing, behavior modification, mental retardation, and psychoanalytic theory the lowest teaching priority. The students gave a higher rating to all topics than did the other groups. The authors suggest that this kind of analysis can facilitate curriculum selection and implementation.

PSYCHIATRIC EDUCATORS have shown increasing interest in applying modern educational principles to their instructional programs for medical students. As a reflection of this interest, a number of papers on psychiatric teaching goals and specific curriculum content have been published (1–5).

In 1975, after failing to find consensus in the literature about the composition of a psychiatric curriculum for medical students, two of us (W.J. and J.S.) (6) undertook a study to objectify course selection. Our study provided data on the relative importance of 21 commonly taught psychiatric topic areas as perceived by 131 mental health professionals and nonpsychiatrist physicians. In the same year Bowden and Barton (7) published a comprehensive paper outlining topical material for an entire psychiatric curriculum that they considered essential to the education of medical students. Their outline represented a composite of ideas derived from the literature, a survey of subjects commonly taught at a number of medical schools, and a synthesis of their own ideas.

Significantly, the topics mentioned in Bowden and Barton's study were similar to those we investigated. The former study outlined a carefully thought-out psychiatric curriculum as defined by educators, and the latter provided some objective data on the relative importance of specific teaching topics as evaluated by

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practicing professionals. A review of these papers will provide designers of curricula contemporary ideas about what should comprise instruction for medical students. However, we feel that data from an additional source should be included during curriculum planning to assure optimum sequencing, relevance, and acceptance of instruction for the students. Along with others (8–10), we feel that an additional source of curriculum data can come from the students themselves, who are the immediate consumers of the instructional program.

It can be argued with some justification that most medical students do not possess the knowledge or experience that would enable them to make meaningful judgments about the future relevance of the subject matter they are taught. However, medical students do have opinions and expectations about the curriculum that affect their learning. We believe it is useful for the educator to be apprised of student opinions and, accordingly, to make allowances for them within the structure of the curriculum as it is being designed. Instruction is likely to proceed more smoothly when students know what is expected of them and when they can perceive the relevance of the material to be learned. If the course expectations are similar for students and teachers, the educational process can proceed with fewer difficulties. On the other hand, if the teacher and students have disparate opinions about the relevance or sequencing of the subjects taught, the educational process may evoke needless anxiety, confusion, or negativism in the students. In order to alleviate these problems the students may need special orientation about the nature, relevance, and sequencing of course work before actual instruction begins.

We hypothesize that there may well be differences between instructor and student expectations about psychiatric course work. If such discrepancies exist, it is good to know about them and plan accordingly. This paper describes our attempt to evaluate and compare student ideas of psychiatric course work material with those of practicing physicians and psychiatrists. We hope this comparison provides a means of improving the instructional process.

METHOD

For our 1975 study (6) we developed a questionnaire that asked, "What specific psychiatric information and skills should a nonpsychiatrist physician have?" The questionnaire contained 21 psychiatric topics commonly taught to medical students. Respondents were asked to rate the relative importance of the topics by assigning one of the following designations to each: not at all important, moderately important, very important, and essential.

We distributed a total of 185 of these questionnaires to a sample of faculty members at the University of Southern California School of Medicine, faculty members at the school's Department of Psychiatry and Division of Psychology, the staff of two local psychiatric hospitals, and the staff of a private general hospital. Completed questionnaires were received from 18 clinical psychologists, all of whom had Ph.D. degrees; 48 psychiatrists; and 65 nonpsychiatrist physicians. Two-thirds of the mental health professionals were involved in the education of medical students and/or residents. Approximately two-thirds of the nonpsychiatrist physicians were pediatricians, internists, and family physicians. The remaining third practiced various surgical specialties. All but 2 of the nonpsychiatrist respondents were regularly involved in providing direct or indirect patient care.

For purposes of the current study the same questionnaire was distributed to second-year medical students during the orientation session for their first presentation in psychiatry, an introductory course in psychopathology. Student data were gathered for two successive years, 1974 and 1975, and the results obtained were compared with the data collected from the psychiatrists and nonpsychiatrist physicians surveyed in 1975 (6).

RESULTS

Complete data were collected from 88 second-year students in the fall of 1974 and from 108 students in the fall of 1975. The two student groups were very similar in their responses. A significant difference between the ratings of the two groups was found for only 1 of the 21 topic areas. The students in 1975 rated an awareness of psychodynamic principles in evaluating a patient's behavior as significantly more important than did the students in 1974 ($t=-3.63$, $p<.01$). The remaining 20 topic areas were rated the same in importance by the two student groups. Because the two student groups were highly similar, their responses were pooled to form a single group for statistical comparisons with the psychiatrists and the nonpsychiatrist physicians surveyed in 1975.

Table 1 presents the mean rating of importance given to each topic area by each of the three groups. The rank order of the topic areas for each group was obtained from the mean ratings. The rankings of the topic areas listed on the right side of table 1 indicate the feeling of each group about the relative importance of each topic in relationship to the others. Kendall's coefficient of concordance for the three rank orders is .92, indicating a high degree of similarity among the three groups. This similarity is particularly remarkable as one examines the 5 topic areas considered most and least important by the three groups.

Four of the 5 topic areas considered most important by psychiatrists, nonpsychiatrist physicians, and students are identical. Specifically, all groups ranked the following among the 5 most important topic areas: the ability to talk with patients about their personal problems, an understanding of the emotional aspects of the chronically ill or dying patient, familiarity with the dy-

TABLE 1
Mean Ratings of Importance and Ranks of 21 Psychiatric Topics by Psychiatrists (N=48), Nonpsychiatrist Physicians (N=65), and Medical Students (N=196)

Information or Skill	Ratings*						Ranks**		
	Psychiatrists		Nonpsychiatrist Physicians		Students				
	Mean	SD	Mean	SD	Mean	SD	Psychiatrists	Nonpsychiatrist Physicians	Students
The ability to talk with patients about their personal problems	3.62	.64	3.61	.72	3.86	.42	1	1	1
The ability to distinguish among psychosis, neurosis, organic brain conditions, and character disorders	2.93	.80	3.12	.85	3.38	.72	9	3	9
A working knowledge of the community resources available for the care of emotionally disturbed individuals	2.68	.80	2.53	.83	3.54	.64	11	12	6
An awareness of psychodynamic principles in evaluating a patient's behavior	2.08	.67	2.46	.86	2.84	.89	17	13	15
The ability to evaluate a patient's potential for suicidal or destructive behavior or need for psychiatric hospitalization	3.38	.73	3.06	.89	3.57	.61	4	5	5
An awareness of when and how to refer a patient to a psychiatrist	3.47	.65	2.86	.89	3.59	.61	2	7	4
The ability to do brief psychotherapy	2.16	.83	2.20	.90	2.70	.80	14	16.5	16
Basic knowledge of current psychiatric treatment modalities	2.10	.66	2.20	.79	2.59	.76	16	16.5	18.5
An understanding of normal personality development	2.43	.74	2.38	.84	3.12	.73	12	15	12
An understanding of the principles of psychoanalytic theory	1.50	.58	1.72	.83	2.14	.75	20	21	21
The ability to diagnose and treat family and marital disturbances	2.12	.67	2.66	.85	2.91	.76	15	10.5	14
A working knowledge of common psychopharmacological medications	3.14	.71	2.84	.85	3.09	.78	6	8	13
An understanding of sexual problems and their management	2.75	.69	2.66	.85	3.36	.65	10	10.5	10
The ability to diagnose and manage the mentally retarded	1.91	.79	1.87	.89	2.63	.80	18	20	17
Familiarity with the principles of behavior modification	1.54	.54	2.01	.83	2.59	.74	19	18	18.5
An understanding of the physiological comcomitants of emotional stress	3.02	.66	2.90	.93	3.39	.69	7	6	8
Familiarity with the dynamics of the doctor-patient relationship	3.31	.72	3.13	.80	3.62	.58	5	2	3
An understanding of the emotional aspects of the chronically ill or dying patient	3.41	.57	3.10	.81	3.67	.49	3	4	2
Familiarity with the major types and uses of psychological tests	1.41	.53	1.98	.80	2.39	.75	21	19	20
An understanding of the common behavior disorders of children and adolescents	2.31	.62	2.41	.86	3.17	.72	13	14	11
A basic knowledge of alcohol and drug abuse	2.97	.75	2.84	.73	3.48	.61	8	9	7

*The rating system is as follows: 1=not at all important, 2=moderately important, 3=very important, and 4=essential.
**1=topic considered most important; 21=topic considered least important.

namics of the doctor-patient relationship, and the ability to evaluate a patient's potential for suicidal or destructive behavior or need for psychiatric hospitalization.

The groups also agreed that the following topic areas were among the 5 least important to learn: an understanding of the principles of psychoanalytic theory, familiarity with the major types and uses of psychologi-

cal tests, familiarity with the principles of behavior modification, and the ability to diagnose and manage the mentally retarded.

Data derived from the comparison of rank orders can be deceptive if differences in absolute value are ignored. For example, all groups ranked in approximately 10th position an understanding of sexual problems and their management. However, an inspection of the actual mean ratings for each group shows that they range from 2.66 for nonpsychiatrist physicians to 3.36 for students.

Table 1 shows that despite the similarities in the rank ordering of topics, the three groups differ in absolute ratings. Most striking is that the students perceived every topic as more important than either the psychiatrists or the nonpsychiatrist physicians. The average rating of the students over all 21 topic areas is 3.12, compared with 2.58 for the psychiatrists and 2.59 for the nonpsychiatrist physicians. On the average, the students perceived the 21 topics as very important, but the psychiatrists and nonpsychiatrist physicians on the average perceived the topics falling midway between moderately and very important. Analyses of variance performed on each question showed that a significant difference in mean rating ($p<.05$) was present for 20 of the 21 topic areas. The only topic area for which the three groups expressed similar mean ratings was a working knowledge of common psychopharmacological medications. Students, psychiatrists, and nonpsychiatrist physicians all rated this topic close to very important.

It is clear that before further comparison of the similarities and/or differences among the ratings of the three groups, one must control for the differences noted in actual mean value. To accomplish this, each person's ratings were converted to standard scores pivoting around that particular individual's mean rating for all 21 topics. These transformed scores represent the relative value a topic had for a particular individual (an ipsative measure).

Analyses of variance of the transformed scores were performed to detect any significant differences in the relative positioning of each topic area by the students, psychiatrists, and nonpsychiatrist physicians. The average standard scores for all topics for each group are available on request. Significant differences were found for 7 of the 21 topic areas. Specifically, the students were inclined to rate a working knowledge of the community resources available for the care of emotionally disturbed individuals and an understanding of the common behavior disorders of children and adolescents as relatively more important than either psychiatrists or nonpsychiatrist physicians. Psychiatrists tended to rate an awareness of when and how to refer a patient to a psychiatrist and a working knowledge of common psychopharmacological medications as relatively more important than did the other two groups. Nonpsychiatrist physicians tended to view the ability to diagnose and treat family and marital disturbances as relatively more important than the students or the psychiatrists. Finally, although all three groups judged familiarity with the principles of behavior modification and familiarity with the major types and uses of psychological tests as unimportant, the psychiatrists rated these topics significantly lower than did either the students or the nonpsychiatrist physicians.

DISCUSSION

This study was undertaken to refine further the selection of psychiatric topics potentially important for medical students to learn by examining the opinions of mental health professionals, nonpsychiatrist physicians, and students. We believe that the selection of a curriculum based on broad consensus, including that of the students, is more likely to result in a relevant and acceptable course. Specifically, this project aided in establishing the rank order of topics for each of the three groups, identifying those topics most important for inclusion in a psychiatric curriculum, and indicating those topics for which pre-instruction preparation may be needed.

The major findings indicated a remarkable similarity among all three groups in their selection of the most and least important topics. An examination of these data should enable psychiatric educators to select a core curriculum that meets both the future needs of the students and their current expectations. However, significant differences were found in the absolute value that the groups placed on various topics. For example, the students tended to rate all of the topics as more important than did the professional groups. Although the high student ratings may be partially attributed to a lack of knowledgeable discrimination, their judgment may also indicate an acceptance of and perhaps enthusiasm for psychiatric instruction. Every effort should be made by means of thoughtful curriculum planning and implementation to maintain this high expectation.

In spite of consensus on most topics, the unique curriculum interests of each of the three groups is noteworthy. It would be well for psychiatric educators to respond to these stated expectations in some constructive fashion during curriculum development and implementation. For example, in any given curriculum one could elect to include only those topic areas ranked high by all groups. Such an approach would give reasonable assurance that student receptivity and topic relevance would be optimal. However, for cogent reasons a curriculum should anticipate possible future needs as well as those presently recognized. It may be quite appropriate, therefore, to include topic areas that are not currently perceived as important by the students. In such an instance, the results of the needs analysis serve to alert one to the importance of special student preparation by demonstrating the future relevance of the topic area and the need to change current attitudes. The recognition and discussion of the discrepancy between currently felt curriculum needs and possible future requirements will help greatly in dis-

pelling the attitudes about psychiatric education held by many physicians, that it is "poorly taught and irrelevant to the needs of medical practice" (11).

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Juvenile Court Versus Child Guidance Referral: Psychosocial and Parental Factors

BY SHELLEY S. SHANOK, M.P.H., AND DOROTHY OTNOW LEWIS, M.D.

The authors compared the psychiatric symptoms, socioeconomic status, family structure, family size, and occurrence of treated parental mental illness in 136 children referred to a juvenile court and 82 children referred to a child guidance clinic. The prevalence of psychiatric and organic symptoms and treated parental psychopathology in both groups of children did not differ significantly. Juvenile court clinic children, however, tended to come from larger families and to lack a biological mother in the home. The authors explore explanations for the routing of sick children to the juvenile court rather than to a treatment facility.

THE PURPOSE of this paper is to report the results of a study comparing the psychiatric symptoms, socioeconomic class, and family constellation of children referred to a juvenile court psychiatric clinic and of children referred to a child guidance clinic from the same geographic area. We will also compare the psychiatric status of parents of children in the two groups as reflected in psychiatric treatment records. Finally, we will explore possible reasons why certain children were referred to the juvenile court and then to its psychiatric clinic, whereas other children with similar symptoms and behaviors were recognized as being psychiatrically disturbed rather than predominantly delinquent and were referred to a child guidance clinic.

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LITERATURE

The fact that the first child guidance clinic in the United States was founded to aid the juvenile court in Chicago (1) indicates an early recognition that many children who came in conflict with the law had psychiatric problems. In recent years, such investigators as Stephenson and associates (2), Warren (3), and Bennett (4) have expressed again the view that delinquent children resemble children with diagnosed psychiatric disorder. However, most of the psychiatric literature on delinquent children has stressed findings of characterological or neurotic disturbances. Serious psychiatric symptoms such as psychotic symptoms have been reported to be rare (5, 6).

On the other hand, although psychotic disorders have rarely been reported in delinquent populations, longitudinal and retrospective studies have docu-

mented the existence of psychotic disorders, particularly schizophrenia, in adults who were delinquent as children (7–13). Furthermore, although some investigators have reported an association between organic dysfunction and antisocial behavior (14–16), others have found no greater prevalence of organic disorders in criminals than in other populations (17).

The present study comparing children referred to a juvenile court clinic with those referred to a child guidance clinic sprang from the observation that 25% of the first 40 children referred to the New Haven Juvenile Court Clinic displayed psychotic symptoms (18). In another study we found that 18 of 285 children seen at the court clinic over a two-year period demonstrated symptoms of psychomotor epilepsy in association with paranoid ideation and assaultive behavior (19). Further, we have found that parents of delinquent children were treated for psychiatric impairment significantly more often than were other individuals in a lower-class population of New Haven and more often than the general population of Connecticut (20). Having noted serious psychopathology in children referred to a court clinic and in their parents, we wondered in what ways these children and their families resembled or differed from those seen in child guidance clinics. To this end our research was directed.

METHOD

Our sample consisted of all of the children between the ages of 7 and 16 who had been referred for evaluation to the New Haven Juvenile Court Clinic during a one-year period in the early 1970s (N=136; there were 90 boys and 46 girls). Our comparison sample consisted of all of the children between the ages of 7 and 16 who had been referred to a child guidance clinic in the same vicinity during the same target year (N=82; there were 58 boys and 24 girls).

The court clinic children studied were somewhat younger and had committed more offenses than the general court population, but they were similar to the general court population with respect to social class, race, sex distribution, and occurrence of treated parental psychopathology. The methodology for these comparisons has been reported elsewhere (21).

Psychiatric Symptoms and Demographic Characteristics

Psychiatric and demographic data were gathered from clinical records at the child guidance clinic and from juvenile court and court clinic records. Because the children did not always fit into hard and fast diagnostic categories, psychiatric information at the juvenile court was recorded in terms of symptoms rather than diagnoses. Psychiatric symptoms were initially recorded as neurotic, characterological, adjustment, psychotic, organic, or a combination of any of the above. For the purpose of data analysis, we divided the symptoms into three categories: 1) adjustment, characterological, and neurotic symptoms as one group, 2) psychotic symptoms, and 3) organic symptoms. A child could be categorized as demonstrating more than one kind of symptom.

Records at the child guidance clinic contained much descriptive information, but the children had not been assigned to symptom categories. Therefore, in the interest of obtaining comparable data and maintaining confidentiality of subjects, one of us (S.S.S.) abstracted each child's chart, taking down verbatim all references to symptoms. The abstracts were then reviewed independently by three experienced clinicians, two of whom were child psychiatrists and one a child psychologist. Each clinician noted the presence or absence of psychotic, organic, or what for the purposes of this study were considered less serious symptoms (e.g., adjustment, characterological, and neurotic).

All three clinicians agreed on the nature of the symptoms in 60% of all cases. There was agreement between at least two of the raters in an additional 37.5% of the cases, and there was no agreement in 2.5% of the cases. Thus there was agreement between at least two of the raters in 97.5% of all the cases. When there was no agreement among the raters, the child was classified as having no diagnosis. In cases in which two clinicians agreed, the child was assigned to the symptom categories agreed on by the two raters.

Measures of Parental Mental Illness

Parental mental illness was measured according to whether or not a parent had ever received treatment or had been hospitalized at one or more of the three state hospitals and/or the major hospital-based psychiatric facility in the geographic area of the study. Our study group consisted of 264 parents of juvenile court children (134 mothers and 130 fathers) and 158 parents of child guidance clinic children (82 mothers and 76 fathers). Mental illness was classified in terms of inpatient or outpatient care with the assumption that inpatient care reflected a more severe disturbance than outpatient care.

Information was also gathered on occupation of head of household, marital status, race, person(s) with whom the child was living, number of siblings in the household, and the child's sex. The Warner 7-Point Scale of Occupation was used to determine social class in both groups (22).

FINDINGS

The children in the juvenile court clinic and the children in the child guidance clinic had a similar prevalence of organic and psychotic symptoms. However, characterological, adjustment, or neurotic symptoms were noted significantly more often among children at the child guidance clinic (see table 1).

The difference between the proportion of parents in each group who had received any kind of psychiatric treatment was not significant. Similarly, when parental

TABLE 1
Psychiatric Symptoms in 136 Children Referred to a Juvenile Court Clinic and 82 Children Referred to a Child Guidance Clinic

Type of Symptom	Delinquent Children		Guidance Clinic Children		Chi-Square	Significance
	Number	Percent*	Number	Percent*		
Neurotic	58	42.65	47	57.73	4.344	$p<.05$
Organic	24	17.65	20	24.39	1.056	n.s.
Psychotic	29	21.32	15	18.29	0.134	n.s.
No diagnosis**	27	19.85	6	7.32	—	—

*Because a child could be assigned to more than one symptom category, the percent totals are greater than 100% of the sample size.
**Chi-square analysis was not done with children in this symptom category.

TABLE 2
Psychiatric Treatment Received by 264 Parents of Children Referred to a Juvenile Court Clinic (134 Mothers and 130 Fathers) and 158 Parents of Children Referred to a Child Guidance Center (82 Mothers and 76 Fathers)

Item	Parents of Delinquent Children		Parents of Guidance Clinic Children		Chi-Square	Significance
	Number	Percent	Number	Percent		
Mothers						
Outpatient treatment	12	8.96	12	14.63	1.135	n.s.
Inpatient treatment	20	14.92	13	15.86	0.00074	n.s.
Total	32	23.88	25	30.49	0.828	n.s.
Fathers						
Outpatient treatment	8	6.15	8	10.53	0.742	n.s.
Inpatient treatment	5	3.85	4	5.26	0.0162	n.s.
Total	13	10.00	12	15.79	1.014	n.s.
Total						
Outpatient treatment	20	7.58	20	12.66	2.412	n.s.
Inpatient treatment	25	9.47	17	10.76	0.068	n.s.
Total	45	17.05	37	23.42	2.177	n.s.

treatment was analyzed separately in terms of inpatient and outpatient treatment, there were no significant differences. Furthermore, when the occurrence of maternal psychiatric treatment and paternal psychiatric treatment were compared separately, no significant differences were found (see table 2).

A comparison of social class, as reflected by the occupation of the head of each household, revealed a significant difference between the two groups of children. In the juvenile court clinic, 86% of the 86 families on whom information about occupation was available came from the lower four classes, compared with approximately 74% of 82 child guidance clinic families ($\chi^2=13.089$, df=6, $p<.05$).

At the juvenile court clinic, 50 of the 136 children (37%) came from intact homes in which their natural parents were married; 65 children (48%) came from broken homes in which parents were divorced, separated, single, or widowed; and 21 children (15%) came from homes in which insufficient data were available to determine the marital status of the parents. At the child guidance clinic, 41 of the 82 children (51%) came from intact homes, and 40 children (49%) came from broken homes. No significant difference was found between groups in relation to the marital status of parents ($\chi^2=.7078$, $p>.05$).

Although there was no difference between the groups in terms of married, separated, or divorced parents, a significantly greater proportion of the children living in homes without a biological mother were seen at the court clinic rather than the child guidance clinic. Twenty of 121 delinquents on whom data were available (17%) were motherless, compared with only 3 child guidance clinic children (4%) ($\chi^2=6.6904$, $p<.05$).

The family structure of our subjects was further explored to determine whether the juvenile court clinic families and the child guidance clinic families differed in size of family. Seventy-three of the 109 children for whom information was available in the juvenile court clinic (66%) had three or more siblings, compared with 24 of 82 children in the child guidance clinic (30%); this difference was significant ($\chi^2=26.00$, $p<.001$).

Because we knew that the two groups differed in social class we wondered whether the increased number of siblings among the delinquent children was related primarily to their lower social class. When social class was held constant, we still found that children in so-

cioeconomic classes 6 and 7 at the juvenile court clinic had significantly more siblings than did children in socioeconomic classes 6 and 7 at the child guidance clinic (class 6, $\chi^2=6.626$, $p<.05$; class 7, $\chi^2=5.235$, $p<.05$).

DISCUSSION

The major finding of this study was that children seen in the juvenile court clinic demonstrated psychotic and/or organic symptoms similar in nature to the symptoms of children seen at a child guidance clinic. Thus the juvenile court clinic appeared to be a repository for many extremely disturbed children.

Since the occurrence of parental psychiatric treatment was also similar in both groups, neither a child's severe psychiatric symptoms nor his or her parents' psychopathology could explain why a child found himself or herself in a juvenile court clinic rather than in a more appropriate child psychiatric facility.

Social class and family size were two factors that helped to explain the difference in referral. Court children tended to come from lower social classes and larger families. Nevertheless, the great majority of the children in both groups were from the more deprived sectors of the community. Our most striking finding was the difference in proportions of motherless children in the juvenile court clinic population compared with the child guidance clinic population. Although the proportions of broken homes were similar, there were more motherless children or children living with stepmothers in the juvenile court clinic than in the child guidance clinic.

These findings suggest that when a natural mother is not present there may be nobody to protect the best interests of the psychiatrically disturbed child and direct him or her to a therapeutic rather than a correctional facility. These findings are consistent with our earlier findings comparing adopted and neglected children seen in the juvenile court clinic (23). We found that adopted children referred to the court clinic were more likely to be sent to training schools than were so-called neglected children with natural parents, even though the latter group of children had committed more frequent and more serious delinquent acts. Natural parents seemed to mobilize far greater resources to keep their delinquent children from training schools than parents of adopted delinquent children.

The present study suggests that in homes without a biological mother, fathers may be unable to take on an effective mothering role. They may not recognize the meaning of their child's behavior problems and may not get treatment for them. In cases in which there is a stepmother, her entrance into the home may be too late for an adequate reciprocal bond to form between the child and the surrogate mother. Attachment difficulties may be compounded in large families composed of natural children and stepchildren. In these homes mothers may lack an intense emotional commitment to a psychiatrically disturbed stepchild and lack an investment in protecting him or her from the legal consequences of delinquent acts by obtaining treatment in a psychiatric facility.

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Fear of the Mentally Ill: Empirical Support for the Common Man's Response

BY JOHN M. LAGOS, PH.D., KENNETH PERLMUTTER, AND HERIBERT SAEXINGER, M.D.

The authors studied the hospital records of 400 hospitalized mentally ill patients for evidence of violent behavior leading to admission. They found that 36% of 321 admissions (excluding patients diagnosed as having a personality disorder or as drug- and/or alcohol-dependent) were preceded by some form of violent, fear-inspiring behavior. They suggest that mental health professionals deal with the general public's fear of the mentally ill on the basis of such findings rather than dismiss it as illogical.

THE PUBLIC'S FEAR of the mentally ill is well known. Among mental health professionals this fear is generally considered to be unfounded, and there is ample support for that contention. In their review of research on the subject, Rappeport and associates (1) concluded, "Crime rates are not higher among ex-mental patients than among corresponding persons in the general population; indications are that the reverse is true" (p. 76). Despite such evidence, the common man remains unconvinced (2).

Some authors account for the persistence of this fear by hypothesized psychodynamics of the general population and the social functions of belief systems (3–5). Such explanations make very thoughtful contributions to our understanding of this phenomenon, but, compelling as they may be, explanations are no substitute for facts, and one cannot help but wonder if all the facts have been gathered. Every study concluding that the mentally ill are not dangerous has gone to police records to seek its data. Perhaps an alternative data source would yield facts that suggest different conclusions.

We approached the question of violence among the mentally ill by examining hospital records of psychiatric admissions. The purpose of this study was to determine how frequently people behave violently when they become mentally ill.

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METHOD

We randomly selected the records of 100 patients admitted during 1974 to each of 4 psychiatric facilities in New Jersey. We reviewed the admission notes for these 400 patients for any description of patient behavior that might be construed as violent. The described behavior had to be recent and presented as part of the basis for admission. Appendix 1 contains a list of the categories of violent behaviors presented in descending order of severity and/or specificity. These categories were used in tabulating the behavioral descriptions obtained from patient records. If a given record presented more than one description that might have been placed in more than one category, only the most serious description was tabulated. Thus there was only one entry per patient.

RESULTS

The data revealed that 37.7% of the 400 admissions sampled manifested some form of violent behavior. Twenty percent of the sample were specifically described as acting in a physically violent way toward persons or objects, and another 11% were vaguely described as acting violently. Because those in the ambiguous category may also have attacked persons or objects, somewhere between 20% and 31% of the population sample may be said to have acted violently in a physical way toward persons or objects.

We thought it possible that these statistics on violent behavior just before psychiatric hospitalization might have been inflated by the inclusion of patients who were not mentally ill according to the most common use of that term.[1] Therefore, we removed the data on individuals who carried a diagnosis of personality disorder or whose primary discharge diagnosis indicated the presence of a toxic condition due to alcohol or drugs. Table 1 shows that 35.8% of the remaining sample (N=321) manifested some form of violent behavior; 18.1% of these were specifically described as

[1]The layman typically distinguishes among those who act irrationally because they have undesirable or deficient characters, those who act irrationally because they have been taking alcohol or drugs, and those who act irrationally because they are "not in their right mind." Only the latter are generally thought of as mentally ill.

TABLE 1
Admissions of Mentally Ill Patients to Four New Jersey Hospitals* with Reports of Violent Behavior

Type of Behavior	Hospital 1 (N=74)		Hospital 2 (N=86)		Hospital 3 (N=91)		Hospital 4 (N=70)		Total (N=321)	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Attacks on persons	16	21.6	7	8.1	4	4.4	12	17.1	39	12.2
Attacks on objects	7	9.5	5	5.8	5	5.5	2	2.9	19	5.9
Threats to persons	4	5.4	1	1.2	1	1.1	1	1.4	7	2.2
Verbal attacks on persons	0		1	1.2	1	1.1	1	1.4	3	.9
Ambiguous violence	7	9.5	11	12.8	4	4.4	13	18.5	35	10.9
Loss of impulse control	5	6.8	2	2.3	1	1.1	4	5.7	12	3.7
Total	39	52.8	27	31.4	16	17.6	33	47.0	115	35.8

*Hospital 1=Martland Medical Center; hospital 2=Essex County Hospital Center; hospital 3=Mount Carmel Guild; and hospital 4=Marlboro Psychiatric Hospital.

TABLE 2
Comparison of All Mentally Ill Patients and Those with Reports of Violent Behavior, by Ethnic Group and Sex

Item	Patients with Reports of Violent Behavior (N=115)		All Patients (N=321)	
	Number	Percent	Number	Percent
Black patients				
Male	30	26.1	63	19.6
Female	22	19.1	82	25.6
Total	52	45.2	145	45.2
White patients				
Male	34	29.6	73	22.7
Female	20	17.4	85	26.5
Total	54	47.0	158	49.2
Spanish-speaking patients				
Male	7	6.1	9	2.8
Female	2	1.7	9	2.8
Total	9	7.8	18	5.6
All male patients	71	61.7	145	45.1
All female patients	44	38.3	176	54.9

acting in a physically violent way toward persons or objects, and 10.9% were vaguely described as acting violently. Thus between 18% and 29% of those defined as mentally ill according to the common use of the term acted in a physically violent way toward persons or objects.

Because 45.2% of the sample were black, we tabulated the statistics to determine if the overrepresentation of one ethnic group or the underrepresentation of another had biased the results. According to the data presented in table 2, no one of the three ethnic groups identified contributed to the total number of patients with violent behaviors in greater proportion than its proportion in the sample would warrant. Of the 115 mentally ill patients whose records showed violent behavior (see table 2), only 3 (2.6%) were arrested.

DISCUSSION

Are the mentally ill to be feared or not? This is a terribly practical question in our era of community treatment. If mental patients are to be helped by this contemporary approach, communities must accept them. However, if communities fear the mentally ill they are likely to reject them. The mental health professions seem to approach the problem of community fear from the stance that mental patients are not dangerous, fearing them is irrational, and more community education is necessary to dispell the anxiety and prejudice. The persistence of the general fear of the mentally ill compells us to reexamine this stance.

There are two sources of support for the contention of mental health professionals that mental patients are neither dangerous nor to be feared: research data and clinical experience. The earlier research studies (6–9) reported that the mentally ill are less likely to be violent than the general population. More recent findings (10–13) do not support the proposition that the mentally ill are *less* likely to be violent than the general population, but they do not offer any firm support for the belief that mental patients are *more* likely to be violent either.

Rappeport and Lassen (10, 11) found that female patients had a significantly higher arrest rate for aggravated assault than the general population and that male patients had a significantly higher arrest rate for robbery. We question the validity of these findings on the basis of the fact that patients with a diagnosis of alcoholic intoxication accounted for a third of the arrests in their sample. Alcoholic intoxication does not qualify for the classification of mentally ill as the category is most commonly defined. Giovannoni and Gurel (12), who studied a population that was 95% schizophrenic, found that ex-patients had higher arrest rates for the crimes of homicide, assault, and robbery than the general population. However, they did not test the statistical significance of these differences, and the very small numbers involved (i.e., 3 homicides, 7 assaults, and 3 robberies) make statistical significance unlikely. Zitrin

and associates (13) found that the psychiatric patients in their study had arrest rates for violent crimes (i.e., rape, robbery, aggravated assault, and burglary) higher than the corresponding rates in their community. Once again, however, the differences were not tested for statistical significance, and the removal of data on subjects with a diagnosis of alcoholism or drug dependence reduces the arrest rates for the patients to a parity with community rates.

The consistency with which research has found mental patients to be as harmless as the general public is matched by the consistency with which these investigations have used arrest records as the operational definition of dangerousness and violence. Undoubtedly this operational definition covers the most serious fear-inspiring behaviors. However, there are many less serious acts of violence that are quite capable of inspiring fear. Clearly, the average person cannot be expected to remain calm and comfortable in the face of a threat, irrational anger, the destruction of objects, or mild physical assault. Moreover, such behavior, especially in someone termed "ill," seldom results in formal charges (14). Therefore, by investigating only extremes of violence, research on the dangerousness of the mentally ill limits itself to a partial answer to the question. This partial answer does not provide a scientific basis for declaring that the public's fear of the mentally ill is irrational.

In broadening our scope to include less serious acts of violence, our study found that 36% of 321 psychiatric admissions were preceded by some form of violent, fear-inspiring behavior. If criminal arrest had been used as the criterion of violence the rate of violent behaviors for this sample would have been 9 per 1,000 because only 3 patients in the sample had been arrested. Given our very broad operational definition of violence, however, the rate of violent behaviors was found to be 360 per 1,000: a fortyfold increase. These data provide no comparisons with the general population and therefore cannot substantiate the proposition that ex-mental patients are more violent than the general public. Nevertheless, these data do indicate that there may be considerable violence in behavior associated with mental and emotional breakdowns. Perhaps this is what the public responds to with fear.

The clinical experience of mental health professionals is also cited in support of the proposition that the mentally ill are not to be feared. The great majority of mental health professionals have never been injured by the people they treat (15). Indeed, skillful psychiatric personnel are able to handle patients' anger and irrationality with such efficiency that they are inclined to be puzzled or amused by the anxiety and clumsiness of the layman faced with such a situation. What is easily overlooked, however, is the supporting organization and the training and experience that make the professional's confident posture possible (16). If the clinician were to make a more complete statement on the matter, he or she would say that one need not fear the mentally ill *if* one learns to identify those whose condition leaves them vulnerable to violent outbursts, and *if* one learns how to handle that subgroup of vulnerable patients, and *if* one can count on assistance in an emergency.

Supporting data for the current posture of the mental health professions on the issue of public fear of ex-mental patients are sorely lacking. One cannot be at all certain on the basis of the evidence that community fear of the mentally ill is irrational. Indeed, the data of this study provide empirical support for the opposite view: that it is quite reasonable to fear the mentally ill. Therefore, we suggest that community education programs, which were designed to teach the public that there is no reason to fear the mentally ill, have failed because what they are teaching is not sufficiently accurate.

Community fear of the mentally ill clearly presents a problem. However, the problem may lie not in the fact that people fear mental patients but in the fact that the public fears them too much, under the wrong circumstances, or for the wrong reasons. Perhaps the solution to the problem lies not in telling the public that it is wrong to fear the mentally ill but in telling them to what extent ex-patients ought to be feared, under what circumstances or for what reasons they ought to be feared, or how one ought to respond when a patient is frightening. In order to provide such specific information, however, the area under investigation must be considerably broadened from its current narrow focus on arrest records.

If further research should show, as this study suggests, that there is some factual basis for fearing the mentally ill, it remains to be seen how this would affect the community approach to treatment. One can never be sure of such things, but being informed of a specific, realistic basis for fearing ex-patients might well do more to eliminate irrational fears than a hundred reassurances.

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APPENDIX 1
Categories of Violent Behaviors

Attacks on persons: specific mention of physical assault on people, including hitting, pushing, choking, etc.

Attacks on objects: specific mention of physical assault on nonhuman entities, including throwing things, breaking things, physically abusing animals, etc.

Threats to persons: specific mention of stated intention or described act that communicated the distinct possibility of inflicting harm on another person

Verbal attacks on persons: specific mention of verbal behavior that, although not explicitly threatening, was nevertheless pointedly abusive and hostile

Ambiguous violence: vague, nonspecific description of behavior as "violent," "aggressive behavior," "uncontrollable," "hostile," etc. (ranked below the others because of lack of specificity)

Loss of impulse control: behavior described by such words as "running wildly," "yelling," "shouting," etc., and giving no indication of this activity's being directed at persons or objects

Briquet's Syndrome, Hysterical Personality, and the MMPI

BY BARRY I. LISKOW, M.D., PAULA CLAYTON, M.D., ROBERT WOODRUFF, M.D., SAMUEL GUZE, M.D., AND ROBERT CLONINGER, M.D.

The authors compared the MMPI scores of 29 inpatients diagnosed by other authors as having hysterical personality and 21 outpatients diagnosed by the authors as having Briquet's syndrome. The two groups of patients differed significantly in age, on the MMPI lie scale, and on the MMPI hypochondriasis scale but not on any of the other MMPI scales. The authors discuss the implications of these findings for further defining the differences and similarities between hysterical personality and Briquet's syndrome.

THE CONCEPTUALIZATION in 1951 by Purtell and associates (1) of an idea suggested by Briquet's work on hysteria in 1859 and Saville's in 1909 has had an increasing influence on current concepts of hysteria. The work of Purtell and associates was resumed by Perley and Guze in 1962 (2) and has been continued to the present day by Guze and associates at Washington University, St. Louis, Mo. This group has carried out many studies using the following diagnostic criteria for hysteria: the presence of a complicated or dramatic medical history; onset before age 35; and a minimum of 25 out of 59 medically unexplainable symptoms spread across 9 out of 10 symptom groups (all of these symptoms must have interfered with the patient's ability to function) (3). With this definition, the Washington University group delineated the difference between Briquet's syndrome and isolated conversion symptoms, verified the diagnostic stability of Briquet's syndrome as demonstrated by follow-up studies (4), and related it to other psychiatric disorders through family studies (5–8).

Attacks on this concept of hysteria have come from various sources. The most predominant is disagreement over equating the historically broad concept of hysteria with the more precise but more narrow concept of Briquet's syndrome (9). This semantic confusion regarding the definition of hysteria has led the

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Washington University group in the last several years to use the term "Briquet's syndrome" in place of the term "hysteria" (4). In 1974 Chodoff, who is not from Washington University, suggested that Briquet's syndrome be considered separately from all other concepts related to hysteria (9). This would necessitate its separation from hysterical personality; hysterical neurosis, conversion type; and hysterical neurosis, dissociative type. According to the report of APA's Committee on Nomenclature and Statistics presented at the May 1975 annual meeting in Detroit, Mich., such a proposal was under consideration for *DSM-III*.

The separation of *DSM-II*'s diagnosis of hysterical personality (10) and Briquet's syndrome as distinct concepts has been seriously questioned. Kimble and associates found that 9 out of 10 women independently diagnosed as having hysterical personality also fulfilled criteria for Briquet's syndrome (11). They concluded that the *DSM-II* diagnosis of hysterical personality selected patients who were very similar to those diagnosed as having Briquet's syndrome. This similarity is not to be expected a priori, considering that hysterical personality is defined in *DSM-II* as "behavior patterns . . . characterized by excitability, emotional instability, over-reactivity, and self-dramatization . . . often seductive . . . immature, self-centered, often vain, and usually dependent on others" (10), and the definition of Briquet's syndrome is based on medical complaints without evident organic basis. Kimble and associates' findings suggest that the two diagnoses describe different phenomenological aspects of the same psychiatric illness.

Another approach to studying the relationship between hysterical personality and Briquet's syndrome is to give the same objective psychological test to groups diagnosed independently as having either hysterical personality or Briquet's syndrome and to look for similarities and/or differences in the results of the test. Slavney and McHugh (12) reported a study of 29 inpatients (26 women and 3 men) diagnosed as having hysterical personality. They administered the MMPI to these 29 inpatients. We compared Slavney and McHugh's MMPI results with our MMPI results for 21 patients diagnosed as having Briquet's syndrome. Our purpose was to determine whether the MMPI could be of value in defining the relationship between patients with hysterical personality and those with Briquet's syndrome.

METHOD

We contacted 29 patients diagnosed as having Briquet's syndrome in the Washington University Psychiatry Outpatient Clinic and asked them to come to the clinic to take the MMPI; each of the patients received $10. One of us (P.C.) reviewed each chart carefully and eliminated from the study all patients who were suspected of having another psychiatric illness or who did not meet the criteria for Briquet's syndrome. This process left 22 patients. One of these patients was eliminated because of failure to complete the MMPI, leaving a final total of 21 patients, all of whom were women. The MMPI data for these patients were compared with Slavney and McHugh's data on the MMPI's 3 validity scales and 10 clinical scales. We also compared the mean age of both groups of patients.

RESULTS

The ages and the MMPI K-corrected T scores for the 21 patients with Briquet's syndrome and for Slavney and McHugh's 29 patients with hysterical personality are shown in table 1. There is a significant difference in age between the patients with hysterical personality and those with Briquet's syndrome; the patients with hysterical personality were younger. Patients with Briquet's syndrome demonstrated abnormality (T score of 70 or above; i.e., T score of 2 standard deviations above normal) on 7 of the 10 clinical scales and 1 of the 3 validity scales. However, there were no significant differences on these scales between patients with Briquet's syndrome and patients with hysterical personality except for the hypochondriasis scale and the lie scale. This is of interest because the hypochondriasis scale is composed of 33 items, most of which relate to a variety of overt bodily complaints similar but not identical to the 59 symptoms used in making the diagnosis of Briquet's syndrome.

DISCUSSION

The fact that patients diagnosed as having Briquet's syndrome had markedly deviant MMPIs indicates the possibility of using the MMPI to screen for such patients. However, more work is needed to determine whether a stable MMPI pattern exists that can be distinguished from the pattern produced by other psychiatric disorders. The finding that Slavney and McHugh's patients with hysterical personality had deviant MMPIs similar to those of patients with Briquet's syndrome indicates the possibility that patients diagnosed as having hysterical personality are similar to or possibly indistinguishable from those diagnosed as having Briquet's syndrome. This possibility is given more credence by the study of Kimble and associates (11), which found that 9 patients with hysterical personality fulfilled criteria for Briquet's syndrome. Perhaps this finding should not be surprising, considering that in constructing the currently used criteria for Briquet's syndrome Perley and Guze (2) chose their subjects from a pool of patients who had been diagnosed as having hysteria, as had Purtell and associates (1).

The significant difference on the hypochondriasis scale between patients with Briquet's syndrome and those with hysterical personality suggests several pos-

TABLE 1
MMPI T Scores of 29 Patients Diagnosed As Having Hysterical Personality* and 21 Patients Diagnosed As Having Briquet's Syndrome

Item	MMPI Abbreviation	MMPI Code Number	Patients with Hysterical Personality		Patients with Briquet's Syndrome		Significance**
			Mean	SD	Mean	SD	
MMPI scale							
Lie	L		47.9	6.9	54.0	7.7	p<.005
Frequency	F		69.7	17.1	70.2	15.5	n.s.
Correction	K		52.0	9.7	50.3	4.7	n.s.
Hypochondriasis	Hs	1	63.6	12.9	75.4	11.0	p<.005
Depression	D	2	76.9	16.1	74.3	9.7	n.s.
Hysteria	Hy	3	70.4	10.1	73.2	9.8	n.s.
Psychopathic deviate	Pd	4	79.3	10.9	73.6	15.4	n.s.
Masculinity-femininity	Mf	5	48.3	15.9	52.3	13.8	n.s.
Paranoia	Pa	6	69.8	12.9	71.3	10.6	n.s.
Psychasthenia	Pt	7	73.1	12.2	72.2	12.4	n.s.
Schizophrenia	Sc	8	78.7	16.6	79.2	15.6	n.s.
Hypomania	Ma	9	66.8	12.9	60.3	12.9	n.s.
Social introversion	Si	0	61.5	13.4	63.9	12.1	n.s.
Age			27.9	10.4	38.4	13.2	p<.005

*From the data of Slavney and McHugh (12).
**By two-tailed t test; significance level used was .05.

sibilities. Patients with Briquet's syndrome might be a subgroup of the most seriously ill patients with hysterical personality or a subgroup of those whose emphasis on somatic complaints overshadows other aspects of their personality. Alternatively, in view of the significant age difference between the groups, the higher hypochondriasis score may reflect the natural progression of hysterical personality with increasing somatic complaints with increasing age. Age differences might also account for the significant difference in the lie scale.

These and other possibilities must remain speculative for the present, as must the precise relationship between hysterical personality and Briquet's syndrome. However, the MMPI may prove useful in resolving the problem noted by Kaminsky and Slavney of operationally defining nonmedical factors that might be useful in diagnosing and treating Briquet's syndrome (13). Finally, in a broader context, recent trends toward increasingly reliable psychiatric diagnosis might allow a more fruitful correlation of psychiatric diagnosis and objective psychological tests than has been possible.

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Psychiatric Observations of State and Local Politicians

BY EDWARD MESSNER, M.D.

The author, a psychiatrist and an elected official in a small town, compares his observations of local politicians with a mental health commissioner's observations of politicians at the state level. The author feels that local politicians tend to be more strongly oriented toward relatively altruistic public service. Several factors, including personal ambition and reduced public scrutiny, may contribute to the apparently greater selfishness of politicians at the state level.

POLITICS HAS attracted the professional attention of psychiatrists from several points of view (1). Greenblatt (2) drew upon his experience as commissioner of mental health in Massachusetts to provide us with some observations by a psychiatrist who had been called upon to exercise considerable power in a state agency. He called attention to the scarcity of information and training available to individuals who sought executive careers in psychiatry.

In "Psychopolitics" Greenblatt (2) urged psychiatrists who have served in "powerful positions in the psychopolitical arena" to report and analyze their experiences. His purpose was to "teach us how to behave when fate thrusts such critical roles upon us." His invitation was thus not intended for me; but I offer this article as a response because I have participated in a complementary facet of political psychiatry (3). The purpose of this article is to provide some comparisons, contrasts, and inferences that may add to the body of knowledge about the interface between psychiatry and politics.

PERSPECTIVE ON THE FIELD OF POLITICAL PSYCHIATRY

In a review of previous publications on political psychiatry (1), I suggested a tentative classification of works on the subject. It listed the following topics:

1. Psychiatrists and theories related to government politics.
2. Psychiatrists and political activity.
3. Psychiatrists and radical political activity.
4. Community psychiatrists and political action.
5. Psychiatrists and international politics.
6. Psychiatrists in elective public office.

Greenblatt's recently published articles (2, 4, 5) fit most closely with the fourth group of publications, community psychiatrists and political activity. The richness of detail drawn from his experiences certainly expands that category markedly, as does his evaluation and analysis of those experiences. It would be appropriate to rename that domain of political psychiatry "psychopolitics."

In "Towards a Definition of Psychopolitics," Greenblatt (4) suggested that

> it might embrace the following areas: 1. The personality and psychology of the politician; his orientation to mental health workers and systems. . . . 2. The politics of the mental health worker. . . . 3. The relationships between political and mental health systems. . . . 4. The development of the political psychiatrist.

The present article focuses upon the "personality and psychology of the politician."

OBSERVATIONAL PERSPECTIVES

My political observations have been made primarily in the town of Amesbury, Mass., a partly rural community with a population of about 14,000 persons, located 40 miles north of Boston on the border with New Hampshire. Its economy is relatively depressed, with rates of unemployment higher and average income lower than the national average. Its various ethnic, racial, and religious groups live together harmoniously.

My experience consists of nearly six years of service as an elected public official—a politician—while continuing the clinical practice and teaching of psychiatry. As a member of the school committee[1] of a small town in Massachusetts, I have been a part-time, unpaid elected official with relatively little power and influence. This is in contrast to the full-time, salaried, appointed state officials, who exercise much greater power and influence as commissioners of mental health.

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[1]In most states the equivalent elected government body is known as a school board or board of education.

I do not have enough information to compare Amesbury politically with other towns, but it is an environment markedly different from the state capital. Greenblatt observed officials at the state capital, most of whom are full-time, paid, career politicians. My observations have been of small-town, part-time, primarily unpaid politicians. Most of them earn their living in other occupations.

OBSERVATIONS

It is no surprise that these two markedly different positions would produce experiences and observations at great variance with each other. Yet common themes such as public service, adversary relationships, and exposure to the news media tie them together. Much of what Greenblatt wrote in "Psychopolitics" (2) corresponds with some of my own political experiences.

Greenblatt (2) described politicians by comparing them with his professional colleagues. He wrote that they are "generally more open about their feelings, more extroverted, more anxious to do a favor and earn gratitude" than are physicians. It is difficult for me to discern any general differences regarding the openness and feelings of local politicians as compared with psychiatrists I have known. They may be more extroverted than most psychiatrists in that they seem to be more willing to speak in public. Politicians are willing to campaign for election, sometimes door-to-door, and to make public a part of their private lives.

Politicians are probably asked to do favors (6) more often than psychiatrists are. My observation is that local politicians, as a group, are probably somewhat more explicit than my medical colleagues in expressing—and in acting to attain or reject—their likes and dislikes. The difference is probably not so great as with the politicians observed by Greenblatt.

Politicians in a small town exemplify a wide range of educational, occupational, economic, intellectual, and social backgrounds. That breadth is bound to account for some important differences between them and groups of psychiatrists or other physicians. The medical groups are skewed toward extensive education, high intelligence, and relatively high socioeconomic levels. Greenblatt (2) observed that our professional colleagues' lives tend to be relatively "restrained and refined"; they are "more serious-minded and . . . [their] conduct and actions are guarded, especially in the social sphere"; and they tend to be "more intellectual and objective." My observations are consistent with his in these areas, but I sense a difference in degree. The politicians with whom I have had contact come closer to being a random selection of people—average citizens—than does the group of state legislators and other officials observed by Greenblatt. The small-town politicians are less "interested in grabbing power"; less intensely inclined toward "personal aggrandizement"; less mired in "narrow partisanship"; and far less likely to practice "duplicity."

The foregoing comparisons are largely matters of degree. However, there are also qualitative differences. For example, I have not at all been struck by an "atmosphere of suspiciousness, the assumption that everyone is motivated by base personal desires" (2). On the contrary, I have often been impressed by the many instances in which my fellow townspeople have extended themselves to serve our community. Most of the elected officials with whom I have had contact seemed to feel a sense of duty and responsibility to their constituents. It is more than a matter of winning their votes. They seem to care a great deal about the town and its residents.

The political system at the local level is fundamentally competitive and operates on adversary principles, just as it does at the state level. I have not observed it to be "a political jungle," as Greenblatt has observed on the state level. There seems to be a strong sense of community interest among town officials, and this tends to override some of the rivalry and pursuit of personal interests in which politicians at the state level seem to engage.

On the surface it appears that state officials struggle for higher stakes—larger numbers—more people and more money. This is balanced on the local level by the fact that many issues are more direct, more personal, and therefore more intensely meaningful. For example, my own primary purpose in seeking election to the school committee was to try to improve the quality and effectiveness of our school system. My aim was (and is) motivated by my desire to provide the best possible educational environment for my children. This approach involves attempting to improve the opportunities available for all of the children in the town.

My interest in my fellow townspeople and their children has increased with time and with my service in public office. In addition, I have developed an increased sense of loyalty to the town as a whole. At times the work is stressful, with occasional public confrontations that can become quite intense. The work requires a great amount of time and study, much of which is devoted to contract negotiations and the development of budgets.

This public service is rewarding in many ways—intellectual stimulation in such fields as education, law, government, and finance and the satisfaction that comes with the accomplishment of something that one values. A special source of interest and satisfaction that it offers is the opportunity to apply my psychiatric and psychoanalytic training, experience, and skills to the field of elective politics. It can also offer opportunities for creativity, innovation, public attention, power, and influence.

PUBLIC SERVICE ORIENTATION

It is my impression that the politicians whom I have observed and with whom I have interacted are more dedicated, less selfish, and more consistently directed

toward the benefit of their constituents than are the politicians described by Greenblatt. I suspect that an actual difference exists even after taking into account possible bias by either or both observers and ambiguities of descriptive terms. This impression should be tested by the experience of other observers.

The difference, if it exists, may be based on less conflict between personal and constituents' interests on the local level. A politician's public service orientation (altruism) may be enhanced by or be in conflict with his personal adaptive needs (egoism). His political actions result from the influence of both the public and personal factors. If his career or livelihood is threatened, the politician is more likely to place his personal interests above the public benefit. Like most of my fellow politicians, I derive my income from sources other than the town treasury. This allows me the freedom to support, when necessary, issues that may be unpopular without having to worry about my family's financial security.

Most state and federal politicians start at the local level. Numerous factors of personality or life circumstances influence the local politician's decision to try to move on to the state level. For example, it has been suggested to me that I run for county commissioner or state representative. These positions do not appeal to me because they would drastically limit the time I could devote to psychiatry. More important, my children are still in school and much remains to be accomplished in terms of the quality of education in the local school system.

For someone who wants to make a career in politics, election to state office usually represents advancement. It also permits considerable latitude for political action away from the scrutiny of the electorate, especially in the intervals between election campaigns. Much can be obscured by the intricacies of legislative committee action and by the complexities of legislative parliamentary maneuvering. These shields are less available to the local official; thus self-indulgent behavior is more readily exposed and reproached.

A factor that exerts a strong influence on an elected official's public service orientation is his relationship with his constituents. This is an area that I have been privileged to observe from the inside. Some of my fellow politicians confide in me from time to time, and I can observe my own subjective experiences. In this process my psychoanalytic and psychotherapeutic experience has proven to be superbly useful.

The elected official's relationship with his constituents can be highly complex and variable. It can include fear, resentment, loyalty, affection, contempt, and envy—many of the emotions that human beings can feel toward one another. The elected official's feelings and their associated attitudes and impulses can fluctuate and vary with time. They are influenced by factors introduced by the constituents, the situation, and the politician himself.

The politician's relationship with the electorate at both the state and local levels is analogous to, and possibly more complicated and varied than, a physician's relationship with his patient (1). It contains transference and nontransference elements (7), countertransference and noncountertransference elements (8), and a profusion of situational factors.

It is this subject area that provides a particularly rich domain of study for the psychiatrist in elective office. Its exploration is beyond the scope of this article, but a basic hypothesis should be stated here. The elected official at any level of government, who is particularly vulnerable to the analog of countertransference reactions (1), may carry into his work some highly inappropriate and unconsciously determined impulses and actions. To what extent these might contribute to Greenblatt's "political jungle" remains to be discovered.

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This section contains reports of new clinical or laboratory observations, single cases of unusual interest or importance, descriptions of pilot studies, suggestions for future research, etc. Reports should be submitted in duplicate, typed double-spaced throughout, and may contain 1,000 words, no more than 1 table, and a maximum of 10 pertinent references. Figures cannot be used. Reports will be reviewed by at least one referee and the Editor; the author will receive a postcard informing him/her of the disposition of the submission. Reports will be edited for clarity and conformance with Journal *style.*

The B-Mitten EEG Pattern and Tardive Dyskinesia: A Possible Association

BY JAMES T. WEGNER, M.A., FREDERICK A. STRUVE, PH.D., AND JOHN M. KANE, M.D.

Tardive dyskinesia is a well-recognized complication of neuroleptic drug treatment (1). The different syndrome definitions, patient populations, and examining techniques that have been used preclude accurate prevalence determination, but estimates suggest that 3–6% of patients in mental hospitals and approximately 20% of elderly chronically institutionalized patients exhibit tardive dyskinesia related to neuroleptic drugs (2). Estimates of prevalence among long-term psychiatric patients range from 2.2% to 56% (3). Although numerous pharmacological approaches have been used, an established treatment for tardive dyskinesia has not emerged and efforts directed toward prevention are of enormous importance (4).

Factors potentially predisposing to tardive dyskinesia have been discussed previously (1). Most reported cases have been in schizophrenics, probably because such patients are exposed to neuroleptics over long periods of time. However, tardive dyskinesia also occurs in other patients who receive neuroleptic treatment, including those with affective illness, psychoneuroses, chronic pain syndromes, or personality disorders. Although prospective studies are lacking, there is no evidence that tardive dyskinesia occurs more often in patients previously manifesting acute drug-induced dyskinesias. One report (5) suggested that tardive dyskinesia occurs more often in patients who previously experienced drug-induced parkinsonism, but this may reflect exposure to higher doses of antipsychotic drugs. The association of tardive dyskinesia with preceding organic brain damage, ECT, frontal leukotomy, advanced age, female sex, and antiparkinsonian agents has been suggested, but substantiating epidemiological data are lacking.

Paulson (6) reported EEG findings in 30 patients (mean age=57 years; range, 34–80) with tardive dyskinesia resulting from neuroleptics. The EEGs were obtained after tardive dyskinesia was diagnosed. Seven patients had either diffuse or focal slowing, 4 had paroxysmal epileptiform bursts, and 19 were normal—although in 11 of these 19 the voltage averaged less than 20 microvolts. There was no matched control group without tardive dyskinesia in the study, and some of the EEG results (i.e., slowing) may reflect a somewhat higher incidence in older subjects in general. Gardos and associates (7) reported that abnormal EEG was one of five variables contributing to a discriminant function analysis that significantly identified tardive dyskinesia subjects. However, subanalysis of relative incidence of EEG abnormality in patients with and without tardive dyskinesia failed to reach significance. The types of EEG abnormality were not specified.

Subjects and Method

This report suggests an unusually high incidence of the B-mitten EEG dysrhythmia in patients with tardive dyskinesia. Twenty patients who had waking and sleeping EEG tracings were identified. The sample consisted of 13 male and 7 female patients with a mean age of 29.8 years (range, 16–66). The diagnoses included schizophrenia, N=17; bipolar affective illness, N=2; and unipolar manic depression, N=1. The mean total time of exposure to neuroleptic medication was 2.64 years (range, 6 months–5 years), and the

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The authors wish to thank Frederic Quitkin, M.D., and Arthur Rifkin, M.D., of the Department of Psychiatry, College of Physicians and Surgeons, Columbia University, and the New York State Psychiatric Institute, for their assistance in diagnosing tardive dyskinesia.

TABLE 1
Appearance of Mittens and Adequacy of Sleep Depth for Detection

Appearance of Mittens	Adequacy of Sleep	
	Yes	No
Yes	10	0
No	1	9

mean highest dose of neuroleptic medication (in chlorpromazine equivalent) was 1006.1 mg (range, 100–2500 mg). The medications were as follows: chlorpromazine, N=9; fluphenazine, N=6; thioridazine, N=3; perphenazine, N=1; haloperidol, N=1.

Results

Table 1 shows the B-mitten EEG findings cross-tabulated with adequacy of sleep depth for B-mitten detection. Of the 9 patients who had inadequate sleep level for mitten detection, 4 showed EEG abnormalities other than the B-mitten pattern. While 70% of the tardive dyskinesia patients had some positive EEG finding, the most striking feature of these data is the exceedingly high incidence of the B-mitten pattern. It must be stressed that the mitten pattern can *only* be detected in moderately deep and deep sleep EEG recording (stages III and IV). Mittens cannot be seen while the subject is awake, drowsy, or in light spindle sleep. Overall, 50% of the tardive dyskinesia patients displayed mittens; of the 10 patients without this EEG finding, 9 had EEGs without sufficient sleep depth and are thus potentially "false negatives" concerning the mitten signal. When the mitten incidence is corrected for sleep adequacy, it is seen that of the patients with adequate deep sleep, 90.9% displayed the mitten pattern.

For 3 patients, mittens were recorded at least 12 months before the onset of tardive dyskinesia and for 2 others the mitten signal was recorded after tardive dyskinesia was diagnosed but at a time when tardive dyskinesia symptoms had reversed and were no longer present. Five patients had symptoms of tardive dyskinesia at the time mittens were recorded. It is possible but not likely that B-mittens are merely a concomitant physiological sign of symptomatic tardive dyskinesia.

Discussion

The B-mitten pattern was originally described by Gibbs and associates (8). A condensed review and complete reference list for this pattern has been provided by Struve and Klein (9). In summary, B-mittens are rare among normal controls and general medical referrals (i.e., those without psychiatric complaint) and occur almost exclusively within psychiatric populations. Mittens are only recorded during deep sleep and are seen best in monopolar recordings where the pattern occurs symmetrically over the bilateral frontal or frontoparietal areas. Several lines of evidence, including tumor studies (10), converge to suggest that B-mittens are subcortical in origin. Recent studies by Struve and Klein (9) have provided evidence that this dysrhythmia may have a highly significant association with symptoms of affective dysregulation (primarily dysphoric) that cuts across broad diagnostic categories.

If our preliminary findings are confirmed, this EEG pattern could be used to identify patients at high risk for tardive dyskinesia, and prophylatic efforts could be made early in treatment. Why mittens may relate to tardive dyskinesia is obscure, and the role, if any, that mittens play in the pathophysiology of this condition is unknown. It is intriguing to speculate that the subcortical mitten dysrhythmia may signal a vulnerability to dopaminergic dysregulation within nigrostriatal pathways—perhaps potentiating dopamine receptor hypersensitivity secondary to prolonged neuroleptic blockade. The association of the mitten pattern with affective dysregulation may also suggest that patients with affective symptoms are more susceptible to tardive dyskinesia. The fact that different biogenic amine system abnormalities are putatively involved in affective illness (norepinephrine and serotonin) and schizophrenia (dopamine) could suggest different susceptibilities to the development of tardive dyskinesia. However, mitten pattern-neuroleptic interactions in tardive dyskinesia development are likely to be complex and our understanding of such relationships confined to mere speculation for a considerable time.

Currently, we are selecting a control group of patients who do not have tardive dyskinesia with deep sleep EEGs to be matched with tardive dyskinesia patients on a number of relevant variables. Furthermore, a prospective study, which is presently being planned, will further clarify the potential of the B-mitten patterns for predicting patients at high risk for tardive dyskinesia.

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Rapidly Recurring Seizures of Psychogenic Origin

BY KUTTIKATT P. POULOSE, M.D., F.R.C.P. (C), AND ARTHUR A. SHAW, PH.D.

Seizure of psychogenic origin is a well-recognized entity. However, repeated "seizures" that outwardly simulate status epilepticus are rare. It is important to recognize these cases in order to avoid administration of unnecessary and large doses of anticonvulsant medications with their attendant risks. A case we recently confronted illustrates some of the problems in the recognition and management of this uncommon problem.

Case Report

Mr. A, a 50-year-old man, was transferred from the psychiatry service to the neurology ward on July 14, 1975, for control of "seizures." He had a background of "seizures" over a period of 20 years for which he receives disability compensation and was under treatment with diphenylhydantoin, 100 mg t.i.d., and phenobarbital, 30 mg t.i.d. Mr. A also had multiple hospitalizations for excessive drinking. His seizures were characterized by staring, followed by a cry and stiffening of the whole body, sometimes with arching forward but not followed by a clonic phase. He appeared quite clear headed soon after the seizures, even after repeated episodes, and would joke with nursing personnel. He had about 50 seizures each day in rapid succession. During some attacks he threw himself off his bed but was not hurt. He often expressed concern for the demands in time and effort his condition caused the nursing personnel and physicians.

A skull series and brain scan were normal, as were blood count, BUN, creatinine, total proteins, and electrolytes. Chest X ray revealed no abnormalities. Random blood glucose was 230 (he was on I.V. dextrose). An EEG done on two occasions when he was seizure-free for brief periods showed no abnormalities, as did an EEG recorded while the patient was in a "seizure" episode. ECG was normal.

He was treated with I.V. diazepam, I.M. phenobarbitone, and paraldehyde during his first 3 days of hospitalization. His seizures continued in spite of these medications. He was also placed on small liquid feedings between seizure episodes. On the fourth day the patient insisted on solid food. He was told that with frequent seizures, introduction of solid food can result in aspiration and therefore he could be given solid food only after his seizures ceased. The seizures promptly subsided and he was seizure-free thereafter until his discharge from the hospital 10 days later at his own request. An evaluation by the psychology service reinforced the impression that the patient had characterological problems (manipulative). The psychologist's impression was that the seizures were less likely to be hysterical than consciously controlled.

Discussion

The dramatic presentation of this case, with repeated uncontrolled seizures, led us initially to consider status epilepticus. However, several aspects of this case contraindicated the diagnosis of a true seizure disorder. Epilepsy is characterized by intense cerebral neuronal activity followed by postictal metabolic cerebral depression of varying degrees and duration (1). Postictal mental cloudiness is commonly observed with grand mal seizures, particularly when the seizures occur in rapid succession, when patients are often comatose following the seizure episode (2). The mental clarity of our patient and his jocularity between seizure episodes would have been impossible to explain had these episodes been true seizures. The three EEGs recorded during his hospitalization, one during an episode of "seizure" activity, showed no abnormalities. Although not definitive, this is a strong argument against a diagnosis of true seizures.

Although the EEG may help in differentiating true epileptic seizure from hysterical seizures, it is unfortunately true that epileptic attacks may occur without evident changes in the scalp recording. This situation is frequently seen in Jacksonian epilepsy, where one presumes that the focus is so discrete and the discharge of such low amplitude that it fails to be conducted to the surface electrodes (3). This patient's desire to have a regular diet was so intense that the suggestion of regular diet as a reward for cessation of seizures was effective. The question of conversion reaction versus a conscious process is relevant in this

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case, but the two etiologies have much in common. The difference is largely one of the relative degree to which consciousness participates in the reaction. Discrepancies, contradictions, and exaggerations of symptoms are more frequent in malingering. The malingerer commonly expresses much concern about his symptoms, and results of the Minnesota Multiphasic Personality Inventory may suggest a tendency toward lying and deception. In conversion hysteria, patients manifest little concern about their symptoms. In cases of malingering, surprise examination may unmask the deception. Conscious and unconscious factors are interwoven in malingering (4).

This case illustrates the point that uncritical acceptance of previously documented "seizure disorder" may help mask its true nature.

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Mother-Son Folie à Deux: A Case of Attempted Patricide

BY LANDRUM S. TUCKER, JR., M.D., AND THOMAS P. CORNWALL, M.D.

Since it was first labeled by Lasegue and Falret (1) 100 years ago, folie à deux has been a rare and interesting phenomenon. Gralnick (2) stressed the importance of folie à deux in understanding psychoses and interpersonal relationships. Greenberg (3) summarized data on the antisocial sequelae of such relationships—crimes committed by a folie à deux pair. In relation to this, Pulver and Brunt (4) speculated on the handling of hostility by projection in cases of folie à deux. The following case report describes a crime committed by a mother and son.

Case Report

Bobby A is a 10-year-old boy who was admitted to a juvenile psychiatric unit after he had been detained for allegedly burning down his father's house.

Two years before this admission, Bobby's parents had been divorced after a stormy marriage of 18 years. Ms. A's mother had been diagnosed as paranoid schizophrenic and her older brother was felt to be "somewhat strange." She had always felt "very close" to her mother. Her father was a "hard-working" man who was away from home often, including an absence of several years during Ms. A's adolescence when he was out of the country on a "business assignment." During that time he became extremely ill and almost died. Ms. A remembers many fantasies of becoming a doctor at that time.

When Bobby was born, Ms. A felt "abandoned" by her husband because he was away on business just as her father had been. She was depressed for some time after Bobby was born. Bobby was a healthy child, met all developmental milestones normally, and did well during his early grade school years. When Bobby was 8 his parents separated and divorced, and he and his mother moved to a town near by.

Six months prior to admission Ms. A and Bobby moved to the west coast, where they lived in various cities for short periods and kept to themselves. She began to tell Bobby that his father wanted to kill them. She started staying up all night with Bobby, telling him that Mr. A had put razor blades in their beds, cut her hair, and poisoned their pets. Bobby did not believe his mother at first since he knew his father was living across the country, but he was afraid to disagree for fear that his mother might hurt him. Slowly Bobby began to feel his mother was right and they began to plot to murder Mr. A. They decided to burn down his home while he slept. After several months of planning, Bobby flew alone across the country to his father's hometown and set his father's house ablaze that night. Mr. A barely escaped in his night clothes and Bobby was found by the police walking away from the flames. Ms. A returned to the state and was placed in a mental institution.

When Bobby was seen initially he was very anxious, with poor eye contact and loose associations. His initial themes in therapy centered around a drawing he did of a spaceship heading toward a large sun. From

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the ship came a cord that attached to a smaller ship. Bobby said the smaller ship went ahead of the big ship to check things out. He was concerned that the big ship's outer shell was melting away. The mission of these ships was to destroy the sun, but they were being destroyed. Bobby wished the ship could get back to earth safely.

In addition to individual therapy Bobby was treated by therapeutic milieu, including school and recreation. One especially important factor in his treatment, a point that has been stressed before (5), was separation from his mother. On the ward Bobby was initially overcontrolled, polite, and bright (his IQ was 112). In the first few weeks he was inadvertently allowed to receive two letters from his mother, and at those times he again manifested severe anxiety, poor eye contact, and loose associations.

After 2 months the themes in Bobby's play began to change. He began recurrent battles using a giant soldier from outer space who came with a flame thrower to destroy a "special" house full of smaller soldiers. He began to talk directly of his fear of his mother and his disbelief that he could have had the "crazy" thoughts about his father being so bad. At this time his thoughts were organized and he was relaxed and more open with his feelings.

At the end of 3 months, Bobby had shown no overt psychosis for several weeks and so, after careful consideration, he was sent home to his father. His father had worked closely with us, including participation in once weekly individual therapy. Themes in the 9 months of Bobby's outpatient therapy since discharge have moved from the earlier battles to playing checkers and talking about school and activities. Bobby also has begun writing songs. His first one said, "I'm lonely since I left you—but I've got my feet back on the ground."

Bobby is doing above average work in school, participates in sports, has made friends, and gets along well with his father. His mother remains hospitalized.

Discussion

Past efforts at explaining criminal activities involving a folie à deux situation have focused on 1) interpersonal regulation of hostility by projection, and 2) imposing of delusions by a psychotic, more intelligent, dominant partner on a less intelligent, passive partner who identifies with the dominant one. Certainly, some of this is evident here. Another factor in his case is a "fit" in the dominant and passive partners' unconscious motivations—the wish to kill the father figure in order to possess the mother. Bobby wished to destroy his father to win an oedipal victory (possession of his mother), while Ms. A., so strongly attached to her mother psychologically, wished to destroy her father symbolically to gain a negative oedipal victory (6). In the regression to psychosis both acted on their unconscious oedipal wish to destroy the father figure, while the conscious delusion was that he wanted to destroy them. Further case studies are needed to determine whether the concept of a "fit" in unconscious wishes can add to our understanding of folie à deux and crimes related to the syndrome.

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A Case of Lycanthropy

BY HARVEY A. ROSENSTOCK, M.D., AND KENNETH R. VINCENT, ED.D.

Lycanthropy, a psychosis in which the patient has delusions of being a wild animal (usually a wolf), has been recorded since antiquity. The Book of Daniel describes King Nebuchadnezzar as suffering from depression that deteriorated over a seven-year period into a frank psychosis at which time he imagined himself a wolf. Among the first medical descriptions of lycanthropy were those of Paulus Aegineta during the later days of the Roman Empire (1). In his description of the symptom complex, Aegineta made reference to Greek mythology in which Zeus turned King Lycon of Arcadia into a raging wolf. Thereafter, references to lycanthropy appeared in the ancient literature. Many medieval theologians envisioned lycanthropy as a consequence of the evil eye (2).

Delusions of being a wolf or some other feared ani-

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mal are universal and, although rare in the industrialized countries, still occur in China, India, Africa, and Central and South America (3, 4). The animals in the delusional transformation include leopards, lions, elephants, crocodiles, sharks, buffalo, eagles, and serpents (3, 4).

Not infrequently, bizarre and chaotic sexuality is expressed in a primitive way through the lycanthropic symptom complex. Patients whose internal fears exceed their coping mechanisms may externalize them via projection and constitute a serious threat to others. Throughout the ages, such individuals have been feared because of their tendencies to commit bestial acts and were therefore hunted and killed by the populace. Many of these individuals were paranoid schizophrenics (2).

Case Report

A 49-year-old married woman presented on an urgent basis for psychiatric evaluation because of delusions of being a wolf and "feeling like an animal with claws." She suffered from extreme apprehension and felt that she was no longer in control of her own fate; she said, "A voice was coming out of me." Throughout her 20-year marriage she experienced compulsive urges toward bestiality, lesbianism, and adultery.

The patient chronically ruminated and dreamed about wolves. One week before her admission, she acted on these ruminations for the first time. At a family gathering, she disrobed, assumed the female sexual posture of a wolf, and offered herself to her mother. This episode lasted for approximately 20 minutes. The following night, after coitus with her husband, the patient suffered a 2-hour episode, during which time she growled, scratched, and gnawed at the bed. She stated that the devil came into her body and she became an animal. Simultaneously, she experienced auditory hallucinations. There was no drug involvement or alcoholic intoxication.

Hospital course. The patient was treated in a structured inpatient program. She was seen daily for individual psychotherapy and was placed on neuroleptic medication. During the first 3 weeks, she suffered relapses when she said such things as "I am a wolf of the night; I am a wolf woman of the day . . . I have claws, teeth, fangs, hair . . . and anguish is my prey at night . . . the gnashing and snarling of teeth . . . powerless is my cause, I am what I am and will always roam the earth long after death . . . I will continue to search for perfection and salvation."

She would peer into a mirror and become frightened because her eyes looked different: "One is frightened and the other is like the wolf—it was dark, deep, and full of evil, and full of revenge of the other eye. This creature of the dark wanted to kill." During these periods, she felt sexually aroused and tormented. She experienced strong homosexual urges, almost irresistible zoophilic drives, and masturbatory compulsions—culminating in the delusion of a wolflike metamorphosis. She would gaze into the mirror and see "the head of a wolf in place of a face on my own body—just a long-nosed wolf with teeth, groaning, snarling, growling . . . with fangs and claws, calling out 'I am the devil.' " Others around her noticed the unintelligible, animal-like noises she made.

By the fourth week she had stabilized considerably, reporting, "I went and looked into a mirror and the wolf eye was gone." There was only one other short-lived relapse, which responded to reassurance by experienced personnel. With the termination of that episode, which occurred on the night of a full moon, she wrote what she experienced: "I don't intend to give up my search for [what] I lack . . . in my present marriage . . . my search for such a hairy creature. I will haunt the graveyards . . . for a tall, dark man that I intend to find." She was discharged during the ninth week of hospitalization on neuroleptic medication.

Psychological data. On the Wechsler Adult Intelligence Scale, the patient's performance showed normal intellect; the subscale configuration was devoid of behavioral correlates associated with organicity, as was the Bender Motor Gestalt Test. On the Holtzman Ink Blot Technique, the performance was indicative of an acutely psychotic schizophrenic with distorted body image and gross sexual preoccupation The Lovinger Sentence Completion Blank was corroborative. The Minnesota Multiphasic Personality Inventory was interpreted as showing an acute schizophrenic reaction with evidence of obsessional thinking, marked feelings of inferiority, and excessive needs for attention and affection.

Discussion

We believed that the patient suffered from chronic pseudoneurotic schizophrenia. What is of particular interest is that the delusional material was organized about a lycanthropic matrix. Her symptom complex included the following classic symptoms:

1. Delusions of werewolf transformation under extreme stress.
2. Preoccupation with religious phenomenology, including feeling victimized by the evil eye.
3. Reference to obsessive need to frequent graveyards and woods.
4. Primitive expression of aggressive and sexual urges in the form of bestiality.
5. Physiological concomitants of acute anxiety.

These symptoms occurred significantly in the absence of exposure to toxic substances. Furthermore, the patient responded to the treatment protocol used for acute schizophrenic psychosis. After reviewing ancient and modern literature, it is felt that the differential diagnosis for lycanthropy should include consideration of all of the following possibilities: 1) schizophrenia, 2) organic brain syndrome with psychosis, 3) psychotic depressive reaction, 4) hysterical neurosis of the dissociative type, 5) manic-depressive psychosis, and 6) psychomotor epilepsy. The last item is mentioned because of reports that individuals suffering from lycanthropy have been described as being

"prone to epilepsy" and suffering from intercurrent amnestic episodes (3).

A search of modern literature[1] produced three cases. In two cases (4), the patients were ultimately diagnosed as having paranoid schizophrenia, facilitated by involvement with hallucinogenic drugs, and chronic brain syndrome with periodic psychoses. In the third case, described by Morrell in 1852, it seems that the patient suffered from a deteriorating psychotic depression (5).

We believe that the metamorphosis undergone by the patient we have described provided temporary relief from an otherwise consuming sexual conflict that might have taken the form of a completed suicide.

Lycanthropy is a rare phenomenon, but it does exist. It should be regarded as a symptom complex and not a diagnostic entity. Furthermore, although it may generally be an expression of an underlying schizophrenic condition, at least five other differential diagnotic entities must be considered.

[1]MEDLARS II and Psychological Abstracts.

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Biofeedback Treatment of Tardive Dyskinesia: Two Case Reports

BY HELEN ALBANESE, M.D., AND KENNETH GAARDER, M.D.

Tardive dyskinesia, a movement disorder associated primarily with neuroleptic therapy, is characterized by constant, stereotyped, involuntary movements of mouth, lips, and tongue, sometimes accompanied by choreiform movements of limbs and trunk. Movements cease during sleep and are more prominent when the patient is anxious. Patients are sometimes able to control the movements voluntarily (1, 2). Biofeedback, in which the patient receives immediate physiological information about his movements, may assist patients to learn symptom control. This hypothesis borrows from the finding that similar movement disorders can in some cases be relieved by biofeedback. Farrar (3) has reported the use of biofeedback to treat orofacial dyskinesias that might represent the same syndrome, and Korein and associates (4) used biofeedback to treat torticollis.

In this paper we will describe the cases of two well-motivated patients with tardive dyskinesia who were given biofeedback training.

Case Reports

Case 1. A 39-year-old stockroom manager with two children had an acute paranoid psychosis a few months after his wife divorced him. He was given trifluoperazine to control his psychotic symptoms and benztropine mesylate to control his pseudoparkinsonian symptoms. Because of his failure to take medication and continued psychotic symptoms, his medication was changed to I.M. fluphenazine decanoate 11 months later. Eight months after the initiation of fluphenazine therapy he developed involuntary chewing movements. The dosage of medication was reduced and the fluphenazine was discontinued after a year. The oral movements improved only slightly. One month after discontinuation of medication, the patient was evaluated for biofeedback training to control the movements. He was free of psychotic symptoms but continued to have almost constant contorting and chewing movements of the mouth, some foot tapping, and slight choreiform movements of the fingers. A baseline psychophysiological profile (5), including EEG, galvanic skin response, frontalis muscle contraction, respiration, and heart beat, showed no physiological evidence of heightened general tension or anxiety. We decided to work directly on masseter muscle movement with electromyogram feedback.

Two bipolar skin electrodes were placed on the long axis of the right masseter muscle. Input from the muscle was fed to an electronic apparatus which then gave three types of feedback to the patient: 1) a sound, with pitch that increased with increasing tension of the muscle, 2) a threshold-controlled beep-like sound signaling the same input, and 3) visual feedback via a meter in front of the patient. The patient was instructed to

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eliminate the beep-like sound, to diminish the sound pitch level, and lower the meter reading. He was encouraged to become aware of sensation in the muscle when he was successful in recapturing this feeling at home. Sessions were held in the laboratory for 45 minutes once a week. Within 3 weeks the patient was able to eliminate movements of the mouth for 15 minutes during the laboratory session. By the 7th session he was able to complete the entire 45-minute session without moving his mouth, and he also had noted marked diminution of mouth movements at home. Movements of feet and fingers decreased markedly also. The 9th session was held after a 2-week interval and the 10th session after a 3-week interval. The patient was totally without dyskinetic symptoms 3 months later.

Case 2. A 51-year-old married farmer, the father of five children, had had several episodes of psychotic depressive illness as an adult. For his last episode, he received antidepressant medication and I.M. fluphenazine decanoate. He developed pseudoparkinsonism immediately, and tardive dyskinesia appeared 3 months after fluphenazine was started. The fluphenazine was discontinued immediately upon the appearance of the dyskinesia, but there was no improvement after a month. At this point the patient was evaluated for biofeedback training.

When we saw him, the patient exhibited constant, severe chewing movements and rhythmic protrusion of the tongue. There were no movements of the extremities. His psychophysiological profile showed physiological signs of tension. Direct work with the right masseter muscle was undertaken, and the patient was trained for 45 minutes once a week with the biofeedback system described above. By the 3rd session he was able to hold his mouth still for 7 minutes in the laboratory. By the 7th session he was movement-free for 20 minutes in the laboratory and reported diminished movements at home. During the 9th session, the only abnormal movement was of the tongue within the closed mouth. At this point his wife became ill and the family moved out of town. No follow-up data are available.

Discussion

The two patients described were both intelligent people who experienced social embarrassment because of severe mouth movements. The first patient had the movements for 5 months before biofeedback training; the second had the movements for only 1 month. Both had been off medication for 1 month without change in movements and improved markedly by the 3rd session of the biofeedback training. A characteristic of tardive dyskinesia is that voluntary quieting of movement in one part of the body often results in shifting of movement to another part of the body. This did not occur in either patient, despite the already existing foot and finger movements in the first patient described. A general reduction of tension was experienced by both patients, and both gave reports of increased well being.

Since spontaneous remission occurs in tardive dyskinesia, it cannot be proven that the biofeedback treatment was responsible for the marked improvement of these two patients. However, the improvement was first noted within training sessions and increased progressively with training, later extending to periods outside the training sessions. Also, in each instance there had been no noticeable improvement before treatment began. We believe this observation warrants further investigation of the treatment approach, and we hope that this report will stimulate others to try similar methods.

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The State Hospital: Facility of Last Resort

BY H. RICHARD LAMB, M.D.

The future of state hospitals has been the subject of continuing debate: Should they be phased out and, if so, how (1)? Are they in fact being phased out (2)? What alternative or additional functions should they assume (3, 4)? The California experience is instructive, for there have been unforeseen, although in retrospect not surprising, effects on the state hospitals as the result of attempts to reduce their use. In recent years there have been intensive efforts to explore and exhaust every local alternative before sending any patient to the state hospital. This has been due largely to a combination of pressure and financial inducements from the California state government (5).

Method

In a previous study (6), a sample of long-term psychiatrically disabled patients in the community was defined by the following criteria: functional psychotic diagnosis, 18–64 years of age, on Supplemental Security Income (SSI) for at least 18 months, and residence in San Mateo County (total population 550,000). It was found that the total group of such patients (488) contributed only 20 patients (8%) of the 264 San Mateo County residents who had been sent to the state hospital over a 2-year period ending March 31, 1975. (The figure 264 is an unduplicated count—the total number of admissions during this period was 421 as indicated in data provided by the county mental health services.) This seemed a remarkably small percentage considering the severity and duration of illness of this sample. If not this sample, then who was going to the state hospital? This question led to a decision to review the records of all persons 18 years of age or older sent to the state hospital from San Mateo County during the last months of the study period (December 1, 1974, to March 31, 1975).

Results

In the survey of persons sent to the state hospital there was a total of 92 persons (unduplicated count) during a 4-month period. Of these, 20 were out-of-county residents who, in most cases, were sent to the state hospital rather than a local hospital because the county did not want to pay for the hospitalization of nonresidents. These 20 were dropped from the sample, giving us a sample of 72 San Mateo County residents sent to the state hospital.

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TABLE 1
Recent History of Physical Violence

Physical Violence	Men		Women	
	Number	Percent	Number	Percent
In community	28	82	3	33
In local facility	6	18	6	67
Total*	34		9	

*Significantly different, $p<.01$.

Twice as many men as women were sent to the state hospital during the 4-month period studied. Median ages were 24.5 years for men (range, 18–47) and 33 years for women (range, 19–64). It should be noted that the oldest individual in the sample was 64. Most noteworthy is the fact that 77% of the men were under 30 compared with only 29% of the women.

A recent history of physical violence (within the past 2 years) in the community was reported in 43% of the sample. An additional 17% were described as being physically out of control and unmanageable by the local facility that transferred them to the state hospital. Thus issues of violence and control involved 60% of the sample; a higher proportion of the men were in this group (71% versus 37.5%). Table 1 reveals that over four-fifths of the men with a recent history of physical violence had been violent in the community before being evaluated at a local facility, as opposed to only one-third of the women.

Discussion

In California it is required that local (county) mental health facilities screen all persons for whom hospitalization may be necessary and treat them locally if possible. Under these circumstances, who goes to the state hospital? Our study of state hospitals admissions indicated that twice as many men as women are being sent to the state hospital. Almost three-fourths of these men are characterized by a history of physical violence and/or a feeling on the part of local facilities that they cannot control them. The same is true, although to a much lesser extent, of the women. We have also seen that three-fourths of the men are under 30 years of age. Our results (table 1) further suggest that professionals' fantasies and fears of young male patients with a history of physical violence influenced the decision to send them to the state hospital instead of trying to treat them locally.

Our findings, then, show that a major function of the state hospital for a California county of over a half million people is to handle young men who have a history

of physical violence or are thought to be physically out of control, persons with whom local facilities cannot or choose not to deal. (This is not true for the large metropolitan areas of Los Angeles, San Francisco, and Oakland, where large numbers of patients are inadequately screened with regard to their appropriateness for local treatment and are admitted directly to state hospitals.) The result is that as local facilities proliferate state hospital usage shrinks, and dealing with persons who are violent, or thought to be, becomes a primary function of what was once an institution with a wide variety of uses. The state hospital, more than ever, has become a facility of last resort.

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Covert Maternal Deprivation and Pathological Sucking Behavior

BY WILLIAM G. SLAUGHTER, M.D., AND CHARLES K. CORDES, M.D.

The varieties of lifelong unhealthy psychological consequences related to inadequate mothering care during the first year of life are well documented (1–5). Prolonged maternal deprivation occurring in an intact family was termed "masked" or covert deprivation by Prugh and Harlow in 1962 (6). Such deprivation can give rise to clinical symptomatology in children that approaches and perhaps equals in severity those syndromes which are usually associated with complete deprivation (separation).

There is now evidence suggesting that some effects of covert maternal deprivation are more immediately apparent than has been previously noted. This report adds to that evidence.

Case Report

George, at the age of 19 weeks, was admitted to the pediatric service of an Army general hospital because of a one-week history of low-grade fever and increasing irritability. His left index finger was found to be "swollen and erythematous with bluish discoloration of the distal tip." There were no other positive physical findings: complete blood count, urinalysis, platelet count, and reticulocyte count were normal, and uric acid was 5.2 mg/100 ml. X rays of the chest and both hands revealed no abnormalities. George had been sucking his index fingers intermittently for several weeks, but his parents had noted that he had started sucking longer and harder. Two days after admission, George was discharged with a diagnosis of "traumatic necrosis of the left index fingertip, secondary to self-mutilation." The parents were instructed to use elbow restraints whenever he was not closely attended, and he was scheduled for outpatient follow-up in the pediatric clinic.

One week after his discharge he was readmitted to the hospital. The left index fingertip was black and necrotic. There was no evidence or history suggestive of physical abuse, and exhaustive laboratory testing revealed no abnormality. Cultures taken from the dry gangrenous area grew out "heavy diphtheroids." George had a course of oral penicillin and was hospitalized for 4 weeks. He was cared for by several members of the ward staff and received a quality of warmth and sustained attention that amounted to multiple mothering. During the course of his four-week hospitalization, there was a gradual but marked decrease in the frequency and intensity of his attempts to suck his index fingers.[1]

George was the product of an unwanted, unplanned pregnancy. His 19-year-old mother was frightened and anxious when she learned she was pregnant. Her marriage to George's 20-year-old father had been arranged hastily. During the pregnancy, she felt alone and helpless. In the 36th week of an otherwise uncomplicated pregnancy, the membranes spontaneously ruptured, and the progress of her labor was such that a cesarean section was performed. She delivered a healthy male child who weighed 3,280 g and was 50.5

Dr. Slaughter is Lieutenant Colonel and Dr. Cordes is Colonel, United States Army Medical Corps. When this work was done, Dr. Slaughter was Senior Fellow in Child Psychiatry, Letterman Army Medical Center, Presidio of San Francisco, Calif., where Dr. Cordes is Chief, Child Psychiatry Service. Dr. Cordes is also Assistant Clinical Professor of Psychiatry, University of California Medical Center, San Francisco, Calif. Dr. Slaughter is now Chief, Community Mental Health Activity, Dwight David Eisenhower Army Medical Center, Fort Gordon, Ga. 30905.

[1]The swelling and inflammation of the proximal portion of the left index finger resolved; the gangrenous tip was allowed to fall off (at age 28 weeks) approximately 1 cm proximal to the nail bed. The stump was functional and clean.

cm in length. Her postoperative course was complicated by pneumonia and a partial wound separation that responded to routine and conservative care. She was discharged 13 days after the cesarean section.

Neither parent felt capable of caring for George when he returned home with his mother. Mother felt depressed, overwhelmed, alone, and helpless in facing what she considered to be an insurmountable and unending task. She did not enjoy caring for her son and felt rejected by him. She spent her waking hours watching television or daydreaming. She never left the apartment unless her husband was with her because she was afraid of being mugged or attacked. The father worked from early morning until late afternoon. He resented and refused his wife's requests that he help care for George. The couple had little money and could not afford recreation. Tension mounted—each spouse felt bitter and cheated by the other. Brief, violent verbal fights followed by long silences were common.

Father reported feeling inadequate to care for his depressed and angry wife, a "screaming baby," and his job. Both parents felt that the doctors had failed to give them adequate instructions in child care or sufficient follow-up care, and they were relieved when George was hospitalized at 19 weeks of age. The admitting pediatrician referred this case to the child psychiatry service after consultation with other services (examinations done on dermatology, neurology, general surgery, and orthopedic services revealed no abnormalities).

Following an evaluation of the family, both parents agreed to begin therapy. Each was seen individually once a week, and George was observed with his mother and/or his father each week. In the course of her therapy, mother was able to make a number of positive shifts and began to feel better about herself, her husband, and her son. She noted that in the past she seldom looked directly at her son and that she now enjoyed watching him and finding that he was also looking at her. Slowly, and much to her surprise, she began to enjoy caring for her son. She liked the weekly visits from a public health nurse and a VISTA couple that had been arranged through consultation with the hospital social work service. She took George with her on walks and visits to newfound friends and spent less and less time watching television and daydreaming. The father was able to be more helpful and began to spend time playing with George while mother prepared meals. As their own anxiety lessened, the verbal fights and prolonged silences ceased. The couple began to enjoy each other's company and to take pleasure in their son's activities. They noted a progressive decrease in attempts to mouth his hands and suck his index fingers. Near the end of the third month of their therapy, George (now 31 weeks of age) was making no effort to suck his fingers. The parents then elected to discontinue their individual therapy sessions.

Comment

A legion of unanswered questions is posed by this case. In the case we have described, one adverse effect of maternal deprivation was reversible with early intervention. The first key to success seems to be early recognition and the second an awareness of the magnitude and importance of parental anxieties. Finally, willingness on the part of the therapist to involve himself or herself with the parents in a therapeutic effort is a concept that requires continued emphasis and evaluation (7).

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double-spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Dopamine Hypersensitivity and Tardive Dyskinesia

SIR: Although "Postsynaptic Supersensitivity in Schizophrenia" by Ghanshyam N. Pandey, Ph.D., and associates (May 1977 issue) was an elegantly conceived and executed study, I must take issue with one of its major conclusions. The authors correctly observed that their data based on the apomorphine-induced growth hormone response did not demonstrate dopamine (DA) receptor hypersensitivity in patients with tardive dyskinesia. However, their implication that DA hypersensitivity is not involved in tardive dyskinesia is misleading. The flaw in their experimental model was the implication that DA neurons and receptors in very distinct areas of the brain are equal in their response to drugs or transmitter. They assayed DA receptor activity in the tuberoinfundibular system, but it is commonly agreed that DA-mediated motor activity arises from the nigrostriatal system. Although there is some evidence (e.g., the Pandey and associates study) that the mesolimbic and tuberoinfundibular systems tend to reflect each other's reaction to drugs, there is good evidence that these systems and the nigrostriatal one do not reflect each other. First, clinical improvement and extrapyramidal symptoms do not correlate well. Second, Smialowska (1), using histofluorescence studies in the paraventricular nucleus of the hypothalamus (PVH) and the caudate putamen (CP), found that spiroperidol, a potent butyrophenone, blocked the enhanced DA fluorescence induced by D-145 (an amantadine derivative) in the PVH but only weakly inhibited the same process in the CP. This result would imply a qualitative difference in DA receptors in the two regions.

Although the authors referred to Retrosen and associates (2) to make the point that there is no increase in DA-sensitive adenylate cyclase activity in the striatum of rats after chronic neuroleptic treatment, this finding in no way means that DA neuron hypersensitivity is not involved with tardive dyskinesia. Burt and associates (3) recently demonstrated that chronic treatment of rats with haloperidol or fluphenazine resulted in a 20–25% increase in DA receptor binding sites assayed with [^{3}H] haloperidol, a phenomenon that does not depend on specific increases in DA-sensitive adenylate cyclase.

In summary, I think that the authors' experimental design was fine for studying the behavioral implications of increased DA receptor sensitivity but was not pertinent in regard to the motor effects of DA neuron or receptor hypersensitivity as seen in tardive dyskinesia.

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R. MICHAEL ALLEN, M.D.
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Dr. Pandey and Associates Reply

SIR: We were careful to limit our conclusions to the tuberoinfundibular dopaminergic system associated with neuroendocrine response; hence, as Dr. Allen correctly states, one cannot rule out nigrostriatal dopamine supersensitivity from the data, which failed to show an increased growth hormone response following stimulation of DA receptors associated with the tuberoinfundibular system. Neither can one rule out the possibility that the DA receptors associated with the mesolimbic system, and presumably with schizophrenia-like symptoms, display different sensitivities than do the tuberoinfundibular dopamine receptors. It is clearly possible that discrete DA systems in different parts of the brain may have predetermined (by either genetics or environmental influences) differences in sensitivity to DA agonists or may be affected differently by chronic neuroleptic treatment.

Many investigators speculate that there are pharmacologically distinct types of DA receptors in brain. A recent study by Friend and associates (1) suggested that DA receptors may respond to chronic receptor blockade in a tract-specific manner. They report that rats chronically pretreated with haloperidol and subsequently withdrawn show *decreased* [^{3}H] haloperidol binding in the pituitary whereas the binding *increases* in the striatum.

Such differences do not detract from the important finding that neuroendocrine associated DA receptors do not show supersensitivity in tardive dyskinesia as also observed by Ettigi and associates (2), and that neuroendocrine-associated DA receptor activity does appear to distinguish between acute and chronic populations of schizophrenics, as noted in our article.

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GHANSHYAM N. PANDEY, PH.D.
DAVID L. GARVER, M.D.
CAROL A. TAMMINGA, M.D.
JOHN M. DAVIS, M.D.
Chicago, Ill.

Explaining Benzodiazepines: Another Approach

SIR: We found the Special Section on benzodiazepines (June 1977 issue) most informative and thought provoking. In a recent issue of *Nature* Squires and Braestrup (1) suggested an additional possible mechanism of action for the benzodiazepines. They demonstrated a high-affinity specific binding of [^{3}H] diazepam to receptors in rat brain, primarily in the frontal and occipital cortex. A total of 21 benzodiazepines were highly selective for the [^{3}H] diazepam binding sites; moreover, several postulated neurotransmitters such as GABA and glycine (or their antagonists) had no inhibitory action on [^{3}H] diazepam binding.

One must not assume immediately that this brain receptor model answers the riddle of the antianxiety effect of the benzodiazepines. Flurazepam, diazepam, and nitrazepam all demonstrated high-specificity binding, while chlordiazepoxide demonstrated approximately 1/30 of this binding potential. The antianxiety effects of both flurazepam and nitrazepam have not been demonstrated (2), so one cannot confidently attribute their receptor binding to an antianxiety effect. If a receptor is hypothesized to be specific for anxiety, then those benzodiazepines with less antianxiety effect should show significantly less binding than the more potent anxiolytics. This is analogous to Synder and associates' phenothiazine model of dopamine receptor binding, which demonstrates that the degree of blockade of the dopamine receptors is well correlated with the antipsychotic effect (3). We also noted a wide discrepancy in binding potential between chlordiazepoxide and diazepam, both well-known anxiolytics. This marked difference should not exist if these sites mediate antianxiety activity. In addition, if the receptor is truly anxiolytic, then other minor tranquilizers (meprobamate, hydroxyzine, phenobarbital) should bind with affinity comparable to the benzodiazepines. The results obtained by Squires and Braestrup tend to negate that hypothesis (1).

Further research is necessary to our understanding of these new receptor sites and their value in delineating the anxiolytic mechanism of the benzodiazepines. We agree with Squires and Braestrup that the search for an "endogenous transmitter substance acting on the benzodiazepine receptor seems worthwhile" (1).

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NEAL R. CUTLER, M.D.
DAVID SHORE, M.D.
Long Beach, Calif.

On Residency: Emotions and Spouses' Involvement

SIR: We read "The Emotionally Disturbed Psychiatric Resident" by Andrew T. Russell, M.D., and associates (January 1977 issue) and were particularly interested in the authors' allusion to emotional problems in residents' spouses. We are now collating data for a study that will report results of questionnaires sent to randomly selected directors and spouses of residents. The questionnaires attempted to ascertain spouses' feelings about the residency and to determine whether their involvement on some level would be helpful. The majority of the directors felt that there was either minimal or no involvement of spouses in their programs, but involvement as perceived by spouses themselves was much higher than would have been predicted by the directors' responses. We concluded that directors have surprisingly little knowledge of residents' spouses and their amount of involvement in the residents' training. In light of these findings, Dr. Russell and associates' data pertaining to spouses must be regarded as incomplete because they are based entirely on questionnaires completed by directors.

It was of particular interest that the majority of spouses seemed satisfied with their present level of involvement, which most often included informal discussions at home and attendance at social functions. Specifically, the responses seemed to indicate that increased structured involvement, such as a group, is not desired, even though the residency program puts stress on the marital relationship as well as on the individual resident and his or her spouse.

The conclusion stated by Dr. Russell and associates that "residency programs may need to consider involving spouses in a group experience" is not supported by our data primarily because most spouses and residents do not seem to want such a therapeutic approach to marital stresses, although some spouses were dissatisfied and felt a need for some formal opportunity to resolve their difficulties.

We do not wish to minimize Dr. Russell and associates' concern about spouses but rather to underline the need for individual assessment and constant watchfulness by the directors for signs of marital difficulties, dissatisfaction, or a desire for greater involvement expressed by individual couples. It would seem that approaching the problem on an individual rather than a group basis would be most fruitful.

JUDITH CHUCKROW GOETZL, M.A.
UGO GOETZL, M.D.
Durham, N.C.

SIR: Dr. Russell and associates presented data on emotional disturbance among psychiatric residents based on a survey of residency training directors. We feel that investigative efforts in this difficult field are needed. There has been a reluctance to investigate the prevalence and incidence of psychiatric disturbance among psychiatric residents, perhaps because of a certain understandable defensiveness on the part of psychiatrists.

The low incidence of emotional disturbance in the psychi-

atric residents studied was striking. There is a methodological problem that may account for this in part. Training directors often are not involved in direct continuing contact with residents and may minimize (or maximize) residents' pathology because of a lack of awareness or administrative pressures. Despite documentation of serious disability provided by fellow residents and clinical supervisors, we have observed in a variety of settings a reluctance on the part of administrative faculty to acknowledge manifest illness in psychiatric residents.

We wonder whether a more complete study might include the administration of the survey questionnaire to chief residents, peers, and perhaps outside observers in order to obtain a more accurate data base with regard to the prevalence and incidence of psychiatric disturbance and impaired performance among psychiatric residents. We feel that it is extremely important to have an accurate idea of the scope of the problem in order to deal with it more effectively.

DENNIS P. MILEY, M.D.
BURTON J. CONN, M.D.
San Diego, Calif.

Dr. Russell Replies

SIR: We appreciate these perceptive letters, which raise several important issues concerning our study of the emotionally disturbed psychiatric resident. Both letters point up the major methodological dilemma in research of this kind—who will be the source of information, and how reliable is that source? As we discussed in our paper, we too are concerned about just how much psychiatric training directors know about their residents and how reliable they are in reporting emotional difficulties. But who knows more? At the least, residency directors are directly involved in the selection of residents and reviews of their performance, and they also receive evaluations by supervisors, faculty, etc. As Drs. Miley and Conn point out, chief residents may be a good source of information, but we wonder if there might be similar gaps in their knowledge. Another alternative would be to ask residents to rate themselves and/or their peers. This indeed would provide interesting comparative data but would involve a much larger study with the inherent problem of self-reporting error. However, in spite of these problems we feel a questionnaire study directed to the residents themselves (with strict guarantees of anonymity) would provide very important information.

A combination approach was undertaken by Dr. and Ms. Goetzl, who queried both residency directors and the spouses of residents. We eagerly await their full report. Our data showing a higher incidence of unmarried and divorced residents as well as disturbed spouses among the "problem" residents raise important questions and concerns about marital stresses within a residency training program. Although spouses in an individual program may not desire or require a group experience, it is vital that residency directors be *aware* of the marital stresses and be prepared to offer individual or group help to those spouses who are dissatisfied and feel a need for some formal opportunity to resolve their difficulties.

These letters underline the dearth of adequate information in the important area of the emotionally disturbed psychiatric resident. Further research such as the Goetzls' project is both needed and welcomed. We hope such research will help us anticipate and deal with emotional problems among our residents before they lead to major personal, family, and career disruption.

ANDREW T. RUSSELL, M.D.
Los Angeles, Calif.

A Question of Zeal

SIR: In her letter "Committing Violent Manic Fatients" (June 1977 issue) Frida G. Surawicz, M.D., suggested that an attorney "who took an adversary approach" and succeeded in having his client released from involuntary hospitalization might have violated legal ethics and been guilty of legal malpractice. I disagree. Had the attorney done otherwise, he might have breached an ethical or legal responsibility to his client. In taking an adversary stance—if that is what his client wanted—he merely guaranteed his client's constitutional right to the effective assistance of counsel. Surely, if Dr. Surawicz were in need of legal assistance, she would want no less than the zealous advocacy required of attorneys by the Code of Professional Responsibility.

When a person is faced with involuntary civil commitment, he already has several strikes against him, which can include the prosecuting attorney, his family, his doctor, and often judges (whose deference to psychiatrists is notorious). In the face of all this opposition, it is indecent to deny him the right to a single, zealous spokesman. And when this spokesman prevails against this opposition, does that not say something quite significant about the strength of the case for commitment?

ALAN MEISEL, J.D.
Pittsburgh Pa.

Dr. Surawicz Replies

SIR: Because of space limitations, I was unable to describe the individual cases in detail; presumably, Dr. Meisel has drawn his conclusions from an incomplete data base. In the case he refers to, the patient was brought to our hospital with commitment papers and immediately demanded his release which we refused. He then hired a lawyer, who read his chart and met with us. At the commitment hearing, the judge released the patient from involuntary hospitalization in our facility but decided that he should be transferred to a private hospital for treatment, an option that the patient and his lawyer proposed at the hearing. This seemed to be an excellent solution. Something happens to the patient-doctor relationship when the doctor has to testify in court—moreover, the patient refused treatment. We subsequently learned that the patient and his lawyer did not follow through with the transfer. After a few months, the patient, divorced and broke after some grandiose financial transactions, entered into a severe suicidal depression for which he had to be readmitted to our hospital. I wanted to point out that psychiatrists (hopefully) know more about the treatment of manic-depressive disease and that there is conclusive evidence in the literature that lithium successfully aborts manic episodes and decreases the occurrence of further manic or depressive attacks.

If I suffered from a sickness that seriously affected my judgment and actions, I would prefer to have a lawyer who acknowledged prevailing medical opinion and realized that there can be disastrous consequences from an un-

treated psychiatric illness. Zealous advocacy should not lead to blind identification with a client to the exclusion of objective evidence.

Many of my colleagues feel that we cannot win. To release an untreated and unimproved patient would make us obvious candidates for a malpractice or medical negligence suit by the relatives, or even by the patient himself, for any damages resulting from his illness. The inconvenience of having to attend commitment hearings saved us at least from that fate, but I continue to feel that the moral and ethical responsibility for the patient's well-being was with his zealous lawyer.

FRIDA G. SURAWICZ, M.D.
Lexington, Ky.

Chronology of Combined Neuroleptics and Antiparkinsonian Administration

SIR: It has been reported that antiparkinsonians can decrease significantly the plasma level of neuroleptics (1–5), thus diminishing their therapeutic effects.

We have conducted an investigation to ascertain whether this interaction could be influenced by modifying the chronology of drug administration. The type and dose of neuroleptic and antiparkinsonian medication remained constant throughout the study for a given patient. The population consisted of 19 chronic schizophrenic patients (11 men and 8 women; mean age, 47.9 years; mean duration of illness, 20.2 years). The patients were receiving various neuroleptics (mean daily dose in chlorpromazine equivalents, 768.4 mg) and various antiparkinsonians (mean daily dose in benztropine equivalents, 4.4 mg). During the first 4 weeks, the patients took all drugs at the same time (7:00 a.m., 11:00 a.m., 5:00 p.m.). During the next 8 weeks, the antiparkinsonian medication was given at 7:00 a.m. and 11:00 a.m. and the neuroleptics at 5:00 p.m. and 10:00 p.m. For the last 4 weeks of the study, the medication was again administered on the schedule used during the first 4 weeks. No significant differences occurred throughout the study in plasma levels of neuroleptic (measured weekly), in the psychiatric evaluation (Brief Psychiatric Rating Scale, Clinical Global Impression, and Nurse's Observation Scale for Inpatient Evaluation), and in the intensity of the parkinsonian symptoms. An interval of only several hours between the administration of the neuroleptic and antiparkinsonian medication had no significant influence on the measures used in the study.

These results are concordant with those of another of our studies (5), undertaken simultaneously, in which it was found that after the withdrawal of the antiparkinsonian drug a statistically significant progressive increase of the neuroleptic plasma level lasted 12 weeks before reaching a plateau.

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The Life Cycle and Psychiatric Education

SIR: I read with interest the supplement to the *Journal* entitled "American Psychiatric Education: A Review" (March 1977 issue). However, I was struck by the absence of a perspective of the life cycle as a whole, particularly regarding the middle and later years. Training and programs in child psychiatry were mentioned, but adult development and its special crises were not given attention in this review.

Data from the Bureau of the Census (1) indicate that the percentage of the population who are 65 or older has increased substantially to the figure (1976) of approximately 10.39%; projections indicate that this figure will increase to approximately 12.9% by the 1990s. These figures make it abundantly clear that physicians, including psychiatrists and other health providers, must be properly trained with respect to the particular normative features and pathological conditions associated with the middle and later years. As I have indicated on previous occasions, including an overview on "Psychiatry and the Elderly" in the *Journal* (September 1975 issue), psychiatry and other mental health professions have avoided the realities of the changing age structure of our population.

I wish on this occasion again to alert the teachers, leaders, and practitioners of psychiatry to the importance of turning their attention to the perspective of the life cycle as a whole.

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ROBERT N. BUTLER, M.D.
Bethesda, Md.

Dr. Eaton Replies

SIR: Dr. Butler's comments concerning the need for psychiatry to give particular attention to the problems of the middle-aged and elderly are most appropriate.

The Psychiatry Education Branch of the National Institute of Mental Health does expect training programs to provide balanced curricula, including the perspective of the life cycle as a continuum of developmental events from birth to death. It is our hope that through increased didactic work and clinical experience in this area, both psychiatric and nonpsychiatric physicians might become better equipped to render appropriate care to people of all ages.

The NIMH Division of Special Mental Health Programs

provides support for discrete training programs in the area of aging, and it is expected that these programs will also lead to improved care for middle-aged and elderly citizens. I thank Dr. Butler for again emphasizing the need for more attention to this increasingly important group of people.

JAMES S. EATON, JR., M.D.
Rockville, Md.

Methodology in a Case Report

SIR: Gregory Asnis, M.D., raised several important questions in his report "Parkinson's Disease, Depression, and ECT: A Review and Case Study" (February 1977 issue). First of all, the case study seemingly provides evidence that ECT was the "active ingredient" in this patient, but no attention is given to the effect of hospitalization in an inpatient psychiatric facility (with its attendant milieu and other modes of treatment) or to exposure of the patients to a study protocol that involves close contact with several physicians on a regular basis. In addition, no mention is made of medications that were given to the patient during his hospitalization. It may well be that these comments do not constitute sufficient grounds to invalidate the report's general premise, but these questions could have been profitably addressed in the discussion; a lack of control over "other" variables is a well-known deficiency of the single-case-report methodology.

Second, the patient's relapse was treated in a manner somewhat inconsistent with the basic hypothesis of the report. Perhaps a course of maintenance ECT should have been used instead of or in addition to the medications that were given. As an extension of my comment above, it would seem that outpatient ECT could serve as a control of sorts for the placebo effect of inpatient treatment. In any event, the single-case-study model would then have been continued in consistent fashion.

In this time of increasing restrictions related to the use of ECT (witness the California experience), it would seem to be incumbent upon us to "tread lightly" in seemingly advocating a treatment that is at once fascinating and controversial. I believe that it would be both useful and advisable to publish articles such as Dr. Asnis' with a discussion, or comments, by the reviewers who have had a role in selecting the paper for publication.

ROBERT A. KUWIK, M.D.
Fort Stewart, Ga.

Dr. Asnis Replies

SIR: The purpose of my report was to isolate the effect of ECT on both depression and Parkinson's disease in a patient with both disorders. Previous reports (1, 2) had commented on the effect of ECT on psychotically depressed patients with Parkinson's disease, noting an alleviation of the symptoms of both illnesses. My article demonstrated that ECT had a specific antiparkinsonian effect even before its antidepressant effect.

Dr. Kuwik suggests that the results are limited because of the case study approach and may be attributable to a placebo effect. It is clear that since my study was based on a single case, the results must be replicated. However, I strongly doubt that these results were due to a placebo effect. Previous studies of treatment in Parkinson's disease have compared L-dopa to placebo in a double-blind within-patient design. Parkinsonian signs deteriorated to pretreatment baseline recordings soon after substitution of placebo and did not improve during weeks of placebo trial, indicating no noticeable placebo effect (3, 4). The extent of improvement in parkinsonism in this single case study argues against a placebo effect. On three rating scales measuring extrapyramidal symptoms, the improvement ranged from 40 to 66%. This marked improvement is superior to therapy with anticholinergics (21%) and comparable to short-term results with L-dopa (53%).

Dr. Kuwik's points regarding medication and follow-up are well-taken. The patient was taken off of all antidepressants and antiparkinsonians for 1½ months before receiving his first ECT. During the hospitalization the only medication he received was an occasional hypnotic for sleep. Shortly after discharge from the hospital (within 7 weeks) the patient's depression and parkinsonism deteriorated to pretreatment levels. Because the patient refused to be treated again with ECT, a neurologist prescribed carbidopa/L-dopa (Sinemet). It might have been informative to treat this patient with ECT to see if his depression and parkinsonism were still responsive to ECT, particularly since his extrapyramidal symptoms responded poorly to the carbidopa/L-dopa (he was placed on this drug 7 weeks post-ECT).

I am thankful to this reader for taking an active interest in my article and I hope that other work will be done to further our understanding of the association between Parkinson's disease and depression and the effects of ECT on these disorders.

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GREGORY ASNIS, M.D.
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Editor's Note. While exchanges such as those proposed in the last paragraph of Dr. Kuwik's letter might be interesting, they would involve numerous problems. The decision to designate papers as controversial might bring in the possibility of bias. Further, reviewers' comments are confidential. The space required to do what Dr. Kuwik suggests would mean that we would have to decrease the number of articles we publish. We feel that our Letters to the Editor section serves this function well. Also, we do have occasional Opinion and Comment sections for subjects that are generally agreed to be controversial.

Dispensing with "Psychosomatic"

SIR: One is always flattered by having an old book review quoted, even when it is taken out of context and cited as an example of "deplorable ignorance of the current state of the

field." Since Z.J. Lipowski, M.D., chose to do this in "Psychosomatic Medicine in the Seventies: An Overview" (March 1977 issue), I should like to comment briefly on his article.

The essential ambiguity of the term "psychosomatic" was clearly acknowledged in a comprehensive World Health Organization technical report (1), which was not included in Dr. Lipowski's long list of references. Nor did he mention the highly critical conclusions reached by so authoritative an observer as Grinker (2). Special pleading apart, it may reasonably be questioned whether general medicine, clinical epidemiology, and psychophysiology do not now cover the field adequately enough to dispense with the need for so commodious a term as "psychosomatic."

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MICHAEL SHEPHERD, D.M.
London, England

Dr. Lipowski Replies

SIR: Prof. Shepherd's only discernible comment on my paper at first glance appears to concern semantics. He contends that the term "psychosomatic" should be dispensed with. This would be a trivial issue, one to be settled by either redefining the term, as I have done, or proposing an alternative one. Prof. Shepherd does neither; instead, he hints vaguely that what the term designates does not need to be differentiated and labeled at all. This, of course, is no longer just a point of semantics but a fundamental issue regarding the validity and usefulness of the psychosomatic concepts. Prof. Shepherd refers to two publications with an implication that they support his contention. In doing so he misleads the readers.

The report of the WHO Expert Committee (his reference 1), chaired by the late Sir Aubrey Lewis, offers a thoughtful and sympathetic discussion of the psychosomatic concept. It points out that the term "psychosomatic" has been used in two senses: 1) to refer to a holistic approach to medical research and practice, and 2) to designate a group of disorders. The report was highly critical of the term "psychosomatic disorders" and stresses that it undermines the unitary approach to medicine and reaffirms the mind-body dichotomy. I fully agree with this and have repeatedly proposed that the term be abandoned. The report pointed out, however, that it is difficult to change firmly established terms and emphasized throughout the value and importance of the psychosomatic approach. It recommended that psychosomatic concepts, research, and training be propagated and extended and suggested how this should be done. Nowhere in the report was it stated that the term "psychosomatic" should be dropped or that what it stands for is invalid and useless. Just the opposite is the case.

Grinker's book was a landmark in the field when it first appeared in 1953. It provided an outspoken critique of the then prevailing trends in psychosomatic theory, especially of Alexander's specificity hypotheses, and helped pave the way for a more critical and scientific phase in the development of psychosomatics. Grinker expressed dissatisfaction with the term "psychosomatic" but neither adopted an alternative one nor claimed that the concepts this term had come to connote were invalid. Again, the contrary is true.

The WHO report urged an "analysis of areas of misinformation and confusion" regarding psychosomatics. To this end I wrote my article and quoted Prof. Shepherd's not-so-old book review as a deplorable example of misinformation. His letter effectively contradicts his complaint that I have quoted him out of context.

Z.J. LIPOWSKI, M.D.
Hanover, N.H.

More on Movies

SIR: I was glad to see the article "Images of the Mind" by Irving Schneider, M.D., in the June 1977 issue. I commend him for the excellent parallels he drew between the history of psychiatry and films. I realize the article was not meant to be totally comprehensive, but I would like to make some comments and additions since I have been using commercial films in teaching psychiatry and psychology for the past several years.

Edwin S. Porter made a film in 1905 called "The Kleptomaniac" that dealt with social injustice. The story was of two women, one poor and one rich, both caught shoplifting. The poor one was jailed but the rich one—the kleptomaniac—was freed.

Geriatric psychiatric problems have been competently delineated in three films. "Make Way for Tomorrow" (1937) was a plea for Social Security in which an elderly couple was magnificently portrayed by Victor Moore and Beulah Bondi. It was the first major film to focus on the problems of the aged. A 1966 British film, "The Whisperers," with Dame Edith Evans, depicted loneliness, paranoia, and hallucinations. "I Never Sang for My Father" (1970), in which Melvyn Douglas played an octogenarian, was so well done that it was painful to watch.

The subject of suicide was covered in "14 Hours" (1951), a true story about a man on a ledge in New York City. It depicted reactions of people on the street and also made a feeble attempt at developing some psychodynamics. Martin Gabel had a small role as the psychiatrist and Richard Basehart played the distraught young man. In actuality the man jumped, but the film had a happy Hollywood ending in which he was saved. "The Slender Thread" (1965) dealt with a suicide prevention center; Sidney Poitier kept Anne Bancroft on the phone long enough for a rescue party to arrive.

Producer Val Lewton specialized in psychological horror films, which were well scripted and directed. "The Curse of the Cat People" (1944) was a sequel to his first film, "The Cat People" (1942), which beautifully demonstrated the fantasy playmates of young children and has sometimes been retitled "Amy and Her Friends" and used for teaching purposes. His 1946 "Bedlam" depicted a 1761 London asylum and the inhumane treatment of patients.

The subject of alcoholism in films probably dates back to the first version of "Ten Nights in a Bar Room" in 1903. The great pioneer D.W. Griffith made two one-reelers in 1909: "What Drink Did" and "A Drunkard's Reformation." "The Lost Weekend" (1945) and "Days of Wine and Roses" (1962) are two fine examples of the many talking films on the subject. The drug scene has been covered in such films as "Man with a Golden Arm" (1955), "Synanon" (1965), and "Panic in Needle Park" (1971).

Two outstanding films on schizophrenia were "Life Upside Down" (1965, France) and "Diary of a Schizophrenic Girl" (1970, Italy).

Two crudely made documentaries of interest are "Warrendale" (1966) and "Titticut Follies" (1967). The former dealt with a Canadian treatment center for emotionally disturbed children that encouraged outbreaks of emotion and violence. The latter was filmed at Bridgewater Hospital for the Criminally Insane in Massachusetts and was somewhat sensational and controversial.

There have been many other interesting psychiatric films, such as "The Cobweb" (1955), which depicted administrative battles in a hospital that had a deleterious effect on patients; "Mine Own Executioner" (1948), dealing with a therapist's overinvolvement with his treatment; and "My Six Convicts" (1952), about psychotherapy in a prison setting.

Dr. Schneider has opened the door to a marvelous subject that merits more exploration.

JAMES K. LOUTZENHISER, M.D.
Kansas City, Mo.

Contractual Agreements with Adolescents?

SIR: In his article "The Ethics of Practice in Adolescent Psychiatry" (April 1977 issue) Derek Miller, M.D., said, "In adolescent psychiatry a contractual system may be antithetical to good treatment because it may imply submission." Generally, freedom to contract implies adulthood and in this respect may be excellent training for the juvenile in learning to accept responsibility for his actions. The contract, far from implying submission, implies equality; if anyone is submitting, it is the psychiatrist who agrees to be equal to his patient, neither dominating nor being dominated. This is not to say that the roles of patient and psychiatrist are interchangeable; they are merely equal in that the therapist offers psychiatric services for which the patient renders a fee.

Legal scholars have noted a movement from a status society, wherein an individual occupies a role that defines him (slave, serf, wife), to a society based on contract, wherein every individual is free to make and break promises and to have consequences flow from these decisions (1). The family is a status social unit in which the individual is a son, daughter, mother, etc. Seen in this context, adolescence is the process of trading in this status for individuality and might very well be the most appropriate time to encourage contracting, with its rights and concomitant responsibilities. Of course this entails a risk for the therapist; a person cannot fire father or mother, but he can discharge a psychiatrist whose services he no longer wants. The therapist who provides high-quality services will have every reason to welcome the opportunity to be accountable.

Another issue in this interesting article is raised by the statement, "It is unethical to betray the privacy needs of young people in treatment; it is also presumably unethical to break the law." It is not unethical to break the law; it is illegal to break the law. Civil disobedience is based on the ability to break the law, but of course the consequences for such a breach may involve a fine or jail sentence.

The California case *In re Lifschutz* (2) involved a psychiatrist who was ethical and spent a brief period in prison because he refused to obey a court order to answer questions and produce records pertaining to a former patient. Dr. Lifschutz maintained in part that the order infringed his right to privacy. The Supreme Court of California disagreed and denied the writ of habeas corpus, stating that not all psychotherapeutic communications are absolutely privileged. Dr. Lifschutz broke the law and paid the price, but he raised the credibility of psychiatrists by his moral and ethical stance.

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SUSANNAH SMITH, J.D.
Chicago, Ill.

Dr. Miller Replies

SIR: The benefits of placing the relationship between a minor patient and a psychiatrist on a contractual basis are claimed to include encouraging a sense of adult responsibility and equating the status of the patient and the psychiatrist. Unfortunately, treating disturbed adolescents as if they were capable of being adults does not make this so. Apart from being a dubious psychological approach, the recommendation flouts established legal doctrine. Even nondisturbed minors are generally considered legally incapable of entering into binding contractual relationships (1).

In some states infant incapacity has been removed by statutes, but generally contractual disability is lifted only for "the performance of a medical or surgical procedure, and only for married minors, pregnant minors, or minors of 18 years of age or older" (2). It is difficult to imagine how an adult concept of responsibility for one's actions can be encouraged by having a minor enter into a relationship that he/she can void at any time, even if this approach made psychological sense.

If legal doctrine assumed that a contract for psychiatric service could be considered binding—a concept that would foreclose the minor's option of disaffirmance—a situation would be created that is antithetical to good treatment. It would require the minor to submit on the basis of having come to an agreement that he or she cannot possibly fully understand because of his or her whole psychological makeup.

Psychologically disturbed adolescents have little sense of time, tending to be prisoners of the present, and often gain pathological instinctual gratification from antisocial behavior. This would indicate a significant degree of mental incapacity, and another branch of legal theory refuses to recognize the validity of a contract entered into by individuals so incapacitated.

It would be unethical for a practitioner to set up a contractual relationship without informing a minor of the right to void the contract at any time. However, such information would surely be seen by the minor as an invitation to void the contract. Thus, contractual relationships for minors seem to be both psychologically unreasonable and legally improbable. States that have statutes lifting infant disability for obligations incurred for hospital, medical, and surgical treatment apply only to married minors.

Ethics are rules of conduct recognized in respect to a particular group culture; in this sense it is unethical for psychiatrists to break the law. If ethics are also a set of principles or values relating to the rightness or wrongness of certain actions, the goodness and badness of the motives and ends of

such actions, then it may indeed be necessary for psychiatrists to flout a legal convention.

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DEREK MILLER, M.D.
Chicago, Ill.

Consultation: What Settings?

SIR: While Dr. Z.J. Lipowski's expertise in consultation-liaison psychiatry is formidable, his attack on community mental health consultation in "Psychiatric Consultation: Concepts and Controversies" (May 1977 issue) is more polemic than reason. He agrees that psychiatric consultants are needed "in all health care settings," but his definition of a health care setting is restricted to medical agencies and hospitals. Whether we like it or not, a great number of mentally ill individuals are receiving care in a variety of nonmedical settings ranging from self-help groups such as Alcoholics Anonymous, to casework agencies such as Family Service, to alternate service organizations such as the Renewal House in Los Angeles. I fail to see why psychiatrists ought not to offer case or program consultation to these agencies. In addition, school teachers and clergymen are in an excellent position to detect mental illness. I do not believe we should respond only to the cases of mental illness detected by other physicians.

I agree with Dr. Lipowski's contention that consultants should work in areas in which they have been trained. At the University of Missouri, I train psychiatric residents to perform community mental health consultation. At the end of the first year of training, all residents participate in an interdisciplinary 32-hour seminar, which covers such topics as epidemiology, principles of community mental health consultation, community needs assessment and evaluation, racism, rehabilitation, value dilemmas in community mental health, and the importance of social class and cultural variables on mental health. The interdisciplinary makeup of the class is helpful in addressing the problem of role diffusion in community work. Each resident is then assigned to consult with one agency one-half day a week for an entire year. Among the agencies with which residents have consulted are Family Service, the state Mental Health Association, college campus counseling programs, an alcoholism counseling agency, rural outreach offices, and a pastoral counseling training program. In addition to individual supervision, the residents meet as a group for peer supervision. The residents have enjoyed and profited from their consultation work, and the consultees have been satisfied with their services.

Our "unique biological training" does not mean that we should consult only with medical agencies; rather, it provides us with significant knowledge and experience which, in combination with our psychological and sociocultural training, allows us to formulate a more holistic understanding of man and his illnesses and of the many variables that influence the incidence and prevalence of mental illness. The psychiatrist who thinks that his consultation will prevent a war is foolish, but the knowledgeable psychiatrist who consults with school teachers to assist them in dealing with mentally disturbed students and in creating a healthier classroom environment is performing a worthwhile and needed task. Rather than issuing an ex cathedra denunciation of community mental health consultation, Dr. Lipowski's talents might be better directed toward research in evaluating the effectiveness of such consultation.

ARMANDO R. FAVAZZA, M.D., M.P.H.
Columbia, Mo.

Dr. Lipowski Replies

SIR: Dr. Favazza rightly observes that my article is polemical: it expresses controversial views on a contentious subject. Being polemical, however, is neither antithetical to reason nor synonymous with attack or denunciation. I would submit that my paper is none of these things. It contains a reasoned if sharp critique of community mental health consultation, arguing that its scope needs to be restricted while its effectiveness remains to be demonstrated. Others have made similar statements. Garber (1) quoted Cumming as saying, "The literature on (indirect or agency) consultation makes depressing reading for anyone interested in evidence." A review of the most recent literature on the subject concludes that research on the effectiveness of community mental health consultation is conspicuous by its absence (2). Some writers speak of a "crisis in consultation," lament that consultees are indifferent to threats that indirect consultation may be discontinued, and call for accountability in this area (3).

Dr. Favazza does not respond to my specific arguments and proposals but confines himself to challenging his own dramatized and thus distorted version of my article. I have not stated that we should consult *only* with medical facilities but argued that there was documented need for psychiatric consultants in the medical context and that this should influence our priorities. Clearly, some psychiatrists will need to continue providing consultation to nonmedical agencies that help to detect, refer, and rehabilitate mentally ill persons. Surely, psychiatric residents need training in community psychiatry. Their enjoyment of consultation work, however, does not necessarily support its value to the consultees, nor is the satisfaction of the latter evidence of impact on the mental health of the population.

I wrote my article as a contribution to the continuing debate about the proper role and functions of psychiatrists. I have tried to show that one's approach to consultation reflects one's stand on more basic issues regarding the definition of our profession. The outcome of this discussion cannot fail to influence our practice and training, our relations with other health professions, our public posture, and our future as a profession. I hope to have set forth my position unequivocally and supported it with some cogent arguments, facts, and figures. I am persuaded that the role of the psychiatrist needs to be more clearly defined and its limits and limitations made far more explicit. Along with many other colleagues, I believe that psychiatrists will "find it increasingly important to reestablish their medical role" (4). I foresee that in the coming years psychiatry will become more medical and medicine more psychiatric or psychosocial. There are obvious and powerful social, political, and economic forces pushing in this direction (5). This is not to say that we are on the threshold of a psychosomatic millenium! It is important that as we become more medically oriented we do not abandon what is worthwhile in community psychiatry and do not replace the hue and cry of the social reformers with a chorus of opportunistic grasshoppers jumping on the

bandwagon of liaison psychiatry. I suspect that once the heat of the polemic has subsided, Dr. Favazza and I could find more areas of agreement and common purpose than dissent. Evaluation of the goals and effectiveness of our respective work is one such purpose.

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Z.J. LIPOWSKI, M.D.
Hanover, N.H.

Criminal Law: Implications for Psychiatry?

SIR: The criminalization of civil commitment procedures over the last decade, recapitulated in a recent article by Stone (1), gives rise to feelings of frustration and impotence in many psychiatrists who feel the emphasis on the patient's civil rights robs him or her of a fundamental right—the right to be restored quickly to a functioning state. It is ironic that during this same period, decisions of the Supreme Court have been nibbling away at many of the due process rights granted by the Warren Court.

Since 1974, the Burger Court has authorized warrantless search of the person of an individual apprehended in the commission of a misdemeanor, sanctioned entrapment by the police as long as "the defendant was predisposed to commit a crime," ruled that police did not violate an individual's privacy or deprive him of his due process rights by circulating his name and picture in a bulletin entitled "Active Shoplifters," even though he had never been convicted of shoplifting, and held that an individual does not have to be advised of his rights according to the *Miranda* decision while being questioned in the police station if he is not technically under arrest. The contradiction between the last mentioned decision and *Lessard v. Schmidt* (2) seems obvious. The former decision says that Fifth Amendment protection does not apply until a formal arrest has taken place; *Lessard v. Schmidt* stated that such protection applies in a medical examination to determine whether commitment is applicable.

Are there implications for psychiatry in these decisions in criminal law? I think there are. Arons and Katsh (3) have suggested that these recent decisions of the Supreme Court are in response to the feelings of an alarmed public that the scales have been tipped too far in favor of the criminal. A similar reaction in regard to the commitment of the mentally ill may be anticipated. Traditionally the community's fear of the "insane" has been greater than its fear of the criminal because of the presumed dangerousness of the former. Ironically, in the present climate, dangerousness is the main justification for involuntary commitment. This fear has been expressed in the reaction of Long Beach, N.Y., to the emptying of the state's mental hospitals. Here in Ohio there is a move afoot to amend the commitment statutes of August 1976 in the direction of modifying some of their more onerous restrictions. Equally significant, families, neighbors, and police are besieging psychiatrists for help in coping with those denied admission under the new laws. Under the pressure of public opinion, a position may eventually emerge that will guarantee adequate protection of patients' rights, balancing that protection against the needs of the community.

During this difficult transitional period, we must not defend nonexistent treatment in some state hospitals or long-term, nonjudicial commitment. We must make our voices heard as clearly as possible, not only in courts and legislatures but in the vital arena of public opinion. We must learn from the abolitionists to speak directly to the public as well as to each other. This period must be used for education about recent advances in treatment and the cost of implementation of the treatment provisions of the new legislation. We may in this way lose some of the battles but win the war.

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HENRY L. HARTMAN, M.D.
Toledo, Ohio

Barbiturates and Tardive Dyskinesia

SIR: There has been increasing interest in tardive dyskinesia recently. I have noticed discussions of the use of deanol, and several neurologists with whom I have spoken are beginning to use clonazepam, a benzodiazepine used in the treatment of minor motor and petit mal seizures. I would like to report my experience with a patient whose symptoms of tardive dyskinesia subsided with barbiturate administration.

I have been following the patient, a 55-year-old woman with chronic schizophrenia and extensive state hospital admissions, for about 2 years. During the time I have known her, she has exhibited bland affect, social withdrawal, and at times has frowned constantly. She spoke abruptly, with a poverty of thought, and volunteered little information.

At one point she had been taking thioridazene, 100 mg q.i.d., and after 2–3 months developed slow, continuous sucking and chewing mouth movements. She had no complaints of pain or dental problems. She did complain of fear and uneasiness, and as she spoke of her fears the mouth movements became more prominent.

The thioridazine was discontinued. It was difficult to decide whether her uneasiness and frowning resulted from her basic illness, situational factors, or an extrapyramidal reaction.

Since she had been on numerous antipsychotic medications with no improvement, I placed her on phenobarbital. Before she started taking the phenobarbital, she still had intense abnormal mouth movements 1 week after discontinuation of thioridazene. At this point she was alert, oriented to time, place, and person, and spoke to me in a coherent fashion. She was asked to see a neurologist at one point but failed to keep the appointment.

After 1 week on phenobarbital, 30 mg q.i.d., she was dramatically calmer and was sleeping well. Further, she no longer had abnormal mouth and tongue movements. I have continued prescribing phenobarbital for about 4 months

without phenothiazines and she has remained calm and is still sleeping well. She does not have the abnormal mouth movements unless she fails to take her medication for several days at a time.

I should like to inquire whether other psychiatrists have had positive results with barbiturates and tardive dyskinesia.

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Corrections

The table of contents of the July 1977 issue lists the author of "Fluphenazine Decanoate During Pregnancy" as "M.F. Cleary et al." Dr. Cleary is the sole author.

In the August 1977 issue of the *Journal* (letters to the editor, "Confidentiality: A Solution to the Problem?"), there was an error in the spelling of Dr. Francis J. Durgin's name.

The staff regrets these errors.

BOOK REVIEWS

Style, Character and Language, *by Victor H. Rosen, M.D.; edited by Samuel Atkin, M.D., and Milton E. Jucovy, M.D. New York, N.Y., Jason Aronson, 1977, 388 pp., $15.00.*

This volume contains 14 previously published essays on a wide variety of subjects written by the talented author Victor H. Rosen before his tragic death in 1973. They have been carefully and skillfully edited for posthumous publication. We who value studies in language as one key avenue to understanding the behavior patterns of our patients and their families and our interaction with them in therapeutic endeavors are greatly indebted to the editors. Dr. Rosen was a brave explorer whose writings deserve close study. He opened up many avenues in conventional psychoanalytic thinking to studies by professionals in other disciplines, particularly linguistics, semiotics, linguistic philosophy, cybernetics, thinking, imagination, creativity, and the adaptive functions of language.

The 14 essays are divided into two sections. The first, Language and Psychoanalytic Technique, has 10 chapters with clinical examples of the mutual illumination of basic psychoanalytic assumptions and psycholinguistic theories on such subjects as "Mathematical Thought Process and Abstract Thinking," "Strephosymbolia," "Abstract Thinking and Object Relations," "Schizophrenic Language Disturbance," "Sign Phenomena and Unconscious Meaning," and "Language and Psychoanalysis." It is noteworthy that in the chapter on schizophrenic thinking Dr. Rosen revises considerably Freud's formulation that patients with this psychosis use words as though they were the equivalent of things. Dr. Rosen states,

> According to this revision the psychotic individual does not suffer a "decathexis of things." It is suggested that as a result of regression and withdrawal from social relationships, which both necessitate and facilitate verbal communication, language code connections are disrupted. (p. 169)

The introductory summary by Dr. Atkin, a senior training and supervising psychoanalyst, is a valuable personal critique that helps the reader of Rosen's essays understand the background for them in Rosen's Language Study Group, founded in 1963 at the New York Psychoanalytic Institute, and the group's arduous search for interdisciplinary interaction and enlightenment. This summary is strongly recommended as required reading for all psychothotherapists who believe that language functions are crucial to therapeutic transactions.

The second section of the book has four essays on creativity, talent, the comic, and character style. The introduction to the section by Dr. Jucovy is a splendid biographical review of Rosen's career and his interest in art. The epilogue (pp. 353–360) tells us about the genesis and activities of the Language Study Group and the usefulness of ideas from Charles Morris and Noam Chomsky and their transformations in the group. There is also a chronological list of Rosen's writings.

This collection, although not a textbook, can be highly recommended as a supplement and reference to all students of psychotherapeutic transactions.

HENRY W. BROSIN, M.D.
Tucson, Ariz.

Traité des Hallucinations, Vol. 1 and 2, *by Henri Ey. Paris, France, Masson & Cie, 1973, 1,502 pp., 440 French francs.*

In 1932 Dr. Ey's first paper on the subject of hallucinations appeared in the French periodical *L'Encephale*. Over the following 40 years he persistently pursued the subject, producing some 21 papers and monographs on this topic. Hallucinations and the idea of consciousness have been his two main clinical and research interests. *Traité des Hallucinations* represents a lifetime of effort. It is a more complete and thorough study of hallucinations and delusions than we have in any Western language. The bibliographical wealth alone is enormous; Ey seems to have covered every significant paper or book related to the subject, carefully evaluating this literature from 1950 to 1971.

Traité des Hallucinations is a truly monumental work. Ey's exceptionally felicitous style makes this book a real delight to read. Although not always easily or quickly read, his exposition is a model of the clarity for which the French have so often been congratulated.

On the basis of the fourfold foundation of Janet, Freud, Hughlings Jackson, and Husserl, Ey sets himself the huge task of analyzing what we know about hallucinations and delusions. He also tries to evaluate critically and interpret honestly and unpolemically just what has been learned since 1950. He also goes back to the great classical works of the nineteenth century. It is clear that for many years Ey has been dissatisfied with what he calls the "mechanistic-linear cerebral mythology" and the "myths of the unconscious primary processes." He has applied the insights of Jackson and Sherrington to the architectonics of the CNS and added the rich lore and analytic possibilities inherent in the newer phenomenological philosophy.

Ey freely acknowledges and pays homage to the insights of Freud; Freudian dynamics are integrally incorporated in his thinking and formulations. The result is what Ey chooses to call an organic-dynamic theory of not only hallucinations and delusions but of all psychiatry. The thesis has been worked out, not in the quiet of his study but on the basis of the clinical phenomena nature offers. Forty years of hard clinical experience is at the core of Ey's work.

Dr. Ey gives us a very thorough and critical analysis of the many theories advanced to elucidate the fundamental problem of hallucinations and delusions. Although we have usually considered these to be separate phenomena, Ey views them as closely and inevitably linked: "Hallucinations are

embedded in delusions . . . hallucinations are basically a form of delusion" (p. 829). He demonstrates their essential relatedness.

The author constantly uses four basic ideas in developing his theory and its clinical relevance. The first basic idea is that a hallucination is a pathological phenomenon: "It is a heterogeneous structure, anomic in regard to the infinity of illusions which appear in the normal exercise of our imagination." The second basic thesis consists of considering hallucinatory phenomena, at every one of their levels, irreducible to any elementary and mechanistic theory that would regard the hallucination as a simple result of neurosensory stimulation. The third basic tenet is formulated as an antipsychotogenic thesis of the apparition of hallucinations, which cannot be simply the projection of an affect, even if the affect be unconscious. The hallucination is fundamentally a "negative structure incompatible with the mere force of the wish, or wish fulfillment." The appearance of hallucination requires still another dimension, that of a deficit or breach in the system of reality testing. This antipsychogenic thesis affirms that consciousness is man's defense against the development of hallucinations and delusions. A break in this defense will inevitably result in such phenomena as delusions and hallucinations.

The fourth idea requires for the explanation of the appearance of hallucinations recourse to a hierarchical plan of organization by the psyche. Dr. Ey is very emphatic in his insistence on the fact that a hallucination does not make its appearance on the basis of disorganization of the psychosensorial systems involved. The pathogenesis of hallucinatory phenomena always reveals and betrays the existence of a disorganization of the psychic organism. (It must be noted here that there is no room anywhere in his thinking for body-mind dualism or parallelism.)

Ey rejects the traditional division of hallucinations into the two groups of psychosensorial and psychotic. Rather, he divides them into two large categories: *eidolies hallucinosiques* (which we commonly term "hallucinosis") and delusional hallucinations. These categories inevitably do much to clarify the confusion that usually accompanies discussion about hallucinations.

The chapters on hallucination and cerebral pathology as well as the chapter on hallucinogens are models of clinical description. There is also some very fascinating material on the phenomenology of hallucinations. In the chapter on "Hallucinations in Neurotics" Ey is bitterly critical of the simple, mechanistic-linear theories of the pathogenesis of hallucinations; for the most part his strictures are more than well taken. He carefully dissects the inadequacy of psychodynamic models or theories, not because they lack merit or richness but primarily because, although necessary, they are not sufficient explanations.

It is clear that the author's organic-dynamic model leans heavily on the Jacksonian principle of the dissolution of the superior levels and its inevitable release phenomena, or negative phenomena. Ey refers to hallucinations as "negative structures."

It is difficult in a relatively short review to convey the richness of this massive work and the enormous body of material that the author organizes and presents to the reader. It would indeed be a great pity if this book remained untranslated into English; it is as seminal a work as is Bleuler's monograph on dementia praecox.

JOSEPH ZINKIN, M.D.
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Behavioral Intervention: Contemporary Strategies, *by W. Robert Nay. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1976, 370 pp., $19.95.*

This textbook on behavior modification theories and techniques is designed for what the author calls BCA (behavioral change agent) trainees, such as teachers, counselors, and social workers who work with institutional populations. He disparages the disease concept of behavior disorders and prefers to treat them as problems in living that are subject to change by overt conditioning and covert modeling and training.

Nay has enlarged on the earlier, more conventional concept of mechanistic behavior patterns and their modification by stressing the potential for self-direction and demonstrates methods for developing self-control. He calls his system of therapy "phenomenal-behavioral," meaning the use of thoughts, feelings, and sensations (phenomena) as well as extrinsic controls to influence behavior changes.

Although this book is not psychiatrically oriented, it seems to offer some useful suggestions for behavioral management, especially in institutional settings, where there is so often a shortage of personnel and a need for clear-cut and achievable therapeutic goals. There are extensive references and a final chapter on the ethical and legal ramifications of certain types of behavior intervention strategies.

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Deinstitutionalization and Institutional Reform, *by R.C. Scheerenberger, Ph.D. Springfield, Ill., Charles C Thomas, 1976, 250 pp., $17.50.*

This book distinguishes itself in one important aspect from many other contributions to the literature on this presently fashionable topic: it is more objective and detached and less emotional. It is not so filled with the slogans and admonishing phrases "to do the right thing" that one unfortunately has to tolerate in the literature on the changes in approach to the problems of the mentally retarded. One may disagree with one or another statement, for example as to the value of litigation versus the damage it does, but at least the book has opinions that can be challenged rather than meaningless phraseology. Fortunately, Scheerenberger keeps his book fairly free from statements of his case in empty phrases when summarizing present trends.

The title may be misleading in one aspect: the material covering deinstitutionalization and the material covering institutional reform are rather unevenly distributed. Presumably following the present swinging of the pendulum away from institutionalization, Scheerenberger devotes the first 186 pages of this 250-page volume to deinstitutionalization and only one 30-page chapter to institutional (residential) reform. I find this regrettable because Scheerenberger is a superintendent of a state institution and could have contributed a great deal more to this subject. When the pendulum swings back to a reasonable use of residential settings when needed, as it already has in some enlightened areas of the country, we shall need all of the constructive thought available to us to improve the institutions.

We have observed the pendulum swing from "everybody should be in a residential setting" to "everybody should be in the community." Gradually, the reasonable, balanced,

and logical approach will prevail; specific indications of individual problems of human beings will be considered. Books such as Scheerenberger's are an asset to this process. His book is recommended to everybody who is interested in the problem of how to find proper living arrangements for mentally retarded people, including the educational, medical, habitational, and other services they need. The reader may not always agree with the specific opinions expressed, but he or she will learn and will be challenged.

BENEDICT NAGLER, M.D.
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The Psychoanalytic Study of Society 7, *edited by Werner Muensterberger with Aaron H. Esman and L. Bryce Boyer. New Haven, Conn., Yale University Press, 1976, 399 pp., $19.50.*

Although the number 7 on the cover of this book suggests that this is the seventh volume of *The Psychoanalytic Study of Society*, nowhere else is there any information about the preceding volumes and their contents. It is therefore not possible to learn whether there is some kind of overall plan or continuity in the series. Presumably, each volume must stand on its own.

Each of the 12 chapters deals with a different subject. With the exception of the first chapter, "Janusian Thinking and Creativity," which relates exclusively to an individual process, the chapters range over a seemingly diverse field. For example, there is a study of *Peter and the Wolf* as a means of understanding, through music, something of the imagery of the contemporary Soviet man. Other chapters deal with a cross-cultural study of prolonged adolescence and early identification, ghost sickness and superego development in the Kiowa Apache man, the life history and creative psychopathology of a shaman, magic and healing, and the Mackenzie Delta Eskimos. The concluding chapters focus on the origins of Judaism and the Passover Seder.

Although the 11 chapters cover diverse areas, there is a remarkable continuity that is cross-cultural and inclusive of both time and place. The continuity is made possible by the use of a psychoanalytic framework for understanding the phenomena under study. Data are extracted from interviews, observations, and recorded documents and subjected to psychoanalytic scrutiny. What comes through in all of the papers is the remarkable consistency with which people from diverse cultures struggle to control similar primary process functions and impulses and the manner in which diverse cultures exercise social influences in the struggle for control.

The innate destructive impulses that guarantee the dissolution of any society come under the influence of shared unconscious symbolism and consciously shared societal beliefs. It thus becomes possible for a group to live together in some degree of harmony. The powerful ties to the universe in general and to the mother and mother earth in particular give rise to culturally shared superstitions as a means of explaining things. At the same time each individual struggles with questions of sexual identity and the wish to return to the safety of the mother.

These papers make a strong case for a collective unconscious. One reads that oedipal conflicts live out their active existence in the different cultural groups with both regressions to and fixation in pregenital fantasies. Over all hovers the rather desperate yet self-preservative efforts of each culture to bring order and some modicum of harmony through its rituals and institutions. The family stands as the intermediary between the individual and the group in this struggle.

What may seem to the reader to be rather exaggerated interpretations of symbolic thought, feeling, and behavior are nevertheless heard regularly on the couch in the analysis of patients today. Perhaps a brief can be made for a greater use of rituals and ritualized institutions in our society as our mounting scientific understanding of the world in which we live seems to dissolve rather than reinforce controls. Man appears to be incapable of living in groups harmoniously by reason alone.

Having remarked on the sameness of interpretations in the analysis of patients today and the analysis of primitive cultures, I must nevertheless note a degree of unevenness in these papers. The unevenness lies in the fact that in some papers the data are insufficient to warrant some of the extended psychoanalytic formulations. In other papers this documentation is impressive and even sheds light on those in which data are lacking.

The Psychoanalytic Study of Society is not easy reading, but it is illuminating. It gives further credence to my long-held belief that a good psychiatrist can treat any patient, regardless of where the patient is coming from, as long as he or she is aware that all human beings suffer the very same kind of pain but, because of different family and cultural differences, the pain tends to be expressed in different ways.

JAMES MANN, M.D.
Boston, Mass.

Criminality and Psychiatric Disorders, *by Samuel B. Guze, M.D. New York, N.Y., Oxford University Press, 1976, 177 pp., $9.50.*

It is now almost 150 years since Pritchard's formulation of the concept of moral insanity extended the spectrum of mental disorder to include cases variously described as sociopathy, psychopathy, or personality disorder. This opened the doors of the prisons to psychiatry, but it also led to a blurring of the boundaries between psychiatry and criminology, an effect that has been intensified by the further inclusion of alcoholism and drug dependence among the mental conditions. The result has been that although the general public is now willing to see many criminals as mentally disturbed, it has at the same time come to identify all other forms of mental disorder with criminal behavior. This is no more logical than seeing all humans as criminals because all criminals are human. Logic aside, this syllogism has become a reality problem.

A corrective to this trend is badly needed, and *Criminality and Psychiatric Disorders* is a significant contribution in that direction. It reports new scientific data showing that the mental disorders found in prisons are of a different class than those seen in mental hospitals and clinics and that there is little overlap between them. About 85% of the cases of mental disorder in prisons are diagnosed as sociopathy, alcoholism, and/or drug dependence, but the major psychoses and neuroses that make up the bulk of cases in mental hospitals and clinics are seen no more often in prisons than in the general population. A second line of evidence for this point comes from a comparison of behavior after release from a prison or a hospital. Drawing on his own studies, Guze shows that recidivism was involved in a majority of cases

released from prison. The literature, on the other hand, shows that criminal behavior is a relatively rare complication of mental disorders other than sociopathy, alcoholism, and drug dependence.

This book is based primarily on some 15 years of study of almost 300 felons from Missouri prisons. They were interviewed before their discharge and followed up by means of examinations over a period ranging from 3 to 9 years. The interviews were structured, conducted by psychiatrists, and supported by similar interviews with more than 350 first-degree relatives as well as examination of all available records. When direct contact could not be made, information was taken from secondary sources.

The book opens with a broad discussion that reflects Guze's long-standing interest in psychiatric classification and his recognition of its limitations as well as the limitations of statistics on crime. The author provides a definition of terms, a description of procedures, and a general description of the case material; the interview forms are contained in an appendix. The study itself examined the interrelations among such factors as recidivism, mental diagnosis, life history, and various social and demographic characteristics.

Many of the findings tend chiefly to confirm and support existing professional opinion; e.g., the overall high arrest rate after prison release and the correlation between criminal recidivism and youth, previous recidivism, alcohol and drug abuse, sociopathy, and demographic measures of social stress. Some of the findings are new and subject to further confirmation; e.g., the strong evidence that hysteria in women often appears to be the equivalent of sociopathy in men. This formulation is supported by the clinical histories of Guze's index cases and by the incidence of the two conditions among male and female relatives of the felons.

The diagnosis of sociopathy seems to be a stable one, distinct from other forms of psychiatric disorder, rather than a transitional or atypical form of other conditions. Guze briefly mentions a possible organic type, but he does not mention the rather extensive experience with encephalitis lethargica, which in its time showed how damage to the CNS could produce the most lurid cases of sociopathic behavior.

As one becomes interested in a book with extensive presentations of primary data there is always a temptation to pick up the statistical analysis where the author leaves off. Almost always this is a futile undertaking because of errors on the part of the reader and because he or she does not have all the facts (no matter how detailed the presentation the author cannot publish everything). However, the temptation grows stronger the more carefully one reads. For example, it is tantalizing to read that 66 female felons accounted for 15 homicides among their index crimes while 223 male felons accounted for only 11 homicides and 4 attempted homicides. Another puzzle is that only 5% of the men were diagnosed as drug dependent before their index conviction; in the subsequent 9 years of follow-up this figure did not rise spectacularly, even though drug addiction was widely held to be a major cause of crime by that time. Even the figure of 26% of the women seems unimpressive in this context. Another interesting fact is that 15% of the women believed that they had talked to or received messages from God.

Questions of a different type are raised by the finding that among 144 children of women felons, 13 had died by the age of 3 and 6 more were stillborn. Only 9% of the women had been born out of wedlock, and 21% of their pregnancies were not legitimate. As one reads these figures and the data on suicide attempts, mental hospitalization, deaths (violent and otherwise), serious accidents, illnesses, and educational levels, one cannot help wonder what the corresponding figures for the noncorrectional population might be. For the most part this remains a topic for future research.

At the close of the book the author makes a bold suggestion that will undoubtedly raise some eyebrows. He observes that available treatments for sociopathy, alcoholism, and drug dependence are not dependable in most cases (and I think he refers primarily to persons seen in the correctional setting). Further, he notes that conviction for recidivism falls from 60% for ex-prisoners under the age of 40 to 6% for parolees over the age of 40. He concludes that "imprisonment until middle age, at least for recidivist criminals, should result in a major reduction in recidivism after discharge from prison." A discussion of this proposal should be especially interesting and timely in view of the recent moves in the opposite direction, i.e., to declare the prison system bankrupt because of its failures and to deinstitutionalize its population.

The timeliness of this book is further heightened by the recent collapse of the addiction theory of crime in the streets. This explanation of the cause of crime seemed to satisfy the public demand for some sort of causality, but that phase of conventional wisdom has now passed—even though addiction still remains with us. Another explanation must now be found. One can only hope that it will not be another simplistic pancausality like social stress. Guze points out that many other factors must be considered and evaluated and that more research is the only rational approach. Work like that reported in this volume is a good beginning, but all of the studies now in progress seem minuscule in comparison with the magnitude of the problem.

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Delinquency and Psychopathology, *by Dorothy Otnow Lewis, M.D., and David A. Balla, Ph.D., with the assistance of Shelley S. Shanok, M.P.H. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1976, 199 pp., $14.75.*

In his brief foreword to this slim volume, Dr. Dennis P. Cantwell says, "This is an important book. It records with precision and clarity a detailed psychiatric study of a group of children referred for evaluation from a juvenile court." In reviewing this book, I can only echo Dr. Cantwell's statement and evaluation. The book is indeed important. In this era of questioning whether the psychiatrist has a role in the juvenile court, a monograph such as this provides a valuable contribution. It details with careful, clear, and logical thinking the important contributions a clinician can make to the understanding and treatment of delinquent children. Most importantly, the authors challenge the pseudoscientific diagnosis of sociopathy.

On the basis of meticulous and careful observation of interviews with delinquent children and their families, the authors identify a group of valid psychiatric diagnoses that truly describe the psychopathology of some delinquent children and their families. Of special importance is their chapter on "Psychomotor Epileptic Symptomatology." After noting a high prevalence of psychomotor epileptic symptoms in a portion of a delinquent population referred for psychiatric evaluation, the authors suggest that these symptoms are probably more common among delinquent children than has previously been recognized and that they should be part of the differential diagnosis of all court-referred children. Obvi-

ously, they should also be a part of the differential diagnosis of all children. If one really believes that a critical mandate of the juvenile court is to treat and rehabilitate children, the treatment implications of this diagnosis are inescapable.

Another clinical observation of the authors is the high prevalence of psychotic symptoms in children and parents referred for evaluation to the court. The treatment implications inherent in these observations may seem obvious to psychiatrists but must be impressed and reimpressed on our legal colleagues who call into question the value and necessity of a psychiatric evaluation of adolescents, asserting that a psychiatric evaluation adds the label of "sick" to the "bad" child.

The authors caution those who are likely to use their findings in the treatment of delinquent children and those who are involved in policy decisions regarding the handling of delinquent children to adhere to the medical admonition, "Above all, do no harm." The authors have followed their own caution and admonish the readers to see this book in its proper perspective, that is, as a beginning study of the relationships between delinquency and psychopathology. If *Delinquency and Psychopathology* serves as a stimulus to future studies, the authors' purposes will indeed be achieved.

ELISSA P. BENEDEK, M.D.
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Malnutrition and Intellectual Development, *edited by John D. Lloyd-Still. Littleton, Mass., Publishing Sciences Group (CHC Corp.), 1976, 186 pp., $12.00.*

The prospect of 100 million malnourished children growing up to become brain-damaged adults, who in turn will parent another deprived, mentally subnormal generation is not altogether science fiction. This significant and well-edited book places current knowledge about the long-range effects of malnutrition on mental development in perspective and shows that nutritional problems require more than nutritional answers. Malnutrition is an important problem not only in underdeveloped countries but also in the United States, where 5% of hospital admissions of children under the age of 2 result from the failure-to-thrive syndrome.

There is no question that severe malnutrition during early life is associated with cerebral atrophy and mental subnormality. Whether the cerebral atrophy is causally related to protein-calorie deficiency, to vascular thrombosis because of dehydration, or to subdural hematomas because of physical abuse is less clear. The accumulated evidence suggests that the longer and more severe the malnutrition during the first 2 years of life, the more serious the effects on intellectual development.

Of special interest is the finding of class differences in malnourished children: language and adaptive behavior were more affected in deprived populations and fine motor function was more impaired in middle-class populations. This class difference highlights the importance of socioeconomic and sociocultural factors in lower-class malnutrition and the likelihood that diets deficient in protein and calories have the greatest impact on the functioning of the cerebellum. All of these effects tend to diminish with increasing age, suggesting that recovery is related to a combination of adequate nutritional replacement and emotional and psychological support. Therefore, efforts to alleviate malnutrition in deprived areas of the world require not only nutritional replacement but emphasis on improvement in the emotional and social well-being of families.

The vulnerability of the CNS during early childhood is highlighted; the critical period for brain development is from early fetal life to the fourth year of postnatal life. Although most neuronal cells are formed in utero, glial cells form primarily during the first and myelin accumulates largely during the second year of postnatal life. Although the prenatal nutrition of the mother remains an important health care need, the point is made that unless nutritional adequacy is maintained for the infant and young child, physical and mental development will be impaired. One fortuitous study based on the Dutch famine of the winter of 1944–1945 suggested that prenatal malnutrition in itself, although it stunted the brain growth of the fetus, was not associated with demonstrable diminution of mental competence during adult life.

The book presents a helpful four-level multisystem approach to nutritional problems. First of all is the capacity of the world to provide food. The current shift from a global food surplus to scarcity is a harsh reality that must be faced. The World Health Organization estimates that 50% of the world's children are undernourished. At the second level is the degree to which food is available to family units within a given country. Where there is a tendency toward urbanization and an allocation of food on the basis of income, starvation can occur in the face of national abundance. At the third level is the family's ability to nourish its children, the failure-to-thrive syndrome illustrating what happens when there is a breakdown in a family's ability to provide adequate nutrition for a child. At the fourth level is the child's own contribution to nutritional deficiency, either through the presence of disease, such as cystic fibrosis, or psychological impediments, as illustrated by anorexia nervosa and oppositional feeding disorders.

It is likely that the remarkable resilience and adaptability of the human species accounts for the less than totally devastating long-range effects of early childhood malnutrition. Most sobering, however, is the fact that, although there is a quantitative difference, there is no qualitative difference in malnutrition between the United States and underdeveloped countries. Whether it be kwashiorkor in Africa or the failure-to-thrive syndrome in America, the impact on the child is the same.

For the clinical psychiatrist, this book calls attention to malnutrition as yet another potential source of insult to the CNS, highlighting the need to carefully evaluate the nutritional aspects of a child or adult's background. At the same time, this book succeeds in making the point that even under circumstances of extreme malnutrition, social and family factors are critical in the cause and remediation of later developmental deficits. The inseparability of the CNS and its environment is once again demonstrated.

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The Mentally Retarded Citizen and the Law, *edited by Michael Kindred, Julius Cohen, David Penrod, and Thomas Shaffer; sponsored by the President's Committee on Mental Retardation. New York, N.Y., Free Press (Macmillan Publishing Co.), 1976, 692 pp., $18.95.*

Any major new work on retardation stimulates curiosity about former attitudes and practices. For many years the primary reference work on retardation was Treadgold's *Mental Deficiency*, first published in England in 1908 (1). Reading Treadgold provides a perspective on what has changed and

what has not. Treadgold's dedication still rings true with proper humane concern: "To all those persons of sound mind who are interested in the welfare of their less fortunate fellow creatures."

Curiously, the tables of incidence of retardation, or below-average ability as it now is called, show only a little difference between the 38–40 per 10,000 in England at the turn of the century and the 30–35 per 10,000 in the United States today. Treadgold was interested in legal issues, which are still of concern, and made the following interesting comment:

> For my own part, I'm convinced that the nation which wishes to escape degeneracy will sooner or later have to give serious attention to the matter of the innate constitution of its citizens, and the manner in which that innate condition may be controlled by laws concerning marriage. Nevertheless, I do not think that any legislation on these lines is possible at present, for two reasons: firstly, because the laws regarding hereditary transmission are not sufficiently known; and, secondly, because we have no data regarding the antecedents of the mass of the people. (1, p. 538)

On the other hand, the issues of concern have shifted considerably. Treadgold's text is overwhelmingly preoccupied with taxonomy, classification, and clinical descriptions of the rarest forms of retardation, even though it acknowledges that these are of the least social significance. Of the 600 pages, only 10 relate to the laws concerning "amentia," only 23 to treatment and training, 32 to sociology, and a scanty and not very useful 2 pages on remedial measures in the psychosocial sense.

Even at the turn of the century, however, the mentally retarded were recognized as present and future citizens. Today this is reflected in the change of name of the National Association for Retarded Children to the National Association for Retarded Citizens. Treadgold tersely commented on society's obligations to these citizens throughout his text, but one comment, still applicable, is interesting enough to be repeated: "How illogical is the system which spends thousands upon the training of mentally defective children, and then turns them adrift to shift for themselves as best they can!"

In May 1973 the President's Committee on Mental Retardation held a conference to review progress as to the legal rights of mentally retarded citizens and to consider the obstacles to realization of these rights. The proceedings are published in this handsome, readable volume *The Mentally Retarded Citizen and the Law*. Psychiatrists will find much here that is useful; e.g., the development of concepts, legal and otherwise, related to the retarded have proceeded in parallel with those for the disturbed, but we are a far distance from the development of a National Association for Disturbed Citizens, even though, as children, they too represent the citizens of the future.

The book contains a bill of rights for the retarded which includes the right to life, education, choice of care, work, sex, family, and equal protection under the law. There are 22 primary position papers, each backed by comments on the positions taken. Even though the volume focuses on the law, it is particularly rich in pithy summaries of the current state of thinking about general issues of retardation. Usually these summaries are in the form of editorial comments binding the separate papers together; by themselves these commentaries are useful to the psychiatrist.

Other useful parts of the book include a chapter by Sorgen on labeling and classification, which is a wonderfully concise summary of the history, attitudes, and current state of mental retardation in the United States. Effland's section on "Trust and Estate Planning" and Heckt's comment on it are excellent presentations of the lawyerly skills needed to manage complicated medical, social, and legal issues. Price's comment on the "Right to Community Services" is a clear reminder and warning of the dangers as well as the seductions of community-based services. Bogg's section on "Quality Control of Community Services" is immediately relevant to psychiatric services in the community, as is Chambers' "Least Restrictive Alternative." The sections range from the craftsmanlike, with sharply defined issues and specific implementable suggestions, to those mainly representing the passionately indignant but informed advocate.

Generally, the papers avoid the use of special languages, tending to see specialness in language as hindering understanding among disciplines. On the other hand, the comments too often simply supplement the position papers; i.e., they are amens rather than critical assessments of positions taken. Perhaps it is too much to ask that a fox could have been invited into the chicken coop, too much like inviting a distiller to a Baptist convention. The guests might have tempered some of their advocacy and shed more light. Indeed, the resources are limited and priorities must be made.

Although the authors pay a great deal of attention to the rights of the below average and to the availability of the least restrictive alternative, they do not pay any attention to the similar rights of the average and the above average to the same least restrictive alternative. Perhaps it is the conflicts that keep progress toward common goals at an agonizingly slow pace. Only once, in a section on zoning restrictions, is there a glimpse of the "other side": Deutch argues his objections as a neighbor to group homes and the right of the retarded to live in the community. Even here, however, advocacy takes precedence over working through the resolution of conflicts generated and the solution of complex human problems.

REFERENCE

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HAROLD BOVERMAN, M.D.
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The Origin of Consciousness in the Breakdown of the Bicameral Mind, *by Julian Jaynes. Boston, Mass., Houghton Mifflin Co., 1977, 446 pp., $12.95.*

This is a fascinating book to read. In his range of interests Jaynes includes neurology, archeology, ancient history, and modern social phenomena. He has the reportorial skill of a Tolkien. In his rhetoric he is as startling as Freud was in *The Interpretation of Dreams*, and Jaynes is equally adept at forcing a new view of known human behavior. Whether he will be as successful as either Tolkien or Freud remains to be seen, but it seems to me that the potential is there.

The term "bicameral mind" might be unfamiliar to some readers. "Bicameral" means two chambers, of course, and Jaynes uses it to refer to the left and right temporal hemispheres of the human cortex. Those of us who studied our

neurology some decades ago are generally aware that speech mechanisms are located on the left (in right-handed people). Although the right hemisphere could take over some functions of the left if the left were injured, we were taught that speech was the exclusive province of the left. This assumption has changed on the basis of massive neurological researches in the last two or three decades. For example, there is growing evidence that intuitive as opposed to deductive comprehension seems to be located on the nondominant side of the temporal cortex and that the dominance of the right side shifts during some sleep activities. The famous Penfield experiments provide additional examples.

Starting primarily with Penfield's work, Jaynes develops the thesis that until 3000 B.C. mankind was bicameral and that bicameral man spoke and acted out of impulses originating on the left cortex (in the right-handed) but received the impetus for any such action from the right cortex. The impetus from the right was not in the form of visual or motor activity but was experienced as actual sounds—voices heard within—dominating and controlling man's activities. The right cortex was especially active whenever man was faced with a stressful situation, either threatening or unprecedented. The voices were direct, often correct, and followed as if direct orders. It is not clear whether these voices were first believed to be part of oneself or voices of the gods; it seems clear to Jaynes that they were soon understood to be from the gods.

When this primitive system brought animal-like carnage and destruction to much of the world (there was no mercy, no pity, no comprehension of others, merely narcissistic pillage), the voices, so like the voices of the schizophrenic patient today, occurred less and less. At this point, when man could no longer act, feel, or behave as a direct response to his voices, he was forced to introspect, to take some responsibility for his own actions, even hiding them or inhibiting them, if appropriate to this judgment of the situation. This is the origin of consciousness.

Of course, the development of adequate consciousness is still going on and often fails, but Jaynes believes the environment is capable of influencing the brain, in the manner just described, so he is optimistic for the future. However, he paints a drab picture of the thousands of years of disparate attempts to reinstitute the lost voices of the gods. He devotes the last section of this book to present-day survivors of the bicameral mind: hypnosis, religious conversion, schizophrenia, and all the magical efforts to learn what to do—from the auguries of astrology to the auguries of scientific mysticism.

The central section of this book is a Tolkien-like excursion through *The Iliad* and *The Odyssey*. Jaynes feels that the former is bicameral in that action is direct, never devious or introspective on the individual level, while the latter represents the struggle away from bicamerality toward consciousness. However, Jaynes presents archeological data, poetry, and ideas from so many disciplines that the reader begins to wonder how "one small head could hold all that he knows." I doubt if there exists a reader sufficiently skilled in all of these disciplines to know how to evaluate this book.

In neurology, I feel that Jaynes cites Penfield too literally and fails to cite other recent experimental work, especially that having to do with the immense interconnections among other portions of the brain besides the temporal cortex. In psychiatry, Jaynes seems more oriented; he uses and cites the pertinent recent researches.

The book is never dull. It forces a revision of customary thought patterns and may be germinal. It is not a book for readers seeking more data in a particular field of thought, be it behaviorism or psychoanalysis. It will be stimulating and provocative for all other readers. This book was reviewed in the *New York Times* (March 13, 1977) by Gerald Jonas, who, according to the *Times*, "comments regularly on science fiction."

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Milwaukee Wis.

Chronic Childhood Disorder: Promoting Patterns of Adjustment, *by Ivan Pless, B.A., M.D., and Philip Pinkerton, M.B., M.D., D.P.M. London, England, Henry Kimpton (Chicago, Ill., Year Book Medical Publishers, distributor), 1975, 240 pp., no price listed.*

In his foreword to this excellent book, Dr. Robert Haggerty reminds us that chronic disease is the major health problem of children today. Drs. Pless and Pinkerton have pulled together a vast amount of information about the many sorts of chronic disorders that afflict children, and they have done it in a concise, organized, and clear way that I found both illuminating and a pleasure to read.

The authors deal first with the concept of adjustment, presenting an integrated model of adjustment to chronic disorder. They are aware that their particular idea of a "significant" and "causal" relationship between maladjustment and chronic disorder may be argued against, but they feel that the wide-ranging evidence they present might best help the clinician caring for these children. In discussing the assessment of adjustment, they show a careful effort to evaluate many studies on the subject and many points of view. This is a difficult task, but I found the well-reasoned and dispassionate tone of the authors reassuringly sensible. There is no feeling that special theoretical views are being adhered to at all costs, and as Dr. Tizard mentions in his foreword, "There is . . . a marked agreement in the findings of many investigators, working more or less in ignorance of each other and concerned with children (and families) whose diagnoses and disabilities are apparently very different."

Pless and Pinkerton cite studies that reflect the long-term impact of chronic disorder on adolescents and adults. They remind us again of the need for early intervention to try to minimize difficulties in scholastic and vocational adjustment. There is an excellent chapter on short-term adjustment to chronic disorder; this is divided into sections on such general issues as scholastic adjustment of the large group and on specific problems of particular disorders, allowing the reader to review areas of his or her own interest.

The final chapter takes up the subject of therapeutic intervention to promote adjustments, carefully breaking down the determinants, strategies, and emergent principles of intervention. Finally, the authors reemphasize that the key to successful treatment is the positive relationship of confidence and trust among physician, patient, and family.

This is a relatively short volume that impresses me as containing a great deal of well-thought-out information, presented in an easy style, and including an extensive bibliography. The book should be valuable to pediatricians and to a wide range of physicians caring for disabled children. It should certainly be of interest to the growing number of people involved in the care of chronically disabled children—nurses, nurse practitioners, and physician assistants, for example. I feel it is a particularly good book for medical students learning about disabled children, not only because of the clarity of

its presentation but because it presents a picture of the need for an understanding of all that goes into the care of chronically ill children, written with concern and a broad grasp of the issues.

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Pharmacology of Sleep, *edited by Robert L. Williams and Ismet Karacan. New York, N.Y., John Wiley & Sons, 1976, 347 pp., $26.00.*

This highly condensed volume of papers includes both extensive reviews and specific clinical studies. It represents a useful addition to workers in this field. Some chapters, such as "Concepts and Constructs About Sleep" by Salamy, are superb summaries of the entire field up to the present moment. Chapters like this are quite useful to new workers because they can quickly go over what has been discovered and its history and be brought up to the frontier of what is being worked on at the present time.

Most of the chapters suffer from the problem of breadth and insufficient depth. The review of drug effects by Kay and associates covers studies on a large number of drugs and their effects on sleep but gives no clear critical comments about these studies or a summary that could serve as a guide to the practicing physician. Indeed, there is such a preponderance of abbreviation in this chapter that there is a page of summaries of the various abbreviations used. This abbreviation page is an index of one of the problems of this volume—to whom is it addressed?

It is fairly narrowly addressed to workers in the area of sleep and drugs, despite frequent attempts throughout the volume to make it useful to the clinical pharmacologist. A brief concluding chapter about poor prescription practices represents an attempt to make it useful to the psychiatric or general medical practitioner, but this falls short of being truly useful.

In summary, the vast amount of work that has gone into this volume and the material it covers make it useful for libraries, but it is not a volume for purchase by the individual practitioner unless he or she is specifically working in the area of sleep and pharmacology. Even then the practitioner may elect to use it more for its bibliographic completeness than for the quality of the articles.

ROY M. WHITMAN, M.D.
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Psychiatry. Medical Outline Series, 3rd ed., *by Merrill T. Eaton, Jr., M.D., Margaret H. Peterson, M.D., and James A. Davis, M.D. Flushing, N.Y., Medical Examination Publishing Co., 1976, 487 pp., $9.00.*

This book, now in its third edition in the United States and published simultaneously in six other countries, deserves its popularity as a concise review of psychiatry in a manageable size. Although the authors suggest that it might also be used as an introduction to clinical psychiatry, its succinctly didactic style, often accompanied by dogmatic statements, may tend to alienate those for whom this book is their first contact with our specialty. Having expressed these concerns, I can highly recommend this text as an ideal refresher and review of the essentials of personality development, psychodynamics, nosology, and the major clinical entities.

The discussions of psychotherapy, the somatic therapies, and psychiatric practice in community facilities are pertinent, relevant, and significant for modern-day practice. In the area of psychopharmacology, advances in neurochemistry and pharmacokinetics are being made with such rapidity that it is nearly impossible for any general textbook to be sufficient in itself because of the publication time lag. Up-to-date information about such clinically significant areas as drug dosage, methods of administration, blood levels, and clearance need to be supplemented by other resources, such as journal articles.

There is a very helpful chapter on psychiatry for the primary or family practice physician that includes advice on how to make referrals to psychiatrists, other therapists, or community facilities. There is also a chapter on forensic psychiatry and what I think is an overly foreshortened glimpse of the history of psychiatry. The legal chapter not only includes the usual elucidations of criminal responsibility, testamentary and contractual capacity, confidentiality, and involuntary hospitalization but also discusses the increasingly important aspects of medical ethics.

At the end of each chapter there are review questions that require both factual knowledge and some original creative and judgmental thinking to be answered comprehensively. The final chapter, titled "Reviewing for Board Examinations," is a storehouse of fairly good advice on testsmanship interspersed with an occasional touch of inelegant advice, e.g., for factual data to be memorized one may use "the inside of the bathroom door . . as a good bulletin board for such things (if you have a tolerant spouse)." This is concluded with a long multiple-choice examination composed of 165 test items and their correct responses.

The bibliography has been updated from the earlier editions, but it is somewhat sparse in parts. The authors do not presume to present this book as a substitute for the more voluminous comprehensive texts. In sum, there is a richness of information in this relatively small book, presented in a lucid and readable style that can serve as an excellent review of clinical psychiatry for medical students, physicians, and psychiatric residents, especially those preparing to take state or specialty board examinations.

S. MOUCHLY SMALL, M.D.
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Obsessional Neuroses: Developmental Psychopathology, *by Humberto Nagera, M.D. New York, N.Y., Jason Aronson, 1976, 222 pp., no price listed.*

Psychoanalytic theories have been altered and transformed since Freud's earliest discoveries. The cornerstone of psychoanalytic theory, however, has remained the clinical study of individuals with varied disturbances. More abstract conceptualizations, some going beyond the clinical dimension, have emerged from such clinical investigations. Some of these changes were directly initiated by Freud and others by his colleagues, students, and successors. Despite these applications and extensions, psychoanalysts are still intimately linked to their patient work.

Obsessional Neuroses approaches these disorders from the developmental point of view—a perspective that has characterized the research of the Hampstead School established by Anna Freud. Dr. Nagera worked for many years as a close colleague and student of Anna Freud. In fact, this

monograph is presented as a tribute to her on the occasion of her 80th birthday.

Nagera has written many other psychoanalytic essays, reports, and books. In this treatise he presents the systematic study of one of the classical disorders originally investigated by Sigmund Freud. Nagera's talent for clearly organizing and stating his own views as well as those of others makes this volume very readable. He begins with a review of Freud's formulations and theoretical discussions that is comprehensive yet not complicated. Appropriate references and quotations allow the reader to follow Freud's development of thought as his experience increased. The second chapter is equally comprehensive in its coverage of the contributions of other psychoanalysts to our understanding of obsessional neuroses.

In the following chapters, the author explains his current theoretical positions and then devotes a chapter to three in-depth clinical examples. The cases selected are those of younger children and adolescents. These reports are well organized and carefully presented; the reader can closely follow the assessment of the patient initially and during the treatment process. It is my belief that this clinical chapter will be of great use to students as well as more experienced clinicians. It can serve as a model for meaningfully condensed and ordered clinical reports as well as a source of data. A comparison of obsessional and hysterical personalities, a discussion of the developmental approach to psychopathology, and a concluding chapter on general and diagnostic considerations complete the volume. The bibliography is appropriate, as is the index.

The foreword by Anna Freud is an excellent overture to the book. Sigmund Freud characterized the obsessional neuroses as the most interesting subject of analytic research. Some may have found other areas of analytic study to be of equal interest, but Nagera's book positively buttresses Freud's observation. This monograph is highly recommended to serious students, researchers, and clinicians.

GEORGE H. POLLOCK, M.D., PH.D.
Chicago, Ill.

The Creativity Question, *edited by Albert Rothenberg and Carl R. Hausman. Durham, N.C., Duke University Press, 1976, 366 pp., $14.75; $7.95 (paper).*

The editors, a psychiatrist and a philosopher, have assembled brief excerpts from the contributions of 45 people on the subject of creativity. These span 2,000 years and an equivalent diversity of disciplines; they would be cumulatively chaotic were it not for a considerable editorial effort to organize them so that they illustrate the development of the various contemporary attitudes toward the subject. Even so, the thesis that all this material relates to a single subject is so bewildering that halfway through the book I began to suspect the editors of cleverly arranging a test of my own creative ability. Perhaps after a suitable period of incubation I was to attempt that novel synthesis of apparently unrelated material which many assert is the mark of creativity. With dismay I realized I was failing; I heard a babel of definitions, approaches, and bits of theorizing instead of a beautiful synthesis.

How can a phenomenon (creative people and products) whose definition is so thoroughly subjective be studied? The approaches in this book range from the most sublime and awesome to the most banal and redundant. Plato refers to the work of genius that is said to derive from divine or supernatural motive forces. At the other extreme are those who define a creative person as one who makes unusual responses on psychological tests, those who are intent on teaching problem-solving skills to schoolchildren and businessmen, and those whose "theories" are little more than reductionistic dichotomies. I concluded that creativity is one of those subjects about which much is written but little is known.

Given the Herculean task the editors have undertaken, they have done a good job selecting and organizing the excerpts, introducing each with a succinct summary and providing an overall frame of reference with which to view them. In making an attempt to embrace both diversity and brevity in a single volume, however, the editors invite criticism about whose work they have included, whose they have omitted, and which fragment of a person's work is considered representative as well as skepticism about the result of condensing whole books and papers by such people as Plato, Aristotle, Freud, Rank, Jung, and Bergson (to name a few). From a psychoanalytic perspective (1), I think the Freud excerpt is not representative. Such seminal thinkers as Greenacre are not included at all. On the whole, however, the book is an interesting introduction to the almost anarchic diversity of writing on creativity; it would make an excellent source text for a course.

The book stimulated me to conclude that there may be many creativity questions rather than just one, as the title implies, and that they remain to be differentiated. Some may be more fruitful to pursue than others, and it is my guess that these are not the grandiose questions that have lured so many people of genius to speculate about creativity. Indeed, the more a phenomenon is truly original rather than merely unusual, the more the approach to it may be limited to retrospection, if any approach is possible. Phenomena that are unusual but not unique may be susceptible to attempts at predictive as well as postdictive understanding.

The Bergson excerpt most compellingly states the case that a creation is novel, unfamiliar, and to a significant extent removed from the deterministic realm. Therefore, the genuinely creative, as contrasted with the merely unusual, may pose an unanswerable question.

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1. Robbins MD: On the psychology of artistic creativity. Psychoanal Study of Child 24:227–251, 1969

MICHAEL D. ROBBINS, M.D.
Newton, Mass.

Houdini: A Mind in Chains. A Psychoanalytic Portrait, *by Bernard C. Meyer, M.D. New York, N.Y., E.P. Dutton & Co., 1976, 192 pp., $10.00.*

How does one review a "psychoanalytic portrait" or a psychohistory, particularly when one is neither a psychoanalyst nor a historian? It would seem one may do so by judging the accuracy and thoroughness of biographical data, the style, the content regarding the subject and related elaborations, and the cohesion and apparent logic of psychoanalytic speculation. Certainly Houdini (born Ehrich Weiss) is one of the most fascinating of men—a narcissistic, grandiose, mean, egocentric, daring, expansive person whose life

was riddled with unique psychopathology and unique accomplishment.

He led a magical life, transmuting magical thinking into action. As a magician, escapologist, actor, and writer, he played so many roles that the "real" Houdini remains a wispy character who cannot be readily grasped. Meyer has a psychoanalytic heyday with Houdini's denial of death and mortality, the symbolism of his feats, his castration and anxiety fears, preoccupation with rebirth and resurrection, ambivalence, sibling rivalry, multifold identifications, and counterphobic tendencies. Above all and encompassing all is the odd romance between Cecilia Weiss and her adoring son Ehrich. Whatever one's background and attitude toward psychoanalysis, one can readily see Oedipus wrecks in the shambles of the relationship of Houdini and his mother.

Houdini's works are replete with womb to tomb entrapments and liberations, dismemberments and reincorporations, denial of death, vacillating spiritualism, and God playing. This bizarre, childless man, whose activities symbolized everything from erection to resurrection and from bondage to ectoplasmic liberations and preoccupations, was the first to fly an airplane in Australia.

With all this exotica, Houdini comes across as an empty human; when the character armor is removed, there is little underneath. Many observations are based on known biographical data; others are speculations derived from psychoanalytic theory. In spite of the fact that Houdini's psychodynamics seem blatantly in keeping with traditional Freudian sexual theories, little seems to be known about his actual sexual life and practices. Perhaps more could have been said about the cultural determinants in this son of a poor Jewish immigrant (a rabbi in the midwest), one of many of the rabbi's children who married Christians, who was so uncharitable, untrusting, and unable to share and yet who had an affinity toward the mentally ill and the criminal.

Meyer's vocabulary is imposing, and often the writing is most artful, although, as with much psychoanalytic prose, it is repetitious at times. There are digressions to various personalities who impinged on Houdini's life, including the Conan Doyles of Sherlock Holmes fame. Interestingly, Meyer's son, Nicholas, introduced Sherlock Holmes to Sigmund Freud in *The Seven-Per-Cent Solution* (1). Bernard Meyer blurs the distinction between biographical reality and fiction when he states in a footnote, "Recent investigations indicate that in 1891 Sherlock Holmes was in Vienna, where he was treated for cocaine addiction by Sigmund Freud" (p. 159).

This book is an interesting look at a fascinating person who seems more fictional than real. After reading *Houdini: A Mind in Chains*, one is stimulated to want to know more about what he was really like when he was not acting. This psychoanalytic scrutiny is enlightening, entertaining, and informative, although possibly fraught with some unfounded speculation. The reader is likely to feel somewhat unsettled by the biographical and psychological developmental gaps. Perhaps an alternative title could have been *The Seventy-Per-Cent Solution*.

REFERENCE

1. Meyer N: The Seven-Per-Cent Solution. New York, EP Dutton & Co, 1974

IRWIN N. PERR, M.D., J.D.
Piscataway, N.J.

Successful Psychotherapy, *edited by James L. Claghorn, M.D. New York, N.Y., Brunner/Mazel, 1976, 203 pp., $12.95.*

This book is a collection of papers given at the 1975 annual symposium of the Texas Research Institute of Mental Sciences in Houston. The contributors include well-known authorities in the field. Its scope is broad, and, in the words of its editor, it has an eclectic, deliberately ecumenical outlook. According to Dr. Claghorn, "The recipe for intervention can now be viewed as multiple and as varied as the needs of the patient." In keeping with this, the authors offer a range of approaches, emphasizing issues and techniques currently enjoying special interest, all of which are given approximately equal standing. The selection is therefore fairly representative of current trends in American psychiatry. This makes for variety and liveliness but not for cohesiveness. In fact, the reader may find himself or herself stimulated by the diversity of the chapters but wishing for more unity and continuity in content and philosophy.

A look at the topics covered should serve to clarify this impression. There are papers on psychotherapy research (e.g., on the assessment of outcome, therapeutic components, and forms of helping alliances); integrating psychotherapy and pharmacotherapy in the treatment of schizophrenia and the neuroses; management of chronic psychotic patients; the psychotherapeutic technique of "reframing"; family therapy in anorexia nervosa; sex therapy; and the treatment of women, black people, and the poor. This selection represents a large enterprise for a book of just over 200 pages.

Successful Psychotherapy deals not so much with the actual conduct of psychotherapy or its inner complexities as with its relation to other elements of treatment (e.g., drugs, behavioral techniques, and the impact of social forces). Even though Hans Strupp cautions on this very point, one notes overall a tendency to speak of psychotherapy generically, as if it were all of a kind. This is most noticeable in the discussion of the usefulness of combining psychotherapy with other modalities of treatment, which the editor and several contributors regard as the coming practice. There seems to be an assumption that all psychotherapy is equally capable of being combined with other treatments. However, although it is evident that supportive, "relationship" therapy mixes readily with pharmacotherapy, other modalities, such as psychoanalytic psychotherapy, may not mix so well. This certainly is one of the many questions about psychotherapy that calls for study.

The book contains its fair share of stimulating and worthwhile presentations. Of special interest is the section on psychotherapy and social issues. Matters of controversy are treated reasonably and with restraint. For example, writing on depression in women, Myrna Weissman suggests that a prime factor may be the status of women in society and their "learned helplessness." She bases her argument primarily on the finding that depression appears to be more common in women. Although she does not present her views as assertions and is aware that methodological problems abound in this area of research, she also does not quite confront the question of whether depression is really different clinically in women and men. The basis for the difference in prevalence, if it is a valid difference, remains unclear.

Also of interest is a paper by Alvin Burstein on psychotherapy for the poor. With liveliness and humor he challenges two propositions that became instant dogma in the late 1960s. One is the idea that insight psychotherapy is only

for middle-class patients, and the other is the claim that indigenous paraprofessionals can substitute cultural congruence for technical training.

The examples cited above should suffice to convey the flavor of the book. *Successful Psychotherapy* offers the reader a good cross section of American psychiatry in the mid-1970s.

PIETRO CASTELNUOVO-TEDESCO, M.D.
Nashville, Tenn.

Personality, *edited by Rom Harré. Totowa, N.J., Rowman and Littlefield, 1976, 246 pp., $17.50.*

This book consists of seven essays organized into three sections on personality acquisition, description, and assessment. Although most of the essays have been published elsewhere, the material is not likely to be familiar to readers in the United States. Most of the contributing authors are British, and their ways of thinking about personality and personality research are quite distinct from the view of these subjects in the United States. In fact, the book can fairly be seen as a response to Walter Mischel's impressive attack on trait theories (1) by a group of personality researchers who, for good reasons, were unwilling to join him in embracing situationism as an alternative. In *Personality*, we have efforts to redefine the problems that the personality researcher seeks to solve, to create new methods of investigation, and to employ new explanatory constructs with sounder theoretical underpinnings than those used in the past.

Two articles discuss the issue of personality consistency directly. Alston provides a careful textual analysis of Mischel's work, and Argyle presents an interesting and well-organized review of the empirical research. Argyle then introduces a generative-rules approach to social behavior borrowed from psycholinguistics and discussed too briefly to have a real impact. Ziller offers a hierarchical theory of personal change that integrates disparate research in attitude change, behavior change, role strain, and self-concept. Clinicians will probably find this chapter to be the richest of the seven in its implications for their work. Other authors and topics include John Bowlby on the child's acquisition of self-reliance, Harré on the social and psychological aspects of personal names, and Shotter on what he calls the development of personal power as opposed to natural power in children.

A unity of sorts runs through these articles. All, or nearly all, express dissatisfaction with the current state of personality research, all eschew "naïve experimentation," and all are willing to draw on a wide variety of disciplines (e.g., psycholinguistics and ethology) for conceptual and methodological help. In most cases, personality is not defined in intrapsychic terms but as a set of social competences; most of the problems of interest here would be considered within the domain of social psychology in the United States. The editor freely admits that none of the essays constitutes a finished treatment of its topic. Rather, they may be regarded as promissory notes that signal a change in direction in personality research and whose promise may or may not be fulfilled.

Although I welcome the novel approaches to the study of personality exemplified in this book, I have two criticisms. First, the reading level is generally difficult. Second, new terms are introduced at times without sufficient discussion of why older, more familiar terms are no longer suitable. Shotter's concept of personal power, for example, shares much in common with such concepts as internalization, ego autonomy, and socialization, yet there are no references to these concepts or to the research literatures they subsume. In the absence of such references one is left wondering whether the author runs the risk of rediscovering the wheel. More of a sense of continuity with earlier personality theories and research might have made this book more influential than it is likely to be.

REFERENCE

1. Mischel W: Personality and Assessment. New York, John Wiley & Sons, 1968

PETER B. ZELDOW, PH.D.
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The Bipersonal Field, *by Robert Langs, M.D. New York, N.Y., Jason Aronson, 1976, 462 pp., $20.00.*

According to Dr. Langs this book is intended to provide clinical data to substantiate the concepts he developed in his two volumes on *The Technique of Psychoanalytic Psychotherapy* (1). To do this he has edited and reworked audiotapes of psychoanalytically oriented psychotherapy seminars he conducted with a group of students. The case material in the book is thus gathered and presented by student therapists and then discussed by Dr. Langs with the students. Dr. Langs lucidly illustrates theoretical concepts and principles of psychotherapeutic technique by using the case material presented. Because the book contains many precisely documented and clearly discussed clinical vignettes, it gives an excellent and solid demonstration of some of the technical principles of psychoanalytically oriented psychotherapy. In addition to illustrating certain aspects of the psychoanalytic psychotherapeutic technique, the book also reveals Dr. Langs's method of teaching psychoanalytic psychotherapy.

The main criticism I have of this otherwise clinically and didactically useful book is that the author employs new words whose meanings differ only slightly, if at all, from the more usual and standard psychoanalytic terms. A significant portion of the book is spent defining and explaining these new words. I will mention and define a few of these unfamiliar terms as I summarize some of the valuable content of the book.

The author stresses that this book is a "major contribution toward the integration of the interactional dimension into classical psychoanalytic theory and technique." To promote this integration, he borrows the phrase "bipersonal field" from the Barangers. He uses this phrase metaphorically to describe "the interplay between the interactional and intrapsychic realms and between the patient and the therapist." The concepts denoted by the phrase "bipersonal field" have been handled effectively in the past by using such generally accepted and understood terms as "therapeutic interaction" and/or "transference situation." When Dr. Langs correctly stresses the central importance of considering the bipersonal field in psychoanalytic psychotherapy, he is reiterating Freud's emphasis on the central importance of the transference situation in psychoanalytic therapy.

A considerable and important portion of the book is taken up with clinical data that clearly demonstrate Freud's impor-

tant, but at times overlooked or forgotten, principle that the therapist should maintain a state of relative abstinence; that is, the therapist should avoid gratifying any of the patient's transference needs. Dr. Langs presents a strong and convincing argument for the maintenance of the strictest abstinence, but unfortunately he uses new terms to describe this old principle. By "framework," "boundary," or "frame" he means the realistic, therapeutic contract between the therapist and the patient. When he stresses that it is important for the therapist to maintain the framework, boundary, or frame, he means that the realistic, therapeutic contract between the therapist and the patient should be preserved to avoid gratifying the patient's transference wishes. Dr. Langs calls a break in the therapeutic contract a "modified frame"; there is a "misalliance" when, due to collusion between patient and therapist, the therapeutic contract is broken and curative efforts are avoided. By "misalliance cure" Dr. Langs means what Freud called a "transference cure" based on transference gratification.

The author uses the phrase "adaptational context" to refer to the patient's world situation, which is composed of inner and outer worlds. He refers to projective and introjective identifications as interpersonal analogues of intrapsychic projections and introjections, and he states that it is the therapist's "container function" to serve as the receptacle of such projective identifications.

In the last chapter Dr. Langs demonstrates with clinical material that some patient behaviors which could wrongly be interpreted as transference manifestations are actually reactions to real messages unconsciously sent by the therapist.

This book lucidly illustrates some important principles of psychotherapeutic technique. It should prove valuable to all psychotherapists.

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1. Langs R: The Technique of Psychoanalytic Psychotherapy, vols 1, 2. New York, Jason Aronson, 1973, 1974

FELIX F. LOEB, JR., M.D.
San Diego, Calif.

Progress in Behavior Modification, Vol. 3, *edited by Michel Hersen, Richard M. Eisler, and Peter M. Miller, New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1976, 342 pp., $18.00.*

This book represents the third in an annual series of original reviews of various topics in behavior modification. The editors promise a diversity of subjects, and this volume more than fulfills that promise by presenting topics as diverse as hypnosis on the one hand and animal analogues of treatment procedures on the other. The editors' rationale for these volumes is that "even the most diligent reader will find it difficult to keep abreast of all of the developments in the field." If the field has become as broad and far-ranging as the list of subjects indicates, one logical question would be, Is there anyone who wants to keep abreast of all these topics? To answer this question for myself I decided to read carefully every contribution, including some with which I have only passing acquaintance, such as animal analogues and learning disabilities.

The subjects in this volume can be divided into three areas: clinical applications (smoking, learning disabilities, sexual disorders, and classroom applications), discussions of research strategies (animal analogues of behavioral treatment procedures and analogue therapy outcome research with humans), and, finally, two theoretical discussions—one on a comparison of hypnosis and behavior therapy and one on transferring treatment effects outside of therapeutic settings.

The two theoretical discussions are excellent. The lead chapter by Spanos and Barber is alone worth the price of the book in synthesizing two large but hitherto unrelated bodies of research. The authors review Barber's well-known work on the illogic of the hypnotic state and nicely summarize the hypnosis research of the last 15 years. They then integrate this research with the growing literature on the importance of cognitive influences in behavior therapy and suggest a process they call "involved imagining" as critical to both the hypnotic state and many of the behavior therapy approaches (as well as other psychotherapeutic approaches). This line of reasoning makes excellent sense and should be studied by all who are interested in psychotherapeutic processes. The final chapter by Marholin, Siegel, and Phillips is also an excellent review of the difficult issues involved in determining that therapeutic changes occurring in the office, clinic, or school transfer to other settings, where these changes are most desired.

Lichtenstein and Danaher contribute a comprehensive and up-to-date review of smoking. After reviewing data on psychological and pharmacological ideas of why people smoke, they describe the different measures of smoking, including some new "breathalyzer" techniques, which provide more objective measures. A thorough review of various treatment strategies underlines the many failures in this difficult area, but the authors conclude by summarizing treatments that have been demonstrated effective and suggesting methods by which these treatments might be made more effective.

Copeland and Hall's chapter emphasizes recent shifts in the targets of research in classroom applications from controlling disruptive behavior to enhancing academic performance. They review the latest developments in accomplishing these goals. Lahey's chapter on learning disabilities also provides an excellent, up-to-date review of this area that should be of interest to physicians, psychologists, and educators.

In their chapter on sexual disorders Ascher and Clifford note that most sex therapy is not currently supported by controlled experimentation and that this forces them into a review of the state of the art of sex therapy rather than a data-based review of treatment effectiveness. The authors accomplish their goal by comparing the classification and treatment procedures of Wolpe, Masters and Johnson, Kaplan, and others. They follow this up with an interesting discussion on where this field is going.

Discussing basic research approaches, Adams and Hughes describe the purpose of animal analogue experiments as to learn something about the prediction and control of behavior as it occurs in more complex human environments. Borkovec and O'Brien review human analogue situations, most often subclinical fears. I harbor considerable doubt about the value of analogues of human treatment efforts; yet these chapters provide a very good review of these areas that is appropriately critical but points out where such approaches can be useful.

The diversity of chapters in this book indicates that behavior therapy is probably losing its identity as a unified approach and disintegrating into various subtopics, clinical entities, or areas that have traditionally divided the broad area of behavior change. This is the fate of all new approaches that have proved their usefulness and become integrated into

the various areas of specialization. I enjoyed reading every chapter in the book (the first edited book I have read in years that did not contain at least one poor chapter). It became apparent in doing so that whatever one's area of specialization, much can be learned from the successes and failure of clinicians and researchers working in other areas. From this point of view, this volume serves a useful function which is valuable to everyone working in the area of behavior change.

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The Reality Therapy Reader: A Survey of the Work of William Glasser, M.D., *edited by Alexander Bassin, Ph.D., Thomas Edward Bratter, Ed.D., and Richard L. Rachin, M.P.A. New York, N.Y., Harper & Row, 1976, 673 pp., $15.00*

In his foreword to this compilation Dr. Glasser says, "The material here will not stand by itself—it is designed to supplement my books, and all of my books, starting with *Reality Therapy*, are a necessary prelude to what is printed here." He goes on to say that even then one could not learn the technique of reality therapy from books without supervised instruction. He is probably right, but his caveat need not discourage the potential reader who wants an overview of Glasser's ideas and contributions.

Reality therapy rejects the concept of mental illness, the therapeutic relevance of the patient's history, the importance of unconscious conflicts, and transference. It emphasizes "the morality of behavior" and attempts to "teach patients better ways to fulfill their needs." If one is not troubled by the apparent ambiguity in denying that mentally ill people are sick (whatever that may mean; "sick" is derived from the Middle Irish *socht*, meaning "depressed") but referring to them as patients and offering them treatment, the basic ideas are not difficult to grasp.

The reader contains papers by 31 contributors, of whom 4, including Glasser, are physicians. The book is divided into six parts. The first is titled Glasser the Man and includes several papers by and about him as well as interviews with him from the *Los Angeles Times* and *U.S. News & World Report*. Part two deals with the theory of reality therapy and part three with its practice.

Part four, on education, contains papers reviewing Glasser's contributions to this subject, including those contained in his book *Schools Without Failure* (1). In advocating that schools eliminate the failure orientation, which he feels is destructive to the self-esteem of students, he does not favor the elimination of standards and requirements. The principles of reality therapy are used, partially through class meetings, to help students solve problems, develop social responsibility, and acquire a sense of self-worth.

Part five, on corrections, deals with the use of reality therapy in correctional institutions. Part six, on role playing, consists of transcriptions of three tapes of Glasser's role-playing sessions. All three are initial simulated patient interviews, and in each a main goal seems to be to establish a therapeutic relationship with a poorly motivated patient or client.

The papers chosen for this book uniformly reflect an enthusiasm for reality therapy. Although there are some comparisons of the theoretical basis of reality therapy with that of other treatment modalities, one does not find anything that might be regarded as a critical appraisal of Glasser's ideas. One also does not find studies comparing the outcome for people treated, educated, or corrected by Glasser's methods with control groups served by other means except for one wistful note about pupils in a school using reality therapy methods who had "not so far forged ahead in testable qualities." The author then mentions the limitation of the tests used and the fact that these students were not used to taking tests. Perhaps one should not expect a book called a "reader" to furnish critical appraisals or research data.

One might enjoy this book more by reading a paper or two at a time rather than reading it all at once because there is a tendency toward repetition, not just of principles but even of anecdotes. Although this may reveal one of my many biases, I wish that the proponents of new treatments would offer the treatment on its own merits, sparing us repetitious destruction of strawpeople (e.g., "a slashing, no-holds-barred attack on conventional psychiatry and its current psychodynamic Freudian foundation"). Instead of advocating the new treatment as a panacea, I wish they would attempt to establish the conditions for which, or the circumstances under which, the new treatment or treatment modification is most useful.

At the least, the reader of this book will get a general idea of what reality therapy is all about; at best, he or she will get a few ideas that can be applied in conventional, eclectic psychiatric practice. The reader who wants more than this, who, for example, wants to become a reality therapist, should heed the warning in the foreword.

REFERENCE

1. Glasser W: Schools Without Failure. New York, Harper & Row, 1969

MERRILL T. EATON, M.D.
Omaha, Neb.

Marital Communication and Decision Making: Analysis, Assessment, and Change, *by Edwin J. Thomas. New York, N.Y., Free Press (Macmillan Publishing Co.), 1977, 227 pp., $12.95.*

For the experienced behavior therapist, this book is an excellent review. It updates research and presents a step-by-step exposition of procedures to be followed for the assessment and modification of problems in the communication and decision making of married couples. Edwin Thomas is a leading researcher and clinician who has worked effectively in this area of marital counseling. He clearly states that the main target responses in this type of intervention generally turn out to be problems of verbal behavior with a locus in partner interaction. Thus he wisely does not proclaim this approach to be the panacea for counseling of all marital problems. He recognizes that communication problems are one aspect of the total concerns of family members. His emphasis on the correct selection of partners for this approach is that of an experienced clinician. There is a welcome absence of the hucksterism seen in several other recent publications pertaining to the communication area of marital interactions. In keeping with his measured approach is Thomas' recommendation to the counselor that he or she tailor his or her plans for modification procedures to each partner and each couple.

Thomas recognizes that "alpha" problems or referent conditions such as couple decision-making problems take precedence over the communication problem approach. This leads to his devoting an equal amount of space in this book to the remediation of decision-making problems between marital partners.

This is not a beginner's book. Clinical experience and a knowledge of the vocabulary that has developed in this field are necessary to read it easily and appreciate it fully. Many of the statements made here are not explained in detail; the author assumes that the reader has the background necessary for understanding unelaborated statements. To the dynamically oriented psychiatrist, the total acceptance of manifest content and the obliviousness to the latent content shining through material is at first disconcerting. However, when he or she recognizes that the author presents a specific approach to a limited, controlled set of conditions, the approach seems fair.

PETER A. MARTIN, M.D.
Ann Arbor, Mich.

Psychosexual Problems: Psychotherapy, Counselling and Behavioural Modification, *edited by Sidney Crown. London, England, Academic Press (New York, N.Y., Grune & Stratton, U.S. publisher and distributor), 1976, 465 pp., $14.75.*

This book is directed at a broad audience, including not only psychiatrists and psychologists but social workers, counselors, welfare personnel, pastoral counselors, and general medical practitioners. Its principal aims are to "cover" psychosexual problems in groups of patients hitherto not considered (e.g., postsurgical patients and people with venereal disease) and to present principles of therapy and counseling for them. To accomplish these aims the 19 chapters are divided into four major sections: Sex Therapies, Psychosexual Problems and the Life Cycle, Psychosexual Problems in Medicine and Surgery, and Sex Education. All of the authors are English except for one Dane and one North American. The writing varies in style from quaint to scientifically terse to humorous verging on the sarcastic. The tone of this book is quite different from other current material on human sexuality because of its use of such terms as "frigidity" and "perversions" and its heavy emphasis on dealing with paraphilias rather than sexual disabilities in couples.

The first section contains 2 excellent chapters on behavioral and group treatment approaches to sexual problems. These chapters are clear, comprehensive, and highly informative. They are excellent examples of how behavior and group therapists work in general and how they apply their approaches to sexual problems in particular. The other chapters in the section on counseling and psychoanalytic approaches are elementary and rarely specifically relate to sexual problems.

The section on psychosexual problems and the life cycle is the weakest one in the book, not only because the life cycle is not covered but also because it lacks examples and represents a superficial, often antiquated approach to dealing with sexual problems.

The section on sexual problems in medical and surgical practice is very useful in that it deals with many aspects of sexuality not often discussed in other sources (e.g., surgical, urological, and obstetrical conditions). These chapters are usually to the point and clearly written, although they occasionally suffer from outrageous comments, such as, in referring to the attention paid to the clitoris, "To some extent this is a symptom of the West, where women are conditioned to expect orgasm unlike their Oriental sisters, or their simian cousins" (p. 375).

The section on sex education has a generally narrow and somewhat outlandish perspective; e.g., "In a nutshell perhaps the sexually transmitted diseases are best regarded as sports injuries" (p. 446). It is doubtful that any people opposed to sex education would have their opinion altered in the slightest by this section.

Overall, this book has some very positive aspects. It draws attention to the sexual implications of a number of areas hiterto not considered in depth, it reviews some areas of sex therapy very well, and it contains a wealth of information about sexuality. However, it does have problems. The book could have been greatly shortened if all the authors had agreed on one system of classification of sexual problems so that they each would not have had to describe his or her own and if they had not repeated the same material about counseling. There is an undue emphasis on dealing with sexual variations at the expense of covering such frequently seen problems as marital difficulties. The terminology used in the book sounds rather antiquated and could easily alienate many readers (e.g., the use of "frigidity" and "perversions"). It also shifts in its level of psychological sophistication from naïve to virtually confusing.

The book has several deficiencies that severely limit its usefulness. It does not review current concepts of sexual physiology (generally, the most recent reference it gives is Masters and Johnson's work published in 1966), nor how to examine a patient with a sexual complaint from a psychological and physical perspective and establish a differential diagnosis. Many chapters repetitively deal with limited aspects of this issue, but there is no comprehensive discussion of it. Nothing is mentioned about sexual attitude reassessment methods or the use of sexually explicit materials in sex therapy, except for the chapter on behavior therapy. With the exception of the chapters on behavioral and group approaches, a novice would have great difficulty figuring out what to do if faced with a patient who had a sexual complaint on the basis of what he or she learned from this book.

OLIVER BJORKSTEN, M.D.
Charleston, S.C.

Keeping Patients in Psychiatric Treatment, *by Chaim M. Rosenberg and Anthony E. Raynes. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1976, 161 pp., $16.00.*

The special interests of Rosenberg and Raynes are the epidemiological and sociological aspects of psychiatric care, with an emphasis on consumerism. The title implies and the contents of the monograph make explicit the failure of what the authors designate as "the official mental health treatment system" to provide an adequate array and deployment of treatment facilities to intercept people in psychiatric need and, more particularly, to keep them in treatment sufficiently long. The mental health planners are at fault for not surveying the varying needs of the community from area to area. Treatment personnel are at fault for organizing psychiatric treatment facilities that cater to their own personal needs as well as to those of their patients. In consequence of these faults the rate of catchment is pitifully low, and the rate of dropout following referral or assessment is very high.

However, the authors are aware that many dropouts are not caused by either poor planning or any fault of treatment personnel.

The volume comprehensively surveys demographic studies of psychiatric morbidity; the literature on social class in relation to psychopathology, psychiatric referral, and the congruence or incongruence of aims of patients and treatment personnel; and the contentious and the uncontentious papers on rates of spontaneous remission of the less severe psychiatric disorders. It deals with publications on the motivations and needs of patients, which are very different from each other. These several threads are brought together in a survey revealing the occasional success and the frequent failure of the official mental health treatment system to respond effectively to the myriad of variables, particularly the failure to keep the patient to the extent that keeping (an unfortunately chosen verb) is necessary.

The authors do not indicate toward whom they have targeted the book. They bring together a great deal of pertinent information but nothing essentially new to psychiatrists. What was novel 20 years ago in the Midtown Manhattan and the Stirling County studies and in the brilliant social-psychiatric work of Hollingshead and Redlich is familiar today. Little seems to have been added since beyond the predictable spate of studies filling in the details and providing illustrations of those pioneering perspectives of the psychiatric scene.

Perhaps the book is intended for lay planners (if there are any) of psychiatric treatment facilities. It has the persuasive and manifestly thorough quality of a commissioned report for some worthy governmental agency. It deserves a place in the libraries of health science departments and government departments of mental health.

It seems time (at least to me) that the accusation of self-interest on the part of health treatment personnel should be given this final airing and hereafter decently buried. Naturally, the motivations of physicians, nurses, social workers, etc., are mixed and include self-interest. Health professionals must be mentally and emotionally healthy if they are to do their jobs effectively, and this implies properly taking care of their basic personal interests, too. If anything, health professionals are more likely to be concerned with their own psychological and biological self-interests than to those of their clients at large. If they do, as the authors contend, so much the better.

DONALD J. WATTERSON, M.D.
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Gilles de la Tourette's Syndrome, Vol. 1: International Registry, *edited by F.S. Abuzzahab, Sr., M.D., Ph.D., and F.O. Anderson, M.D. Minneapolis, Minn., Minnesota Medical Foundation, University of Minnesota, 1976, 220 pp., no price listed.*

This book is a compilation of papers presented at the 123rd annual meeting of APA in May 1970 or written by authors who could not make it to San Francisco that year. It is therefore dated.

The first section of the book presents an abundance of case histories from the University of Minnesota's international registry and other sources, but these are not drawn together in a way that would increase their usefulness to practicing clinicians or researchers. There are some fascinating observations, such as the increased movements and utterances associated with allergies and infections and reports of cases of Tourette's disease from China and Europe.

The second section of the book is most notable for its presentation of neuropathological findings of a Polish teenager who died in 1969 and the report of increased dopamine excretion before haloperidol (Haldol) therapy followed by return to normal levels after treatment. Unfortunately, the narrative report of decreased dopamine is not supported by the appropriate numerical data.

The third section is a single 16-page paper that puts the material in the previous two sections in perspective; this paper lists 508 references. The principal value of this volume lies in its comprehensive bibliography and as a repository for case histories of primary Gilles de la Tourette's syndrome. Most clinicians will find other sources more useful.

KENNETH M. WOODROW, M.D.
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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Culture and Psychotherapy, *by Theodora M. Abel and Rhoda Métraux. New Haven, Conn., College & University Press, 1974, 317 pp., $12.00.*

Advances in Neurochemistry, Vol. 2, *edited by B.W. Agranoff and M.H. Aprison. New York, N.Y., Plenum Press, 1977, 341 pp., $29.50.*

Quality Assurance in Long Term Care, *by Thomas H. Ainsworth, Jr., M.D. Germantown, Md., Aspen Systems Corp., 1977, 209 pp., $17.00.*

Facial Pain, 2nd ed., *edited by Charles C. Alling III, D.D.S., M.S., and Parker E. Mahan, D.D.S., Ph.D. Philadelphia, Pa., Lea & Febiger, 1977, 267 pp., $22.00.*

Psychiatric Nursing, 5th ed., *by Annie Altschul and Ruth Simpson. Baltimore, Md., Williams & Wilkins Co., 1977, 366 pp., $9.00 (paper).*

Female Psychology: Contemporary Psychoanalytic Views, *edited by Harold P. Blum, M.D. New York, N.Y., International Universities Press, 1977, 438 pp., $22.50.*

The Subnormal Mind, 3rd ed., *by Sir Cyril Burt. New York, N.Y., Oxford University Press, 1977, 383 pp., $14.95.*

Freud and Future Religious Experience, *by Anthony J. De Luca, Ph.D. Totowa, N.J., Littlefield, Adams & Co., 1977, 261 pp., $4.95 (paper).*

Psychotherapy—The Promised Land? *edited by Michael Dinoff, Ph.D., Mildred Elliott Berl, M.A., and Robert L. Vosburg, M.D. University, Ala., University of Alabama Press, 1977, 132 pp., $10.00.*

Anthropological and Cross-Cultural Themes in Mental Health: An Annotated Bibliography, 1925–1974, *by Armando R. Favazza and Mary Oman. Columbia, Mo., University of Missouri Press, 1977, 336 pp., $30.00.*

Insights from the Blind: Comparative Studies of Blind and Sighted Infants, *by Selma Fraiberg. New York, N.Y., Basic Books, 1977, 292 pp., $11.95.*

The Origins of Psycho-Analysis: Letters to Wilhelm Fliess, Drafts and Notes: 1887–1902, *by Sigmund Freud; edited by Marie Bonaparte, Anna Freud, and Ernst Kris; translated by Eric Mosbacher and James Strachey. New York, N.Y., Basic Books, 1977, 462 pp., $5.95 (paper).*

Clinical Biofeedback: A Procedural Manual, *by Kenneth R. Gaarder, M.D., and Penelope S. Montgomery, M.S. Baltimore, Md., Williams & Wilkins Co., 1977, 222 pp., $12.95 (paper).*

Peptides in Neurobiology, *edited by Harold Gainer. New York, N.Y., Plenum Press, 1977, 448 pp., $34.50.*

Old Folks at Homes: A Field Study of Nursing and Board-and-Care Homes, *by Raymond M. Glasscote, M.A., and Allan Beigel, M.D., Alexander Butterfield, Jr., Eleanor Clark, M.S.W., Bonnie A. Cox, M.S.W., M.A., J. Richard Elpers, M.D., Jon E. Gudeman, M.D., Lee Gurel, Ph.D., Ruth V. Lewis, M.A., R.N., Donald G. Miles, Ed.D., James B. Raybin, M.D., Clifford B. Reifler, M.D., M.P.H., and Edward H. Vito, Jr., A.C.S.W. Washington, D.C., Joint Information Service of the American Psychiatric Association and the National Association for Mental Health, 1976, 148, pp., $6.50.*

Promise and Performance: Children with Special Needs. ACT's Guide to TV Programming for Children, Vol. I, *edited by Maureen Harmonay. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1977, 255 pp., $12.50; $6.95 (paper).*

The Hite Report: A Nationwide Study of Female Sexuality, *by Shere Hite. New York, N.Y., Dell Publishing Co., 1976, 638 pp., $2.75 (paper).*

Biofeedback and Self-Control 1976/77: An Aldine Annual on the Regulation of Bodily Processes and Consciousness, *edited by Joe Kamiya, T.X. Barber, Neal E. Miller, David Shapiro, and Johann Stoyva. Chicago, Ill., Aldine Publishing Co., 1977, 604 pp., no price listed.*

Sexual Counseling, *by Eugene Kennedy. New York, N.Y., Continuum (Seabury Press), 1977, 197 pp., $9.95.*

Rites of Passage: Adolescence in America 1790 to the Present, *by Joseph F. Kett. New York, N.Y., Basic Books, 1977, 312 pp., $16.50.*

Supervision of Applied Training: A Comparative Review, *by Dewayne J. Kurpius, Ronald D. Baker, and Irene D. Thomas. Westport, Conn., Greenwood Press, 1977, 264 pp., $17.50.*

Growing Up To Be Violent: A Longitudinal Study of the Development of Aggression, *by Monroe M. Lefkowitz, Ph.D., Leonard D. Eron, Ph.D., Leopold O. Walder, Ph.D., and L. Ro-*

well Huesmann, Ph.D. Elmsford, N.Y., Pergamon Press, 1977, 224 pp., $14.00; $8.00 (paper).

Abrégé de Psychiatrie de l'Adulte, *by T. Lemperière and A. Féline. Paris, France, Masson & Cie, 1977, 423 pp., no price listed (paper).*

The Essential Guide to Prescription Drugs, *by James W. Long, M.D. New York, N.Y., Harper & Row, 1977, 747 pp., $25.00; $8.95 (paper).*

Magie et Mythe en Psychiatrie, *by Pierre Marchais. Paris, France, Masson & Cie, 1977, 204 pp., no price listed (paper).*

Emergence: A Transsexual Autobiography, *by Mario Martino with harriet. New York, N.Y., Crown Publishers, 1977, 273 pp., $10.00.*

Too Old, Too Sick, Too Bad: Nursing Homes in America, *by Frank E. Moss, J.D., and Val J. Halamandaris, J.D. Germantown, Md., Aspen Systems Corp., 1977, 310 pp., $15.95.*

Music Therapy Index, Vol. 1, 1976: An International Interdisciplinary Index to the Literature of the Psychology, Psychophysiology, Psychophysics and Sociology of Music, *by the National Association for Music Therapy, Inc. Lawrence, Kans., National Association for Music Therapy, 1976, 224 pp., no price listed (paper).*

Homosexuality Bibliography: Supplement, 1970–1975, *by William Parker. Metuchen, N.J., Scarecrow Press, 1977, 292 pp., $12.50.*

Puppets and Therapy, *edited by A.R. Philpott. Boston, Mass., Plays, 1977, 153 pp., $4.95 (paper).*

Strategic Psychotherapy: Brief and Symptomatic Treatment, *by Richard Rabkin. New York, N.Y., Basic Books, 1977, 246 pp., $11.95.*

Atlas of Cell Biology, *by Jean-Claude Roland, D.Sc., Annette Szöllösi, D.Sc., and Daniel Szöllösi, Ph.D. Boston, Mass., Little, Brown and Co., 1977, 113 pp., no price listed (paper).*

Psychopharmacology: A Biochemical and Behavioral Approach, *by Lewis S. Seiden and Linda A. Dykstra. New York, N.Y., Van Nostrand Reinhold Co., 1977, 431 pp., $19.95.*

Basic Human Nature . . . Revisited, *by Edward M. Slater. New York, N.Y., Vantage Press, 1977, 62 pp., $4.95.*

Behavior Modification of the Mentally Retarded, 2nd ed., *edited by Travis Thompson and John Grabowski. New York, N.Y., Oxford University Press, 1977, 557 pp., $15.00; $7.95 (paper).*

Case Studies in Medical Ethics, *by Robert M. Veatch. Cambridge, Mass., Harvard University Press, 1977, 413 pp., $14.00.*

Enciclopedia de Psiquiatria, *by Guillermo Vidal Hugo Bleichmar, and Raul J. Usandivaras. Buenos Aires, Argentina, Libreria "El Ateneo" Editorial, 1977, 698 pp., no price listed (paper).*

Brain's Diseases of the Nervous System, 8th ed., *revised by John N. Walton, M.D., D.Sc. New York, N.Y., Oxford University Press, 1977, 1,202 pp., $29.50.*

Psychopharmacology in Childhood and Adolescence, *edited by Jerry M. Wiener, M.D. New York, N.Y., Basic Books, 1977, 221 pp., $15.00.*

Behavioral Approaches to Medical Treatment, *edited by Redford B. Williams, Jr., and W. Doyle Gentry. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.) 1977, 252 pp., $15.00.*

Group Therapy 1977: An Overview, *edited by Lewis R. Wolberg and Marvin L. Aronson. New York, N.Y., Stratton Intercontinental Medical Book Corp., 1977, 293 pp., $22.00.*

Report of the Secretary
Summary of Meetings of Board of Trustees and Executive Committee
May 1976–May 1977

IT IS MY final privilege as APA Secretary to report to the membership the deliberations and transactions of the APA Board of Trustees. With the invaluable collaboration of the Medical Director, Mrs. Ruth Arnold, and others in the Central Office, I have subdivided this report into several categories.

The first category concerns revision of the Constitution and By-Laws, which is immediately relevant for the information of and action by the membership. The remaining categories are listed in alphabetical order.

In true democratic tradition every member of APA is privileged to attend any session of any component of the Association except for the meetings of the Ethics Committee. However, when such personal appearance is impractical, I am sure your next Secretary, Dr. H. Keith H. Brodie, would welcome correspondence on any topic either for the direct attention of the Board of Trustees or for forwarding to the appropriate component for consideration and action.

Finally, I am deeply appreciative of the confidences you have placed in me and shall do my best to merit them in the future.

The Board (or its Executive Committee acting as its agent) took the following actions.

CONSTITUTION AND BY-LAWS

1. Withdrew its approval (given March 7, 1976) of the proposed Constitution and By-Laws. It took this action on the premise that it was irresponsible to offer the membership a document it considered less than its best effort because the Assembly had voted not to accept or reject any of the proposed Constitution and By-Laws changes in order to give the district branches opportunity for full consideration and because no member of the Board of Trustees was completely satisfied with the proposals. The Board urged reconstitution of the Joint Conference Committee on the Key Conference, with a mandate to proceed to develop a better document (Bd., May '76).

2. Adopted the following proposed charge to the reconstituted Joint Conference Committee on the Key Conference Recommendations as a guide: to review all relevant documents, reports, and critiques dealing with recommendations for changes in the APA Constitution and By-Laws emanating from the Key Conference deliberations and subsequent proposals for constitutional change; to note the legal opinions on the potential impact of the proposals for change; and to proceed as rapidly as practicable to draft a new document that deals with the concerns and criticisms expressed by the Trustees on May 9, 1976, when they rescinded the March 7, 1976, approval of the proposed Constitution and By-Laws (Bd., June '76).

3. Authorized publication of the Constitution and By-Laws in *Psychiatric News* (instead of the *American Journal of Psychiatry*) in the event that any one petition for amending the Constitution and By-Laws is presented on the 1977 ballot (Ex.C., Sept. '76).

4. Ruled unconstitutional a proposed amendment (by petition) to the By-Laws designed to transfer fiscal power for dues and assessments to the Assembly without a concomitant amendment to the APA Constitution (Ex.C., Sept. '76).

5. Accepted the Secretary's report of withdrawal of 51 signatures from petition documents A, B, C, and D to amend the APA Constitution and By-Laws and certification that this withdrawal constitutes invalidation of said petitions; they will not be on the 1977 ballot (Bd., Dec. '76).

6. Noted without formal action that it was the understanding of the Reference Committee that the present organizational structure represents minority groups best and therefore should always be presented by the Joint Conference Committee on the Key Conference Recommendations as one of the models for consideration (Bd., Dec. '76).

7. Accepted formally the Secretary's notice that he had received three documents, entitled A, A1, and B1, which were petitions to amend the APA Constitution and/or By-Laws; that each of the petitions was signed by more than the 50 required APA voting members and was received at least 60 days before the annual meeting; and that the signatures on the petitions had been checked and found to be valid (Bd., April '76).

ASSEMBLY

1. Endorsed the Assembly and Budget Committee recommendations that there be an orientation session for executive secretaries of district branches, to be held in 1977 and peri-

This is an edited version of the report presented by the Secretary to the annual business meeting in Toronto, Ont., Canada, May 3, 1977.

odically thereafter—perhaps every five years (Bd., Dec. '76).

2. Approved the recommendation of the Assembly and the Budget Committee that the revolving fund held by APA for divisional meetings could be put to better use by the areas, and endorsed the ideas of abolishing the divisional meeting fund and of sending funds to or holding them for the areas on request (Bd., Dec. '76).

AWARDS

1. Accepted with thanks the grant from the Foundations' Fund for Research in Psychiatry on or about July 1, 1977, to establish a new prize to be called the Foundations' Fund Prize for Research in Psychiatry, the requirements for which will parallel those for the present Hofheimer Prize, funds for which expire after 1977; the Award Committee will consist of six investigators who will be appointed by the APA President to serve three-year terms with staggered rotation dates. The first award will be made at the Convocation during the 1978 annual meeting (Bd., Dec. '76).

2. Established a new award, "The Founders' Award," which will be given annually to a member of APA who has made outstanding contributions as author, spokesperson, and publicist in the services of the mentally ill and disabled and to the art and science of helping them. A plaque and cash award of $1,000 will be presented at the Convocation at the annual meeting, beginning in 1977; funding will be provided by Lederle Laboratories (Bd., Dec. '76).

CONTINUING MEDICAL EDUCATION

1. Approved the expenditure of $5,000 for printing and mailing the continuing education packet to all APA members in September 1976 to explain the new requirement for continuing education activity; noted that the project would be supported by funds available in the Psychiatric Knowledge and Skills Self-Assessment Program revolving fund (Bd., June '76).

2. Authorized $3,200 for the Office of Education, to be used for funding a newly constituted Steering Committee for PKSAP-IV, funds to come from the Psychiatric Knowledge and Skills Self-Assessment Program revolving fund (Bd., Dec. '76).

3. Approved the recommendation of the Council on Medical Education and Career Development that district branches be encouraged to apply for autonomy as accrediting bodies (Bd., Dec. '76).

4. Approved as working guidelines the Policy Recommendations for CME Activities of the Committee on Continuing Education. The Office of Education, in consultation with the council and the committee, will oversee these activities (Bd., Dec. '76).

5. Approved the policy guidelines of the Committee on Continuing Education as follows: General Members and Fellows in active psychiatric or medical practice, administration, education, and research must comply with the CME requirements as part of APA's continuing thrust toward highest quality patient care; Inactive Members, Distinguished Fellows, and Honorary Members are exempt; and Canadian members and APA members from other nations will be held to the requirement for documentation of 150 hours of CME participation over a three-year period but will be exempt at present from adherence to the strict categorical distinction for these hours. (Legal opinion: Life Fellows and Life Members *not* in active practice of psychiatry are to be exempt from CME requirements; Life Fellows and Life Members so designated in the future be required to meet the CME requirements if they *continue* in active practice.) (Bd., Dec. '76).

6. Approved the recommendation of the Council on Internal Organization that any APA program of audio cassette continuing education for distribution to the membership be subject to controls in the selection of material recorded and review of the product before distribution, according to the Council on Medical Education and Career Development policies for audiovisual and audiotaping programs (Bd., Dec. '76).

COUNCILS, COMMITTEES, TASK FORCES, AND COMMISSIONS

1. Authorized 5-year staggered terms for members of the Commission on Judicial Action (Bd., Apr. '76).

2. Authorized the Board's Membership Committee and the Assembly's Membership Committee to meet and work together, under the auspices of the Assembly (Bd., June '76).

3. Authorized a change in the APA *Operations Manual* to incorporate the provision for emergency action by the Joint Commission on Government Relations that enables the President, the Speaker, and the Medical Director to collectively make policy decisions for action, consistent with APA purposes, subject to subsequent reporting to the Board and the Assembly (Bd., June '76).

4. Approved restructuring three components of the Council on Medical Education and Career Development: termination of the present subcommittee on continuing education; termination of the present subcommittee on education of psychiatrists and reconstitution as the Committee on Continuing Education; termination of the subcommittee on education of physicians and reconstitution as the Committee on Psychiatry and Primary Care Education (Bd., June '76).

5. Authorized the President to appoint an Ad Hoc Committee to Review *Psychiatric News* (Ex.C., Sept. '76).

6. Authorized the Ad Hoc Committee on Interprofessional Services to continue for one more year (Bd., Apr. '76).

7. Renewed three committees for another year: the Ad Hoc Committee To Plan a Conference on Law and Psychiatry, the Ad Hoc Committee on Election Procedures, and the Joint Conference Committee on the Key Conference Recommendations (Bd., May '76).

8. Terminated two ad hoc committees, with thanks: the Ad Hoc Committee on the Key Conference Recommendations and the Ad Hoc Committee To Revise the Constitution (Bd., May '76).

9. Approved establishment of an ad hoc committee under the Council on Medical Education and Career Development, with a charge to make recommendations as to the implementation of the 1976 referenda on psychiatric certification (Bd., May '76).

10. Approved the reappointment of Drs. H. Keith H. Brodie, John Davis, and Rebecca Solomon for second four-year terms on the Editorial Board of the *American Journal of Psychiatry* (Bd., Dec. '76).

11. Approved a recommendation of the Reference Committee that Trustees be assigned to each council in May on an annual basis to provide liaison from the Board to the

council and that Trustees so assigned attend meetings of the council throughout the year (Bd., Dec. '76).

12. Approved changing the name of the Committee on Conventions to the Committee on the Annual Meeting (Bd., Dec. '76).

13. Authorized the Task Force on Asian-American Psychiatrists to seek outside funding from HEW to develop a data information bank regarding the treatment of and programs for Vietnamese and Cambodian refugees with mental health problems (Bd., Dec. '76).

14. Voted against reestablishment of the Commission on History at the termination of its present tenure (May 1977) and requested the Museum and Library Committee of the American Psychiatric Museum Association to assume the functions of the present commission (Bd., Dec. '76).

15. Approved the recommendations of the Committee on Graduate Education that APA make every effort with academic organizations to arrange experiences in general medical training for psychiatric trainees in their first postgraduate year and that APA commend the ABPN for its cooperation and effective response in working toward solutions of difficult problems over the past two years (Bd., Dec. '76).

16. Approved establishment of a new Committee to Review and Coordinate the Functions of the Hospital & Community Psychiatry Service, members to include a representative from the Editorial Board of *Hospital & Community Psychiatry* journal and Institute Program Committee, from the Council on Internal Organization, and at least one other member; authorized continued financial support of these activities and development of mechanisms to assess the value of the components of the service to its members (Bd., Dec. '76).

17. Approved sending letters to the President and the Secretary of State expressing alarm about the persecution of the scientific and professional community in Argentina and urging the State Department to take such action as it can to meliorate the situation (recommended by the Council on Emerging Issues and the Council on International Affairs) (Ex.C., Feb. '77).

18. Took no action on a telegram sent by the participants of the APA's Symposium on Culturally Relevant Training for Asian-American Psychiatrists to Mrs. Rosalynn Carter urging appointment of Asian-American representatives to the President's Commission on Mental Health. The President wrote a personal letter calling attention to the needs of that group (Bd., Apr. '77).

19. Accepted the resignation letter of the Editor of the *American Journal of Psychiatry*, Dr. Francis J. Braceland, and his suggestion that the transfer of the editorship be made slowly and with all due deliberation; agreed with applause with President Gibson's words of appreciation for Dr. Braceland's taking "an excellent journal and making it even more excellent." (President Gibson announced that he would consult with President-Elect Weinberg and appoint a search committee) (Bd., Apr. '77).

20. Authorized the Joint Commission on Public Affairs to develop and refine a proposal that APA conduct public opinion and attitude research (Bd., Dec. '76).

21. Accepted the recommendation of the Joint Commission on Public Affairs that the Board reconsider its request for formation of a national advisory group to help promote an improved image for psychiatry; noted alternative suggestions and the statement that there is a definite need for a panel of advisors to the joint commission and for specialized advice and consultation by the Joint Commission on Government Relations; asked that the commission reconsider the whole issue and come up with suggestions (Bd., Dec. '76).

22. Authorized publication of "Guidelines for Psychiatrists in Their Relationships with Communications Media" in the Official Actions section of the *American Journal of Psychiatry* (134:609–611, 1977) with a goal of encouraging every district branch to have designated members available to the media (Ex.C., Feb. '77).

ELECTIONS AND TERMS OF OFFICE

1. Accepted the report of the Committee of Tellers; no candidate received the required 40% of the votes in three positions, and a run-off election to determine President-Elect, Vice-President, and Area I Trustee will be held (Bd., Apr. '76)

2. Asked the Ad Hoc Committee on Election Procedures to consider the matter of how members should conduct themselves in APA national elections, and suggested that a group or individual presenting any document be identified properly on the election publication and that the source of funding be identified (Bd., May '77).

3. Accepted the Committee of Tellers' report of returns in the May 1976 run-off balloting, certifying that Dr. Jack Weinberg had been elected President-Elect and that Dr. Alan Stone had been elected Vice-President (Bd., June '76).

4. Ruled, after closed discussion regarding the Area I Trustee election and run-off, that the procedures had been correct and according to form, affirming that, as reported by the Committee of Tellers, Dr. Ben Feather was elected Area I Trustee (Bd., June '76).

5. Authorized the President to appoint an ad hoc committee to review the constitutional provision for filling vacancies on the Board of Trustees (due to absence or illness); to discuss the election issues (policies concerning nonseating of an elected Trustee; policies, procedures, and the reasons for recall; issues surrounding seating and nonseating); to develop statements concerning challenges of various aspects of the election procedure and the time when a challenge may be filed; to discuss verification of biographical data on the ballot and who should be responsible for such verification; to investigate the complaint by members of Area I, evaluate it, report to the Trustees, and consider why the challenge had been delayed (Bd., June '76).

6. Elected Dr. Judd Marmor and Dr. Lewis L. Robbins as the two Trustees to serve on the Executive Committee of the Board in 1976–1977 (Bd., June '76).

7. Approved the recommendations of the Ad Hoc Committee on Ballot Biographical Data that the Board of Trustees has the power to fill any vacancy on the Board; suggested the matter of Area Trustee replacement be referred to the Committee on Constitution and By-Laws and to the Joint Conference Committee on Key Conference Recommendations (Ex.C., Sept. '76).

8. Approved the recommendations of the Ad Hoc Committee on Ballot Biographical Data that the Board of Trustees and not the Committee of Tellers declares a winner. If there is a previous complaint, the last time that its validity can be examined is before ratification; and, further, directed the Joint Conference Committee on Key Conference Recommendations and the Committee on Constitution and By-Laws to include provisions and policies in the new Constitution and By-Laws for recall of an elected Trustee and for recall elections (Ex.C., Sept. '76).

9. Approved the recommendations of the Ad Hoc Committee on Ballot Biographical Data regarding challenges of

various aspects of the election procedures and the time limitations thereto, with the request that the material be inserted in the APA *Operations Manual*, in the section on Election Procedures (Ex.C., Sept. '76).

10. Accepted the statement of the Ad Hoc Committee on Ballot Biographical Data with regard to verification of biographical data and concurred that the *Operations Manual* sets forth the procedure for presenting the preelection biographical data equitably but cannot be construed as a certification of accuracy and that the membership shares in the responsibility for correct data (Ex.C., Sept. '76).

11. Approved the stand of the Ad Hoc Committee on Ballot Biographical Data that the Board of Trustees acted properly in seating Dr. Ben Feather as Area I Trustee after the 1976 election and their recommendations that if there is any further disagreement with the Board's action, action can be taken to initiate recall, if there is as yet undisclosed evidence to support it; and asked that a notice be inserted in *Psychiatric News* stating that in future elections any objections to or questions on published biographical data be stated to the Board or Executive Committee as soon as possible (Ex.C., Sept. '76).

12. Accepted the approval by the Ad Hoc Committee on Ballot Biographical Data of the form currently used by staff to collect biographical data as the official form and their recommendation that biographical data be accepted *only* on that form (with a certifying statement that the information is correct at time of submission) (Bd., Dec. '76).

13. Accepted the recommendation of the Ad Hoc Committee on Ballot Biographical Data that, in the event of a runoff election, staff reverify the biographical data, making any necessary changes, and asked that members be urged to make complaints as soon as possible (Bd., Dec. '76).

14. Requested that the Ad Hoc Committee on Election Procedures meet with the Assembly at the annual meeting in May 1977 to try to work out the differences between the position of the Assembly and that of the ad hoc committee and the Board on preferential balloting (Bd., Dec. '76).

15. Approved the recommendation of the Ad Hoc Committee on Election Procedures that two statements be sent to candidates to assure compliance with the guidelines: the first statement to be signed by the candidate, acknowledging awareness of the guidelines and promising to abide by them, and the second statement to be signed at the end of the campaign, affirming that the candidate conducted the campaign within the guidelines (notice of compliance to be published in *Psychiatric News*) (Bd., Dec. '76).

16. Approved the recommendation that any campaign literature on behalf of a candidate must include a statement that the explicit approval of the mailing had been obtained from the candidate and a statement of the specific source of funding, with the further notice that personal attacks on candidates are prohibited (Bd., Dec. '76).

17. Approved the Ad Hoc Committee on Election Procedures' recommendations for penalties for campaign violations from most to least serious: admonishment and reprimand, with publication; disqualification from running for election; refusal to seat an elected officer (or unseating, if the violation is made public later); and referral to the Ethics Committee (Bd., Dec. '76).

18. Approved the Ad Hoc Committee on Election Procedures' recommendation that a standing Committee on Election Issues be appointed to handle all campaign matters; that any member accepting appointment will agree to refrain from running for office and from active support of any candidate during his term; and that complaints regarding alleged violations be mailed to the Secretary of APA, who will route them to the Election Committee for review and investigation, report back to the Board, and, where responsibility for a violation is found, recommend actions from the above penalties; and that the deliberations be recorded and forwarded by report to the Ethics Committee (Bd., Dec. '76).

FISCAL MATTERS

1. Approved the suggestion that APA refund a pro rata portion of dues of deceased members (Bd., June '76).

2. Ratified the emergency action of the President, in consultation with the Medical Director and the Speaker, to fund the second stage of the research project to study New Jersey psychiatrists re role conflict in psychiatry.

3. Suggested that the Budget Committee consider partially funding the Falk Fellowship Residency Program (Bd., Apr. '76), and one year later, on receipt of 1) the annual report of the program, 2) notice of a grant of $27,000 to continue the program this year, and 3) notice that the Falk Fund would continue its commitment only until August 1979, approved asking the Budget Committee to budget for full support of the Falk Fellowship Residency Program after the funding from the Maurice Falk Fund has ceased, with increased phased-in funding (Bd., Apr. '76).

4. Approved changes in APA's staff pension plan at an estimated annual cost of $10,000 and authorized staff to take steps necessary to implement these changes (Bd., Apr '76).

5. Approved the recommendation of the House Committee for construction of a doorway between the APA Central Office and Museum buildings and authorized proceeding with development of an alternative plan at a cost not to exceed $4,000 (Bd., June '76). Construction stopped when the House Committee determined the need for an architectural and engineering study of the present structures ($6,000 approved) and the need for exploring alternative means of meeting APA's housing needs in the Washington, D.C , area ($3,500 approved) (Bd., Dec. '76).

6. Approved the recommended 4.8% cost-of-living increase for APA employees retroactive to October 1, 1976, in accord with the policy of maintaining parity with the federal government (Bd., Dec. '76).

7. Reaffirmed the resolution that any council, board commission, or other component that contemplates increasing its costs must obtain Board approval (Bd., Dec. '76).

8. Authorized the Project Director of the APA Minority Fellowship Program and the Deputy Director for Business Administration to seek further funding to continue the program, either from the National Institute of Mental Health or other outside sources.

9. Approved the division of the $75,000 contingency funds (Ex.C., Feb. '77) made by the Treasurer and the Medical Director, and as instructed by the Budget Committee, as follows: Board of Trustees Contingency Emergency Fund, $60,000; and Medical Director's Emergency Operating Fund, $15,000 (Bd., Apr. '77).

10. Received information that the Treasurer had signed an application to the IRS to change the fiscal year of APA and APMA to a calendar-year basis, and noted that assuming IRS approval, the projection of income over expenses will be adequate to fulfill two mandates of the Board: to set aside an operational reserve on December 31, 1976, and to add to reserves from operations during the current fiscal period (Bd., Dec. '76). On April 1, 1977, received information that both APA and APMA had received authorization from the IRS to

change their fiscal year periods to a calendar-year basis.

11. Authorized expenditure of $8,000 from the Board of Trustees Contingency Fund during 1977 for initiation of a proposed Resource Development Project to identify needed improvements, identify sources of funds, develop promotional materials for the Association and the department or program involved, and present proposals to funding sources (Ex.C., Feb. '77).

INSURANCE

1. Reaffirmed the addition of $3,500 to the Assembly Budget for fiscal year 1976–1977 to support a Pilot Insurance Coding Project approved last year but not used (Bd., June '76).

2. Requested the Deputy Director for Business Administration to outline how a central nucleus might be established at the APA headquarters for insurance and other member benefits, and to draw up a cost analysis and model of operation to present to the Assembly and to the Board of Trustees (Ex.C., June '76).

3. Ratified the President's emergency action to approve the proposed retirement plan, contract, and related documents; authorized the Trustees of the APA Retirement Plan to finalize the arrangements as specified (Ex.C., Sept. '76).

4. Ratified the President's emergency action by authorizing an actuarial study of the Association's Professional Liability Program, at an estimated cost of $10,000 (Ex.C., Sept. '76).

5. Asked the President and the Speaker to appoint a small committee to meet with the APA Committee on Member Insurance during the Fall Committee Meetings and reexamine the entire issue of group insurance for APA members, and asked for a report back to the Board of Trustees (Ex.C., Sept. '76).

6. Approved establishment of tax-deferred annuity plans for eligible APA members and staff (Bd., Dec. '76).

7. Approved obtaining an insurance policy covering Association Professional Liability Risk Exposure for members of the Board, constituent components, and staff, at an estimated cost of $5,000 (Bd., Dec. '76).

8. Approved terminating the present Committee on Member Insurance and Retirement Plans, to be replaced by two components: Committee on Member Professional Liability Insurance (whose members would also constitute the Loss Control Committee and Committee on Member Health, Accident, and Disability Insurance), and the APA Retirement Plan Committee, to be responsible for the new member retirement program with Mutual of New York. The three trustees of APA Retirement Plans will be retained until the trust is dissolved (Bd., Dec. '76).

9. Voted reluctantly to approve the new professional liability insurance rates as reported necessary by the Committee on Member Insurance and Retirement Plans and approved establishment of a policy that a member with two chargeable losses within a three-year period become subject to individual underwriting by the company during the surcharge period, with the provision of appeal to the Loss Control Committee (Bd., Dec. '76).

10. Approved a program feature regarding eligibility for insurance rate deviation for psychiatrists who work full-time (35 hours minimum) in an institution and who want to accept private patients in their off-duty hours (Bd., Apr. '77).

LEGAL MATTERS

1. Referred the issue of *Bartley v. Kremens* to the Commission on Judicial Action for preparation of an amicus curiae brief (Bd., Apr. '76); authorized the Commission on Judicial Action to negotiate with the Mental Health Law Project toward a mutually acceptable position for a joint brief in the case of *Bartley v. Kremens*, regarding voluntary admission of children to psychiatric facilities (Bd., May '76).

2. Authorized increasing the budget expenditure in the *Bartley v. Kremens* case from $10,000 to $15,000 to enable APA to submit its own brief (Bd., June '76).

3. Agreed that APA not sign the Texas District Branch amicus curiae brief (Bd., Apr. '76); authorized the Commission on Judicial Action to find some effective way to collaborate in a limited way as amicus in an appeal of the capital punishment statute in Texas, as outlined by the Commission (Bd., Dec. '76).

4. Approved participation with the Mental Health Law Project in a brief against the required reporting of all patients for whom Schedule II drugs are prescribed, as being a law that might cause undue stigmatization to the patient (*Whalen v. Roe*) (Bd., June '76).

5. Approved the recommendation of the Commission on Judicial Action that the Association not pay for legal briefs written by anyone other than the Commission (Bd., June '76).

6. Ratified the President's emergency action to retain the law firm of Bregman, Abell, Solter, and Kay for a fee of $5,000 to study the matter of the petition by the Association for Advancement of Psychology to the Federal Trade Commission for investigation of the Joint Commission on Accreditation of Hospitals and the American Medical Association in regard to violation of antitrust laws (Ex.C., Sept. '76).

7. Instructed the Budget Committee to consider the allocation in the 1978 budget of $30,000 to the Commission on Judicial Action to be used exclusively to assist district branches to implement judicial actions originating in local areas but having national significance (Bd., Dec. '76).

8. Approved the decision of the Commission on Judicial Action not to participate in the amicus curiae brief of the Mental Health Law Project in the case of *Memel v. Mundy* (Wisconsin) in which allegations are that due process rights of persons appearing for involuntary commitment hearings were violated and that the proper procedures for following the adversary rules were not followed (Bd., Dec. '76).

9. Approved the decision of the Commission on Judicial Action to write a brief to urge the Supreme Court to grant certiorari in the appeal of the Caesar contempt citation, in an effort to direct the attention of the court and the public to the importance of confidentiality (Bd., Dec. '76).

LEGISLATIVE LIAISON

1. Advised the Joint Commission on Government Relations to reword their proposed legislative guidelines to state them positively (Bd., Apr. '76); endorsed the 10 policy recommendations of the Joint Commission on Government Relations as guidelines for legislative representatives and APA to use in legislative and other negotiations, with the proviso that if any one of them is objected to by the Assembly it shall be reconsidered (Bd., June '76); received the Assembly's endorsement of the guidelines (Bd., Dec. '76).

2. Asked the assistance of the Joint Commission on Government Relations in offering APA cooperation in future leg-

islative investigations such as that recently conducted by the Select Committee on Aging (Ex.C., Sept. '76).

3. Authorized the President to write to the Director of the National Insitute of Mental Health requesting an opportunity for APA to review the "Mental Health Legislative Guide" developed by the Mental Health Law Project for NIMH in terms of its impact on the provision of mental health care, in order to express criticisms and make alternative suggestions before its publication (Ex.C., Sept. '76).

4. Ratified the President's emergency action, taken at the request of the Assembly and in collaboration with the Medical Director and the Speaker, to protest the issuance of Executive Order No. 10.1 by the Governor of New York to limit or prohibit psychiatrists in the New York State system from engaging in private practice outside their working hours in state employment (Bd., Dec. '76).

5. Approved publication of *Psychiatry Reports*, a quarterly newsletter for members of Congress (Ex.C., Feb. '77).

6. Ratified the emergency action taken by the APA President on January 10, 1977, on the recommendation of the Council on Mental Health Services with the concurrence of the Medical Director and the Speaker-Elect of the Assembly (acting in the absence of the Speaker), to send the following mailgram to the Secretary of DHEW: "Regarding Public Law 94-484, Title VI—Foreign Medical Graduates. Urge you delay implementation of limitations on foreign medical graduates entering the United States pending further study of impact on delivery of essential services to the mentally disabled. To avoid a serious disruption in service delivery, logic would dictate a gradual phase-in over several years. Furthermore, we urge that the Educational Commission for Foreign Medical Graduates examination be allowed to temporarily substitute for national boards until such time as a suitable examination is prepared and is available overseas. We also urge that instructions be issued allowing a liberal interpretation of waiver provision of the law" (Ex.C., Feb. '77).

7. Asked Mr. Joel Klein, APA Special Counsel, to prepare a procedural format for district branches as general guidelines on how to avoid incurring liability in the course of ethical investigations (Ex.C., Feb. '77).

LIAISON WITH OTHER ORGANIZATIONS

1. Approved the recommendations of the Ad Hoc Committee on the Referenda on Psychiatric Certification for establishment of a new component to facilitate communication between APA and the ABPN (Bd., Dec. '76).

2. Approved submitting three names to the ABPN, with identification given this year by rank order, from which the final selection of the Director will be made (Bd., May '76).

3. Approved a change in the By-Laws of the Inter-American Council of Psychiatric Associations, to allow for greater flexibility in the timing for succession in the post of Secretary-Treasurer; authorized support for the Inter-American Council of Psychiatric Associations for another three years at the rate of $1,200 per year (Bd., May '76).

4. Reaffirmed strongly its conviction that the treatment of psychiatric patients within a hospital setting must be under the supervision of a physician; affirmed that in the interests of total patient care, APA is prepared to take whatever legislative and judicial actions are necessary to implement this (Bd., June '76).

5. Formally requested the Council on Mental Health Services to discuss APA's relationships with other mental health professionals and policy in dealing with them and to formulate concepts and recommendations by which the Association can promote the best interests of the patient (Bd., June '76).

6. After further discussion of the already unwieldly size of the Accreditation Council for Psychiatric Facilities and the belief that only organizations that represent types of services different from the ones currently represented should be admitted, took a stand against further enlargement of the Council (Ex.C., Sept. '76).

7. Approved continuing the current liaison with the National Institute on Alcohol Abuse and Alcoholism; accepted Dr. Auerback's resignation with thanks, and asked the President to appoint a member residing closer to Washington, D.C. (Bd., June '76).

8. Asked the President to delegate a representative to attend the September 27 meeting of the Council on Clinical Classification; asked that the Chairman of the Task Force on Nomenclature and Statistics be invited to the December meeting of the Board for discussion of this issue; and deferred action on cosponsorship until then (Ex.C., Sept. '76).

9. Accepted the invitation to join with the American Academy of Pediatrics, the American College of Obstetricians and Gynecologists, the American College of Physicians, the American College of Surgeons, and the Commission on Professional and Hospital Activities in sponsorship of the Council on Clinical Classifications (Bd., Dec. '76).

10. Approved APA's supporting the statement of the American Medical Association decrying violence on TV (Ex.C., Sept. '76).

11. Deferred action on support for the Council of Medical Specialty Societies (Ex.C., Sept. '76) until December and requested that Dr. Richard Wilbur, Executive Director of the CMSS, be invited to submit a report and be present for the discussion; approved increased dues payment by APA to the CMSS (Bd., Dec. '76).

12. Agreed that the answers to questions asked of APA's prospective nominees to the American Board of Psychiatry and Neurology, Inc., should be regarded as confidential (Ex.C., Sept. '76)

13. Approved a proposal that the American Psychiatric Association become an affiliate member of the Committee on Problems of Drug Dependence (CPDD), in order to gain a voice in matters pertaining to drug abuse, give needed scientific support to a unique evaluation activity (the search for nondependence-producing analgesic drugs), without incurring financial obligation (Bd., Dec. '76).

14. Approved a recommendation of the Committee on Liaison with the American Hospital Association that APA endorse efforts of the JCAH to reorganize itself, develop core standards, and train generalist surveyors (Bd., Dec. '76).

15. Approved appointment by APA's President of a liaison representative to the American Nurses' Association (Bd., Dec. '76).

16. Approved continued liaison with the National Consortium for Child Mental Health Services, with the liaison representative to report to the new Council on Children, Adolescents, and Their Families (Bd., Dec. '76).

17. Received suggestions for coordinating activities to determine how APA or individual members will respond to Federal Trade Commission questions concerning the Joint Commission on Accreditation of Hospitals and other medical organizations, and asked that members keep the Medical Director informed; Mr. Stanley Bregman (APA's attorney in this matter) will comment on the feasibility of designated groups and individuals contacting the FTC directly and pro-

viding that agency with information (Ex.C., Feb. '77).

18. Deferred until April a decision on a recommendation to reappoint the ABPN Director; since two issues were involved (this reappointment and policy for similar future situations), it was deemed a matter for the entire Board of Trustees to decide (Ex.C., Feb. '77).

19. Favored in principle supporting the application of the American Board of Qualification in Electroencephalography (ABQEEG) to the American Board of Psychiatry and Neurology for the establishment of special certification of competency; accepted the recommendation of the Council on Medical Education and Career Development that APA support is made with the proviso that the EEG groups coordinate their activities to eliminate conflicting requests to the ABMS and to ensure that the education and training involved is an addition to basic residency education (Ex.C., Feb. '77).

20. Agreed to lend the name of the American Psychiatric Association as a cosponsor of the 27th Annual Conference on Aging, to be held in Washington, D.C., April 17–20, 1977 (Ex.C., Feb. '77).

21. Approved a contribution of $500 to defray the expense of a newsletter and first seminars for a newly formed Committee for a National Health Policy, which is to serve as a forum for debate and communication around current issues for all sectors of the nation's health care delivery system (Bd., Apr. '77).

22. After hearing a presentation from Dr. John G. Meyer, Quality Assurance Coordinator, CHAMPUS Psychiatric Utilization Review, approved in principle the proposal for a contract between CHAMPUS and the American Psychiatric Association to provide CHAMPUS with a nationwide capability for local/state/regional third-party (psychiatrist) retrospective review of psychiatric services reimbursed or about to be reimbursed by CHAMPUS; authorized the President, the Medical Director, and the Speaker to negotiate a contract (Bd., Apr. '77).

23. Noting that "the quality of his work is outstanding," recommended Dr. Marc Hollender for another 4-year term as APA's nominee to the American Board of Psychiatry and Neurology, Inc. (Bd., Apr. '77).

MEETINGS

1. Received brief progress reports on three APA annual meetings (Toronto, 1977; Atlanta, 1978; San Francisco, 1980); authorized staff to investigate alternate meeting sites for the 1979 meeting if ample facilities are not available in Philadelphia (Bd., Dec. '76).

2. Authorized Meetings Management staff to negotiate for a 1979 city other than New York (Bd., Dec. '76) if the New York Hilton cannot vacate its meeting and exhibit space for the APA annual meeting (Bd., Feb. '77).

3. Approved Chicago as the site of the 1979 APA annual meeting, subject to satisfactory site investigation by Meetings Management staff; and authorized staff to investigate moving either the 1982 or 1983 meeting from the East to the Midwest or Southwest to reestablish the normal rotation pattern for APA meetings (Bd., Apr. '77).

4. Approved recommendations to discontinue the post-opening session reception at the annual meeting and hold a reception following the Convocation; to discontinue organized professional tours as part of the official program; and to change the "Policy and Procedural Manual for Annual Meetings" concerning selection of the Grand Marshal and Marshal for the Convocation, meetings of councils and components held during the annual meeting, and prohibition of sales on the exhibit floor or any official area of the convention (to comply with new IRS regulations) (Bd., Dec. '76).

5. Authorized seeking outside funds to support a steering committee that will plan a Conference on Expressive Arts Therapies, to be held in 1977 (Bd., Dec. '76); heard preliminary plans for a conference to bring together representatives from various expressive arts therapies, with a general goal to identify, explore, and seek solutions to problems that confront in varying degrees the associations and practitioners in these therapies (Bd., Apr. '77).

6. Authorized the Ad Hoc Committee To Plan a Conference on the Chronic Mental Patients to seek funds for a conference (Ex.C., Feb. '77).

MEMBERSHIP MATTERS

1. Recommended to the APA Membership Committee and the Assembly Membership Committee that all noncitizen members and Fellows of the APA be granted a suitable dues differential in recognition of the fact that a part of their dues is spent for matters of concern to U.S. residents only; requested that the two Membership Committees meet together and report their recommendations (Ex.C., Sept. '76).

2. Approved the conjoint Membership Committees' recommendation that dues shall be reduced for noncitizens of the United States who live outside the borders of the United States and left determination of the dollar amount to the Treasurer and appropriate committees or individuals (Bd., Dec. '76).

3. Tabled a motion for a procedural change to establish a waiver of dues for those members who have achieved age 70 and have been dues-paying members for the requisite number of years but have *not* achieved Life Fellow status, and a decision whether they should pay for journals and other services (until the fiscal implications have been explored) (Bd., Dec. '76).

4. Deferred all other policy actions regarding dues status of Life Members and Life Fellows and whether publications should be paid for after dues payment ceases; whether Inactive Members should pay publication costs; whether there should be a new category of Life Associate Member; what the length of time should be for eligibility for Fellow status (until fiscal implications of these proposals are determined) (Bd., Dec. '76).

5. Requested the Committee on Constitution and By-Laws and the Assembly to explore the issue of district branch involvement in legislative matters and whether a member who refuses to pay assessments for such activity can be dropped from the district branch (Ex.C., Sept. '76).

6. Approved the opinion of the Committee on Constitution and By-Laws that: "Even if a substantial portion of the budget of a district branch is spent on legislative matters, the district branch is not necessarily in violation of the APA Constitution. The committee believes that this depends on what the legislative matters are and whether the activities carried on are propaganda or the influencing of legislation. Legislative matters are not necessarily propaganda, lobbying, and/or attempting to influence legislation. However, they may be considered so if they fall outside the objectives of the Association, stated, in part, as follows: to advance the standards of all psychiatric services and facilities; to foster

cooperation of all concerned with medical, psychological, social and legal aspects of mental health and illness; to make psychiatric knowledge available to other practitioners of medicine, to scientists in other fields of knowledge, and to the public. Therefore, if a district branch spends a substantial portion of its budget on legislative matters, this is not necessarily a violation of Article II, Section 2 of the APA Constitution; the question of violation depends on exactly what the 'legislative matters' consist of" (Bd., Dec. '76).

7. After deferring action for several meetings until further information and legal opinion could be obtained regarding one APA member who had paid national dues and part of district branch dues but withheld payment of a district branch assessment because of his objection to lobbying activities, reluctantly dropped the member from APA because he had been dropped by the district branch (Ex.C., Feb. '77). Noting Chapter Eight, Section 7, of the By-Laws and that proper recourse is appeal through the Recorder to the Assembly, ratified the February 19, 1977, dropping action of the Executive Committee: a member dropped by the district branch can no longer remain a member of APA (Bd., Apr. '77).

8. Referred study of the issues revealed by the protest membership case reported above to the Committee on Constitution and By-Laws for its recommendations, to the Membership Committee for information, and to the Joint Conference Committee on Key Conference Recommendations for a mechanism for the future (Bd., Apr. '77).

POSITION STATEMENTS AND RESOLUTIONS

1. Supported the Area V action paper deploring the use of mental hospitals as prisons, and asked that information be published in *Psychiatric News* about the Florida law recently enacted and the APA position that a hospital used to treat the mentally ill should not be used as a facility to house prisoners who are not there to receive treatment (Bd., June '76).

2. Authorized the Medical Director to convey to the Social Security Administration the Board's protest of current Medicare policy in regard to weekend passes, expressing alarm over the loss of this important therapeutic modality (Bd., June '76).

3. Withheld approval for publication of the statement on meditation, until action by the Assembly (Bd., June '76); approved the "Position Statement on Meditation," as recommended by the Council on Research and Development and approved by the Reference Committee and the Assembly (Ex.C., Feb. '77).

4. At the request of the National Association on Veterans Administration Physicians, approved in principle the recommendations of the Committee on Federal Government Health Services to support Articles VII and XI of their National Program, regarding the "denial of the due process afforded other Federal employees" and the "physician-administrative relationship" (Ex.C., Feb. '77).

5. Approved a policy statement of the Ethics Committee: "The Ethics Committee opposes release of any information relative to deliberations on ethics matters other than to national officers and members of the Board of Trustees." (Bd., Dec. '76).

6. Approved in principle the proposed statement on "The Crisis in Psychiatric Manpower: Toward a National Psychiatric Manpower Policy," to be sent to the Council on Medical Education and Career Development for presentation at the next meeting of the National Advisory Mental Health Council (Bd., Dec. '76).

7. Approved restating APA's positions of 1971 and 1972 regarding abuse of psychiatric facilities and procedures, and approved the final form of a new statement for submission to the World Psychiatric Association for action: "Proposal of the American Psychiatric Association to the World Psychiatric Association regarding Abuse of Psychiatric Procedures" (Ex. C., Sept. '76).

8. With concern about the World Psychiatric Association Declaration of Hawaii, voted that no representative of APA be permitted to support the canons of ethics of the World Psychiatric Association as now written and that they be instructed to oppose the draft of the Declaration of Hawaii until modifications have been made in the part dealing with detention aspects and the provisions for exceptions to confidentiality (Bd., Dec. '76); after deferring action to allow time for suggested revisions, accepted the World Psychiatric Association Declaration of Hawaii, in principle, and asked that it be transmitted to the World Psychiatric Association Secretary-General with the recommended changes of the APA Ethics Committee and the Board (Bd., Apr. '77).

10. Noting the need for a similar document for the United States, asked the Ethics Committee to draft a statement in regard to ethical considerations for psychiatrist-patient relationships, taking into account institutional relationships, with special input requested from the California Psychiatric Associations about their experiences (Bd., Apr. '77).

11. Once this statement is adopted in principle and forwarded to the WPA, approved publicizing the full content of the statement approved by this Board, with the notation that it is subject to revision by the WPA (Bd., Apr. '77).

12. Approved publication of the *Manual on Peer Review* as a resource document for the district branches and others; general modifications and local adaptations may be required (Bd., June '76).

13. Authorized publication of the "Position Statement on Support for Psychiatric Research," the "Position Statement on the Right to Adequate Care and Treatment for the Mentally Ill and Mentally Retarded" and the "Position Statement on Rehabilitation," approved by the Assembly (Bd., Dec. '76).

14. Authorized publication of *Intervening in Community Crises: An Introduction for Psychiatrists*, together with the material developed from responses to a questionnaire included in the original report (Bd., Dec. '76).

15. Approved the manuscript, "As the Nation Moves Toward National Health Insurance, What About the Mentally Ill?" for publication (Ex. C., Feb. '77).

16. Authorized publication of the "Position Statement on the Problem-Oriented Record-Keeping System," the policy portions of which had previously been approved by the Assembly and the Board (Bd., Apr. '77).

JULES H. MASSERMAN, M.D.

Report of the Treasurer

GENERAL: This report is prepared from audited figures for the fiscal period that ended December 31,1976. Data presented here also appear in the auditor's annual report.

Attached hereto is a statement of financial condition (table 1) and pie charts (figures 1 and 2) reflecting revenues earned and functional costs for the fiscal period. This report, together with the statement and charts, will provide the membership with the information they need to assess the operation and financial condition of the Association.

CHANGE IN FISCAL YEAR: APA received authorization from the Internal Revenue Service to change its fiscal year from April 1–March 31 to a calendar year, effective January 1, 1977. It was therefore necessary to terminate the 1976–1977 fiscal year on December 31, 1976, leading to a 9-month fiscal period (from April 1, 1976, through December 31, 1976). It should be noted that in figures 1 and 2 the comparison of functional costs for the 9-month period for 1976 and for the 12-month period for fiscal year 1975–1976 is not significantly affected by the dissimilarity of fiscal periods. However, the comparison of functional revenue is distorted since revenue from advertising, subscriptions, and book sales for a 9-month period in 1976 is contrasted with full annual membership dues and annual meeting receipts. In addition, this report does not discuss the relationship between actual income and expenditures and related budget figures, since the 12-month budget projections cannot be reasonably compared with budget execution covering the 9-month fiscal period for 1976.

INCOME AND EXPENDITURES: The change in the APA fiscal year, combined with unanticipated budget economies, resulted in income from operations of $5,067,226, compared to expenses of $4,233,522, for a surplus of $833,704. Of this amount, $579,075 has been placed in a reserve account and will be used to offset a portion of the costs of operations during 1977, 1978, and 1979. These funds should preclude the need for a dues increase during this 3-year period. The remainder of the surplus, $254,629, has been placed in a reserve for future expansion and development.

Operations of the Board of Trustees, the Councils, and the Assembly accounted for 18.6% of the expenditures, about the same as for the prior year. Public Affairs required 4.82% of the expenditures, and administrative costs amounted to 10.09%, each up less than one half of one percent.

Member services accounted for 26.78% of the budget, an increase of 3.5%, due primarily to a stronger emphasis on continuing education and other direct services to members. Publications costs amounted to 39.7% for a decrease of 4.59%. This savings stems primarily from renegotiation of printing contracts and increased grant support for preparation of manuscripts.

EMPLOYEE BENEFITS: APA seeks to maintain salaries approximately commensurate with those paid by the federal government. Therefore, a cost-of-living increase (4.8%) was awarded to our employees in October 1976.

While the employer's contribution toward Social Security taxes remained unchanged, the base amount upon which this tax is calculated increased by 8.5%, resulting in an increased expenditure of approximately $8,000 in this category.

APA's annual contribution to the Employees Retirement Plan was reduced from $116,470 to $76,269 (a savings of $40,201) by changing the method of calculating the payment from a mathematical formula to an actuarial projection by Mutual Life Insurance Company of New York.

TABLE 1
Comparison of Fiscal Years 1976 (9 Months) and 1975–1976 (12 Months)

Item	1976	1975–1976
ASSETS		
Cash	$ 424,057	$ 7,620
Certificates of deposit and savings accounts	320,000	770,000
Accounts receivable	386,312	492,203
Accrued interest receivable	17,041	29,559
Deferred and prepaid expenses	103,501	99,711
Marketable securities		
Stocks and bonds (at quoted market value)	1,024,500	1,023,749
U.S. Treasury bills, etc., short-term investments at cost	1,795,388	594,130
Notes and advances receivable from the American Psychiatric Museum Association, Inc.	1,757,085	1,757,085
Less valuation reserve	(1,757,085)	(1,757,085)
Land and buildings (at nominal value)	1	1
Furniture and equipment (at nominal value)	1	1
Total assets	4,070,801	3,016,974
Deduct		
Cash and marketable securities allocated to restricted funds from general fund	366,260	302,223
Total general fund	$3,704,541	$2,714,751
LIABILITIES		
Accounts payable and accrued expenses	64,689	137,464
Due to district branches	46,052	3,639
Deferred income		
Annual meeting, etc.	122,579	20,563
Advance dues collections	1,787,393	—
Funds collected and held for Trustees of the Benjamin Rush Foundation	684	10
Reserve for stabilization of future dues	—	579,075
Reserve for future expansion and development	1,500,000	1,754,629
Less valuation reserve and marketable securities	(74,584)	(38,357)
Total liabilities	3,446,813	2,457,023
Unrestricted surplus		
At beginning of year	580,917	257,728
Net change during the year ended	(323,189)	—
At end of year	257,728	257,728
Total general fund	3,704,541	2,714,751

This is an edited version of the report presented by the Treasurer to the annual business meeting in Toronto, Ont., Canada, May 3, 1977.

FIGURE 1
Comparison of APA Revenues, in Percents

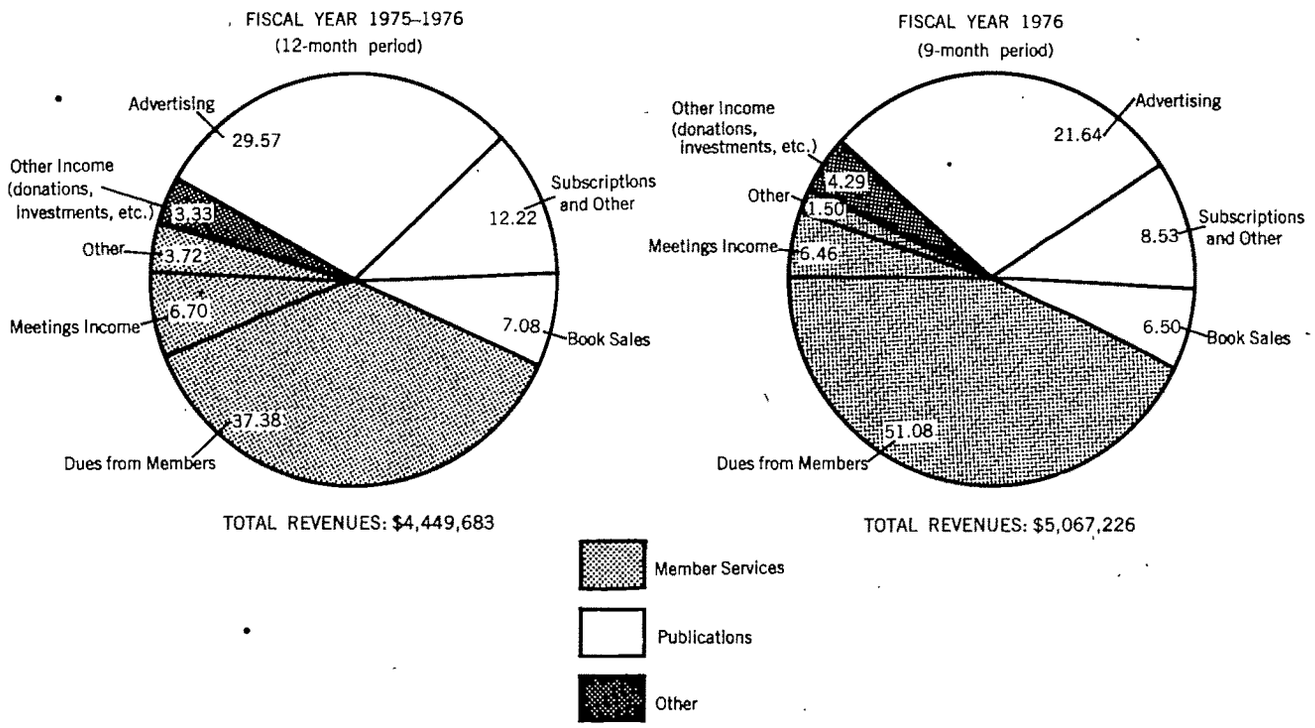
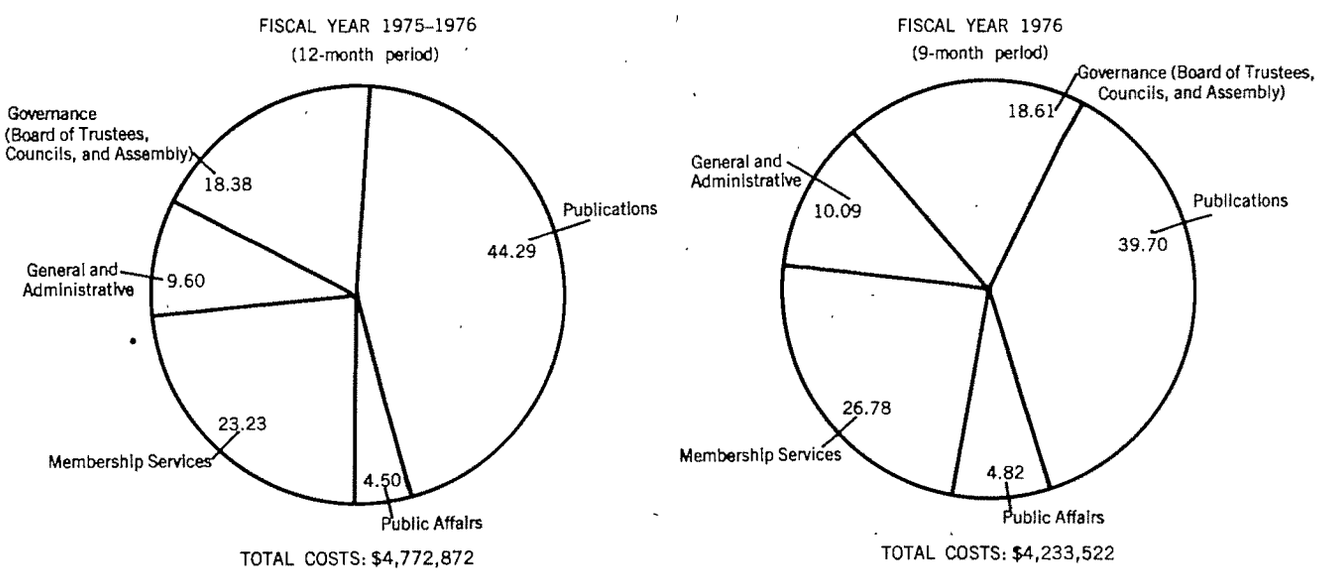


FIGURE 2
Comparison of APA Costs, in Percents



APA was advised by Blue Cross/Blue Shield that the cost of health insurance coverage for staff would increase by 88.7%, or approximately $33,000, per year. Most of this increase related to the major medical portion of the plan. While the Association is maintaining the Blue Cross/Blue Shield basic hospitalization plan, we have selected Mutual Life Insurance Company of New York (MONY) to provide basic surgical coverage and the major medical portion of the plan at an estimated savings to the Association of $20,000 below the proposed Blue Cross/Blue Shield rates. This change also represents considerable savings to a majority of the staff contributing toward their health insurance program.

BUILDING OPERATION AND MAINTENANCE: The trend toward increased costs of building operation and maintenance noted during past years continued during 1976. Costs for the 1976 9-month fiscal period were virtually the same as those

for the preceding 12-month period. Also, the Central Office building currently fails to provide adequate working conditions for staff during warm and cold periods. The situation stems largely from the fact that the heating, cooling, and electrical systems of the Central Office building are reaching the end of their projected life expectancy. This problem, together with other deficiencies related to functional space requirements and structural deterioration, prompted a complete evaluation of the APA buildings by an outside architectural and engineering firm and an analysis of real estate opportunities for APA in the Washington metropolitan area by a real estate consulting group. These studies will provide data necessary for a decision as to whether the Association should undertake a major renovation of its present facilities or purchase new properties in Washington.

INVESTMENTS: The market value of securities in the equities portion of the APA portfolio was $387,058 as of December 23, 1975. The total fund appreciated in market value by $87,139 during 1976 and generated an additional $12,079 in dividends and interest. Total fund earnings for 1976 were $99,218 (25.6%). The fund return was slightly higher than the Standard & Poor's "500" Index and a little lower than the New York Stock Exchange Composite.

The remainder of the portfolio contains intermediate-term and short-term obligations. This fund ranged from a high of $3,166,611 to a low of $1,913,903 during 1976. Intermediate-term bonds accounted for $650,000, earning a 5.42% annual rate of interest. Income from these bonds was $17,275 for the 9-month period. An additional $320,000 invested in savings and loan association certificates earned interest at the rate of 7.5%, for income of $18,000. Late in 1976, $450,000 from maturing government obligations was deposited temporarily in bank savings accounts earning 5% interest because of the unusually low yields available from government obligations at that time. Treasury bills constituted the balance of this fund and varied between $1,850,000 and $300,000 during 1976. Earnings from this portion of the portfolio amounted to approximately $76,500 for the 9-month fiscal period.

PUBLICATIONS: *Psychiatric News* received income of $591,146 contrasted with expenses of $530,868, for a surplus from operations of $60,278. The profit from operations during the 1976 9-month fiscal period represents an increase of almost 100% over 1976 experience, and it is estimated that even greater improvement would have been shown during a 12-month fiscal period. This gain stems primarily from renegotiation of the newspaper's printing contract.

The *American Journal of Psychiatry* produced income of $637,513 and generated expenses of $660,986, for a deficit of $23,473. It should be noted that the Association levies an overhead charge to its three basic publications at the rate established for indirect costs for federal grants and contracts. The overhead rate for the *American Journal of Psychiatry* during 1976 was 28.7% of direct costs. Excluding the APA overhead charge, this publication showed a surplus of $123,926.

Hospital & Community Psychiatry realized income amounting to $200,931 and expenses of $286,290. The resulting deficit was $85,359. After adjusting for the APA overhead charge, the deficit for 1976 was reduced to $21,517, and, on this basis, a surplus is expected for 1977.

The Association maintains approximately 90 publications in inventory. The average monthly sales volume of these publications exceeded that of the previous fiscal year. The Association's publications are produced and marketed at prices that assure a return of the printing and distribution costs. The excess of sales over expenses keeps this activity self-sustaining.

CONCLUSION: I wish to express my gratitude to the membership for entrusting me with the responsibility for the fiscal affairs of APA and to the Assembly of District Branches, the Board of Trustees, the officers, and the Investment Advisory Committee for their support and continuing interest in the financial stability of the Association. Appreciation is also due the Medical Director, the Deputy Director for Business Administration, the Comptroller, and the entire staff for their capable assistance whenever needed during the year.

CHARLES B. WILKINSON, M.D.

Report of the Medical Director

FOR THE PAST several years, I have attempted in my report to the membership to cover the broad issues with which staff has been concerned in implementing Association policies. Although recommendations for increased emphasis and personnel in certain areas have been followed, we have all been concerned with saving our members important dollars and establishing careful methods of accountability.

One of our major efforts last year was an attempt to improve communications with members. This year, I would like to highlight recent changes in the structure and function of the Central Office that we hope will offer members a wider range of services, improve communication, and enable us to deal with the many issues that affect the profession. These changes are a result of recommendations made by the Assembly and authorized by the Board. They do indeed strengthen our capacity to deal with the current challenges to the field.

For historical purposes, I have asked each department and division head, and each Deputy Medical Director, to prepare a detailed report on activities, which is available to any member.

SPECIAL COUNSELS AND DIRECTOR OF GOVERNMENT RELATIONS

On January 1, 1977, Mr. Joel Klein of the law firm of Rogovin, Stern & Huge was appointed APA Special Counsel. He works primarily with the Commission on Judicial Action, but has several additional responsibilities. He has been extremely helpful with advice on the effect of the new IRS regulations (in connection with the Tax Reform Act of 1976) and on the annual meeting in Toronto; he has also worked closely with Mr. Stanley Bregman and Dr. Carolyn B. Robinowitz on the Federal Trade Commission's investigations of medical specialties and the Joint Commission on Accreditation of Hospitals.

In addition, Mr. Klein testified on behalf of the Louisiana Psychiatric Association in Baton Rouge with respect to proposed amendments to the state civil commitment statute. The president of the Louisiana Psychiatric Association, Dr. Robert Newman, indicated that Mr. Klein's assistance "has been very helpful in . . . putting our medical thinking into legal form. His testimony before the legislative subcommittee in Baton Rouge . . . was quite persuasive. He made an excellent case for providing access to treatment for sick people while preserving their civil rights . . . The entire Association is aware of and appreciative of . . . Joel's assistance." Mr. Klein has worked with several other district branches on a number of judicial actions and state concerns.

Joel Klein enhances the ability of the Association and its Central Office to provide legal assistance to members and staff. We are pleased with this new activity and hope that it will become more visible to our membership.

On May 1, 1977, Mr. Jay B. Cutler joined the staff as Special Counsel and Director of Government Relations. Mr. Cutler had served as Minority Counsel and Staff Director for the U.S. Senate Labor and Public Welfare Committee, reporting directly to Senator Jacob K. Javits. He was in charge of organizing and coordinating legislative activities on behalf of the minority (Republican) members of the committee regarding education, labor, health, and public welfare. Prior to this, he was minority counsel for the U.S. Senate Health Subcommittee and Alcoholism and Narcotics Subcommittee. Mr. Cutler has worked on Capitol Hill since 1968.

By adding a staff member possessing the experience and the national stature of Mr. Cutler, the Association is making a significant commitment to be more continuously active in all areas of government affairs. At the annual meeting Mr. Cutler met and talked with the various constituencies within APA to acquaint himself more fully with our membership.

I am particularly grateful to the search committee who aided in the process that led to Mr. Cutler's hiring: Dr. Stanley Yolles, chairperson, Drs. Robert Campbell, Melvin Lipsett, and Johnnie Gallemore, and to the Washington Psychiatric Society members, Dr. George McMahon, president, and Dr. John J. McGrath, chairperson of the Washington Psychiatric Political Action Committee, who aided in interviewing the many candidates for the position.

APA's government relations activities were formalized in 1970 when Mr. Caesar Giolito was added to the staff of the Division on Public Affairs. A separate division was created in 1974 as the Association became more active in legislative affairs. The annual Institutes on Government Operations began in 1972 to provide assistance to the district branches in their legislative efforts; the Sixth Institute held in March this year was attended by 135 legislative representatives and district branch representatives. A highlight of this Institute was a "first" briefing at the White House.

The creation of the Joint Commission on Government Relations allowed a two-way communication mechanism—between the Board and the Assembly—with continuity and guidelines as to the Association's position on various items of legislation. In addition, this commission has formed a Legislative Review Committee to review pertinent federal legislation and interpret legislation vis-à-vis APA policy and the 10 legislative guidelines approved by the Trustees and the Assembly.

In the coming year we plan to become more involved in the state legislative field. The Division of Government Relations already publishes *State Legislative Update*, reporting current developments in various states, and we anticipate the capacity to be of greater assistance in the state legislative process.

An additional publication (a joint effort of the Joint Commission on Government Relations and the Joint Commission on Public Affairs), *Psychiatry Reports*, is a special newsletter aimed at members of Congress that focuses on psychiatric issues and legislation.

This is an edited version of the report presented by the Medical Director to the annual business meeting in Toronto, Ont., Canada, May 3, 1977.

LEGAL AND LEGISLATIVE REPRESENTATIVE

Beginning in 1976 the Association entered into a limited agreement with Mr. Stanley I. Bregman of the law firm of Bregman, Abel, Solter and Kay to participate in such activities (on a part-time basis) as the Institute on Government Operations and the meetings of the Joint Commission on Government Relations. In addition Mr. Bregman assists with selected legislative issues and passes on information of interest to APA. He administratively relates to Dr. D.W. Hammersly. Mr. Bregman serves as consultant to the newly formed Legislative Review Committee (within the framework of the Joint Commission on Government Relations).

In late 1976, Mr. Bregman called our attention to the fact that the Association for the Advancement of Psychology had filed a memorandum with the FTC alleging violation of the antitrust laws by the Joint Commission on Accreditation of Hospitals and/or the AMA. Mr. Bregman assisted in preparing APA's response to this memorandum and continues to follow this case. In addition he has helped various APA members to provide information on an individual basis to the FTC.

PUBLIC AFFAIRS

In the fall of 1976 the Association expanded its Division of Public Affairs to include Mr. Ronald McMillen as Assistant Director, working with Mr. Robert L. Robinson, Director. In addition, a new Joint Commission on Public Affairs was established with Dr. Shervert Frazier as chairperson. The new commission and the availability of Mr. McMillen have increased activities aimed at achieving the Association's commitment to full and open communication with the public. The division prepared "Guidelines for Psychiatrists Working with the Communications Media," which should be helpful during a period when many of our members are being called to comment in the press, on radio, or on television.

The division also worked closely with the APA Committee on Financing Mental Health Care in the publication of "As The Nation Moves Toward National Health Insurance, What About the Mentally Ill?"

The Joint Commission on Public Affairs has been actively planning a study of public attitudes toward psychiatry, which should begin during 1977–1978.

In general, our relations with the media have been improving during this past year. Significant numbers of antipsychiatric viewpoints, however, continue to be presented in the major media, and we need to be quite active in anticipating as well as responding to these criticisms. The staff will be working closely with the Joint Commission on Public Affairs to advocate a fair presentation of psychiatry.

PEER REVIEW—CONSULTANT PROGRAM

To facilitate the activities of district branches in peer review programs, APA hired Dr. Richard Dorsey as a consultant to visit peer review committees, district branches, general members, and insurers. In 1976 Dr. Dorsey visited 32 district branches to explain peer review and APA's policy of seeking financing and review within the same framework as the rest of medicine and parity for psychiatry with other medical specialties.

With the Association's current emphasis on peer review, insurance, and government relations, the Field Consultant program probably will be continued for an additional year. Dr. Dorsey has provided leadership in making it possible for psychiatric peer review to develop rapidly over the next few years; in this regard psychiatry is in the forefront of medical specialties.

This has been a successful "how to do it" project, done at minimum cost, that has provided valuable information to our members and may be a model for similar activities.

EDUCATION

Under the direction of Dr. Carolyn B. Robinowitz, Deputy Medical Director, major changes have taken place in the structure and function of the Office of Education since September 1976. This office now has responsibility for all educational efforts within APA—undergraduate, graduate, and continuing education. A full-time staff has helped to achieve coordinating and leadership functions in continuing education not previously possible. Some 200 programs for continuing medical education were approved and cosponsored by APA through the Office of Education.

The Board and Assembly also approved the guidelines for continuing medical education. In 1974 the membership voted to approve an amendment to the By-Laws requiring participation in continuing education as a requisite for maintaining APA membership in good standing. The program began operation in July 1976; July 1979 is the final date for first reporting.

Dr. Robinowitz will continue her meetings with district branch committees on education and continuing education. In the past 8 months, she has visited Louisiana, Northern California, Washington, Georgia, Connecticut, and Manhattan/Bronx; she attended the Area II divisional meeting where she met with the committee chairpersons from New York State.

The Office of Education has also worked to establish liaison with other psychiatric education and service organizations. Dr. Robinowitz, on behalf of APA, has acted to convene various groups to deal with such emerging problems as the regulations for P.L. 94-484 (Health Manpower), the NIMH budget, the NIMH Forward Plan, etc. The Association has moved from a reactive stance to a more proactive one.

In September 1977 Suzanne Quick reported for duty as the Assistant Director of the Office of Education, and it is anticipated that she will assume major responsibility for the self-assessment test and preparation of evaluation and assessment studies concerning the continuing education activities.

MEMBER BENEFITS

During the Assembly meeting in October, we proposed an Office of Member Benefits. In addition to handling staff responsibility for the five major insurance benefit plans offered to members (professional liability, major medical, disability income and professional overhead expense, family group term life insurance, pension plans), this office will be concerned with continuously reviewing existing insurance programs in light of the needs of APA members; handling all member inquiries and complaints concerning these insurance programs, and coordinating member benefit programs with district branches.

We hope to make this office a focal point for inquiries from

members concerning perquisites and benefits of membership in the Association. We have hired a Director of the Office of Member Benefits, Mr. Thomas J. Yeakle, who began work just before the annual meeting. Mr. Yeakle was formerly with Mutual of New York in various positions, including Mass Marketing Manager and Regional Director of Group and Pension Sales. He has experience in the design, development, sale, implementation, and service of pensions and profit sharing, annuity programs, life insurance programs, major medical insurance, etc. He is familiar with professional liability insurance programs as well. We believe that Mr. Yeakle will be a problem solver who can communicate effectively with our members and improve the Central Office's ability to deal with the membership vis-à-vis member benefits.

There is a keen awareness of the members' concerns over the malpractice crisis, and we are pleased with the leadership of Dr. Chester Trent and the various insurance committees. The Committee on Member Professional Liability Insurance has been exploring the possibility of APA's establishing or purchasing its own professional liability insurance company and will offer its recommendations at a later date.

For the first time, the liability insurance carrier appears to want to negotiate, and the company is considering a plan that will allow partial payment on a malpractice policy for those who work in institutions (which have malpractice insurance on employees) and have some private outside practice.

ASSEMBLY—BOARD OF TRUSTEES

Staff has attempted to facilitate problem-solving in the complex governance of APA with the creation of a position in the Central Office of Coordinator, Assembly and Board of Trustees. Working out of the Medical Director's Office and under the supervision of a Deputy Medical Director, this person will coordinate the secretarial and clerical activities of the Assembly of District Branch Office and the Board of Trustees and will implement policy approved by the Assembly and the Board. We believe that this will facilitate deliberation on action papers, including coordination of consideration by multiple bodies within the Association.

ANNUAL MEETING

As the programming and staffing responsibilities become more complex for the annual meeting (continuing education courses, Category 1 CME credit for the program, etc.), a need arose for staff coordination and the creation of a Staff Committee on the Annual Meeting, ably chaired by Dr. Henry H. Work. With increased communication among staff members who have responsibility for different aspects of the program and social and management aspects of the meeting, we plan to integrate the entire operation from the staff point of view.

We have enjoyed working with Dr. Peter Martin in his role as chairperson of the Program Committee for the past several years and commend him for the excellent innovations that he has brought to the annual meeting program. Dr. Allan Beigel is now the Program Committee chairperson, and the staff looks forward to continued cooperation with the Program and Annual Meeting Committees.

BUSINESS ADMINISTRATION

The second year of operation for this office has now been completed. During the first year of operation, there was a reorganization of the business activities, a balanced budget was presented and approved, and the APA investment portfolio was brought under tighter control. Emphasis during the second year centered on the implementation, expansion, and improvement of programs, development of a personnel classification system, replenishment of reserves to offset deficits from prior years, the establishment of an operating reserve to preclude the need for a dues increase, and a survey of the APA buildings in terms of the feasibility of renovating the existing properties or purchasing other property.

I have been extremely pleased with the excellent leadership of Dr. John Visher as chairperson of the Budget Committee and the increased cooperation among the staff, the Budget Committee, and the Treasurer, Dr. Charles Wilkinson, with the goal of greater fiscal soundness for the Association.

The Investment Advisory Committee, composed of eminent financiers in Washington, D.C. (Dr. Frederick Amling, chairperson, Mr. F. Elwood Davis, and Mr. Hilleary F. Hoskinson) completed its initial review and recommendations on our investments, and the Budget Committee reports that it is very favorably impressed with the present status of our investments and the professional management and advice the Association is now receiving.

This year also marked IRS approval for a change in the APA fiscal year, from April 1–March 31 to a calendar year. Formal approval, received in March 1977, made it possible to initiate the new fiscal year on a retroactive basis to January 1, 1977.

I personally wish to commend Dr. Jack White for his initiative in many areas of the administration of the Association's finances, and the excellent supervision provided to the various departments under his aegis. Our fiscal well-being and the administration thereof have improved immeasurably under his skillful leadership.

CHILD PSYCHIATRY, MINORITY AFFAIRS, AND WOMEN IN PSYCHIATRY

The responsibilities of Dr. Jeanne Spurlock and her staff have increased considerably as a result of the establishment of the Council on Children, Adolescents, and Their Families. This office also provides staff support to the Council on National Affairs.

Work continues on the research project funded by the Falk Medical Fund. "Identification of Strengths in Minority Group Families with a Disturbed/Disturbing Child," a book-length discussion including a literature review and an evaluation of case studies, is near completion.

Additional funding will be required to continue the APA/NIMH Fellowship Program. To date, 38 Fellowships have been awarded and this program has provided an important experience for minority psychiatrists. Before the 1976 Fall Committee Meetings, a research symposium was held that provided a great deal of useful information about research in minority problems.

PUBLICATIONS

The successful scientific journal, the *American Journal of*

Psychiatry, continues its excellent quality, largely because of the efforts of its Editor for the past 12 years, Dr. Francis J. Braceland, and its highly competent staff.

Dr. Braceland asked Dr. Robert W. Gibson to appoint a search committee for a new editor of the *Journal*. The transfer will be slow and with all due deliberation; Dr. Braceland has indicated that he hopes a new editor will be on board by July 1, 1978.

The Association salutes Dr. Braceland for bringing the *American Journal of Psychiatry* into the vanguard of psychiatric journals all over the world. The *Journal* is in excellent shape, and Dr. Braceland deserves the credit for a brilliant period of stewardship.

In the fall, the Council on Internal Organization met with staff and recommended that a committee be established to review and coordinate the functions of the Hospital & Community Psychiatry Service, Institute, and journal.

Largely under the influence of the Editor, Dr. Donald W. Hammersley, and the Managing Editor, Ms. Teddye Clayton, the journal *Hospital & Community Psychiatry* has brought income and expenses into closer alignment; this move toward fiscal accountability is to be commended. The editorial content continues to provide practical, relevant information for psychiatrists and other mental health professionals working in institutional and agency settings. The July 1976 Bicentennial issue of *Hospital & Community Psychiatry*, a remarkable historical account of mental health care in America, was promoted in *Psychiatric News* and some 350 additional copies were sold.

The 1977 Hospital & Community Psychiatry Institute will be held in San Francisco in October and the theme, "The Chronic Patient: Rights, Responsibilities and Resources," ties into the major conference being planned on this topic; it will also provide important data as the President's Commission on Mental Health makes its recommendations concerning delivery of mental health services. We hope to work closely with the commission and their staff; several staff components will be involved in the effort to provide information.

In January 1977 *Psychiatric News* celebrated its 11th birthday and continues to be one of the most widely read publications in the field of psychiatry. I join Mr. Robert L. Robinson, Editor, and Mr. Herbert Gant, Managing Editor, and their staff in being pleased with the appointment of an ad hoc committee (Dr. John Nemiah, chairperson, Drs. Zigmond Lebensohn, and John Visher) to review the newspaper. The newspaper staff has had increased interaction with the membership, with special attention to the needs of minority group psychiatrists, residents, and continuing education activities of the Association. The quality of journalism is high as the paper continues to report news of wide interest and concern to our members.

JOINT INFORMATION SERVICE

Mr. Raymond M. Glasscote and his staff are to be commended for their continued productivity. Two important books were published this year—*Old Folks at Homes* and *Sixteen Indices*. In press and expected by the end of 1977 are two timely studies, one on comprehensive community-based mental health services for the elderly here and in Europe, and a second on primary prevention activities in community mental health centers.

VI WORLD CONGRESS OF PSYCHIATRY

The Association was host to this meeting in Hawaii in August 1977 and provided staff and office space. Activities increased as deadlines for submission of final abstracts approached. The final program and the book of abstracts were distributed to all participants at the time of registration.

INSURANCE

Ms. Evelyn Myers, in addition to her duties as Managing Editor of the *American Journal of Psychiatry*, continues to be our "resident" insurance consultant. Ms. Myers spent a great deal of time working with the Committee on Financing Mental Health Care in the production of APA's new pamphlet, "As the Nation Moves Toward National Health Insurance, What About the Mentally Ill?" This is now available from APA and contains the Principles of Psychiatric Coverage Under National Health Insurance, approved by the Trustees and the Assembly.

Since it has become increasingly clear that the staff must have additional resources to collect necessary data to reinforce our case in Congress on utilization and costs of mental health benefits, we plan to hire a consulting health economist to continue the work of the late Dr. Louis Reed. In addition, it is our intent to have the insurance consultant design health/mental health interface studies that take a closer look at the potential cost savings in general health care when psychiatric care is delivered.

COMPREHENSIVE HEALTH PLANNING

Under Dr. Hammersley's administrative aegis, Mr. Irvin Muszynski has served as Administrative Assistant, and for the past year has continued efforts to secure psychiatric input to the national health planning structure established by P.L. 93-641. An orientation packet on this law was prepared for the district branches and is available on request. To date, 42 district branches have designated specific contacts to act in a liaison capacity with the Committee and Office of Comprehensive Health Planning.

Mr. Muszynski has also been designated APA's Liaison Officer for Federal Agency Affairs; his responsibilities will include additional activities with federal agencies responsible for the administration of programs that affect psychiatry and mental health care in general. To date, this office has responded to issues that include proposed revisions by the Social Security Administration concerning disclosures of physicians' names and medical information to third parties, proposed regulations by the Office of Education for handicapped children, etc. This office is also working closely with the APA Office of Education to ensure timely and substantive responses to the various issues presented by regulations to implement P.L. 94-484, Health Professions Education Assistance Act of 1976. Information on this law and its implementation has been circulated to each district branch.

MEMBERSHIP SERVICES AND STUDIES

The combined manpower and membership operations, headed by Dr. Lee Gurel and a very hard-working staff, completed its first full calendar year. Contact with the district branches and with members has improved, including

updating of district branch and Central Office records; there has been increased use by the district branches of the two-card system for enrolling Members-in-Training and advancement of Members-in-Training to General Members.

We anticipate the publication of the *Biographical Directory* in November 1977; questionnaires have been sent from the publisher to all members. Included on this questionnaire was an APA mini-census that will provide this division with important manpower information. APA will not publish its Membership Directory this year because of the appearance of the *Biographical Directory*.

The division also conducted a census of residents for the academic year 1976–1977, essential as the basis for implementing the two-card system noted above and for providing important information to the profession and government planners concerning input into the psychiatric manpower pool.

We are pleased that the membership of the Association continues to grow; we attracted 1,500 new members during 1976. The net growth (after subtracting deaths and resignations) in total membership was 1,096. The total membership as of January 1, 1977, was 23,301 compared with 22, 205 on that date in 1976.

LIAISON ACTIVITIES

We have attempted to improve our liaison functions and activities during this past year. Dr. Richard Wilbur, executive vice-president of the Council of Medical Specialty Societies, attended the meeting of the Board of Trustees in December and indicated that he believed that the CMSS will become increasingly important as APA clearly defines its role in relationship to the medical specialty societies and their individual interests. Currently, APA has 5 voting members on CMSS: Drs. Paul Fink, Carolyn B. Robinowitz, Anne Seiden, Richard Shader, and L. Douglas Lenkoski. During the CMSS meeting in March 1977, Dr. Robinowitz was elected to the CMSS Executive Committee. Dr. Anne Seiden is the official CMSS representative to the Liaison Committee on Graduate Medical Education, and Dr. Richard Shader has been appointed as one of two CMSS representatives to the American Board of Medical Specialties. It is our hope that there will be a tangible and significant return on our investment in CMSS as new activities begin to show progress.

Dr. Nancy Roeske began 1977 as the APA representative to the AMA Interspecialty Advisory Board and is interested in developing a clear way for APA policies to be included in AMA deliberations. A group of APA members who serve on AMA components will meet during the annual meeting to exchange information.

Dr. John Adams serves as alternate APA representative to the AMA Interspecialty Advisory Board and as chairperson of the AMA Section Council on Psychiatry. The Section Council has increased its activities, and during the Assembly meeting Dr. Rogers J. Smith, Section Council member and delegate to the AMA House of Delegates, will report on increased APA activities there. Dr. Smith has also worked closely with the Assembly in studying the AMA Reference Committee structure.

As a member organization of the Accreditation Council for Psychiatric Facilities, APA has taken a more active role in dealing with the problems that have developed within this council because of increased activities, paucity of surveyors, etc. Our representatives to AC/PF, Drs. Francis de Marneffe, George Tarjan, and Stewart Ginsberg, have communicated closely with staff in an attempt to represent more effectively this Association's views to the Joint Commission on Accreditation of Hospitals and to the AC/PF.

The Board of Trustees wishes to further systematize these Association activities as they relate to other organizations in an effort to provide input of APA policy and to improve communications among the various representatives of this Association who work with related medical and other organizations.

CONFERENCES

APA has taken leadership in two important conferences this year. The Conference on Role Conflicts in Psychiatry, long in the planning, was held in cooperation with the Hastings Institute on March 24–26, 1977, at the Seven Springs Conference Center, Mount Kisco, N.Y. The subject was approached under four rubrics: 1) military, prisons, and schools; 2) psychiatric institutions; 3) private practice; and 4) principles and recommendations. There were 27 participants, representing psychiatry, law, sociology, and other behavioral sciences. APA participants included Drs. Jack Weinberg, Alan A. Stone, Claudewell Thomas, Melvin Sabshin, F.C. Redlich, Robert A. Moore, Robert Michels, Judd Marmor, Robert W. Gibson, Alfred Freedman, Jerome Frank, and Mr. Robert L. Robinson. A substantive report will be prepared by the Hastings Institute staff, with Mr. Robinson as consultant.

The Conference on Discharging Professional Responsibilities Through Peer Review Mechanisms was held at APA headquarters, March 11–12, 1977, in the form of four mini-conferences. In the two morning and two afternoon 3-hour sessions there were 8 participants together with staff and faculty people. Participants in the first session comprised APA leaders in peer review development in psychiatry. This was essentially a self-evaluation session of the progress that has been made and further steps to be taken. The second session involved representatives of consumer interests including representatives of retired persons, the Mental Health Association, the Mental Health Law Project, journalism, and the like. The third session was comprised of third-party payers, including representatives of several leading insurance companies, Civil Service, Blue Cross, etc. The fourth session involved experts in quality assurance and peer review technology including the Social and Rehabilitation Service, Defense Department, Assistant Secretary for Health of DHEW, Bureau of Quality Assurance, and others. A substantive report of the conference is being prepared by Mrs. Pat Vosburgh and will be published within the next three months.

Several major conferences are being planned for the remainder of 1977. A Symposium for Science Writers will be held in the late fall, sponsored by the APA Joint Commission on Public Affairs. A preliminary proposal has been submitted at the request of the Science for Citizens program of the National Science Foundation. This symposium will be an important step in our efforts to improve the quality of science reporting on the medical advances in treating the mentally ill.

A Steering Committee To Plan a National Conference on the Expressive Arts Therapies, with Dr. Robert W. Gibson as chairperson, has begun to meet. The general goal of such a conference would be to identify, explore, and seek solutions to problems that confront the associations and practi-

tioners in the expressive arts therapies. It is hoped that such a meeting would provide a forum between the expressive arts therapists and psychiatrists who have special interests and experience in art, dance, and music therapy, and group psychotherapy and psychodrama.

The Ad Hoc Committee To Plan a Conference on the Chronic Mental Patient, chaired by Dr. John Talbott, is planning a conference during January 1978. It is anticipated that the outcome of such a conference would be a policy statement for the Association on the chronic mental patient and recommendations made to the President's Commission on Mental Health.

SCHEDULING OF MEETINGS FOR 1977

The Assembly expressed its desire to have more time between the Fall Committee Meetings and the meeting of the Assembly in order for district branches and representatives and area representatives to have a chance to digest the mass of material that evolves from the meetings of the various components. In an attempt to try a new scheduling system, we have scheduled these meetings further apart in 1977 and will continue in this pattern, presumably for the next few years. The Fall Committee Meetings were scheduled for September 14–17, with the Assembly scheduled to meet October 28–30, 1977. This year there may be some problem for those who are going on trips after the VI World Congress of Psychiatry.

AREA COUNCILS

Various members of the staff have attended area council meetings during the past year, and we are working on plans and programs to allow better coordination with the area councils. We hope that this coordinating function of the staff can continue to improve. Several area councils have requested an increase in their budgets; staff will provide additional information to the Budget Committee.

ORIENTATIONS

Several years ago, an orientation session was planned by the staff for the new officers, council chairpersons, and Trustees in an attempt to explain the functions and duties of the staff in relation to the Assembly, Board of Trustees, councils, and components. A second orientation program was implemented for the district branch presidents-elect. An orientation session for district branch executive secretaries—the first of its kind—was held at the Central Office in June 1977. These sessions have proven to be quite valuable and useful. It is expected that the orientation for the executive secretaries will be conducted every several years.

OFFICERS MEETINGS

Many topics discussed at the Officers Meetings (held every summer since 1972) have now been implemented: involvement of residents in APA (we now have a new category of Provisional Membership, in addition to Members-in-Training, which will allow even earlier membership for residents); orientation sessions for officers, Trustees, council chairpersons, district branch presidents-elect, and now executive secretaries; continuing education, a requirement for continued APA membership; better Assembly-Board relationships; increased fiscal responsibility and greater efforts to manage expenses; a national review of mental health policy as reflected in the President's Commission on Mental Health; establishment and expansion of the Office of Education; and a conference on the chronic patient and on role conflicts, etc.

CLOSING REMARKS

Much has been accomplished during this past year; much more needs to be done to help in implementing recent policy decisions. Next year I hope to report additional information about our expanded activities in government operations, public affairs, education, and member benefits.

We anticipate a lively year as we work with the President's Commission on Mental Health, coordinate and improve our liaison functions, provide staff back-up for the groups considering reorganization of the APA governing structure, host a World Congress, and keep in close touch with our members through visits to district branches and area councils, etc.

I particularly want to mention the good relationships that we have had with the officers and express my personal appreciation to Dr. Robert W. Gibson who, as President, worked closely with staff and made frequent visits to the Central Office during his presidential year. It has indeed been a pleasure to have a President who has enthusiastically supported the staff's efforts to implement policies.

I would also like to offer my special thanks to the Board of Trustees, the Assembly of District Branches, its Executive Committee, the Reference Committee, the area councils, and the officers of the district branches and the Assembly for their help and support to the staff.

MELVIN SABSHIN, M.D.

Report of the Speaker

THE LAST YEAR has been marked by increasing involvement with the national community and a more aggressive stance by the representatives of the American Psychiatric Association. Psychiatrists, as professional practitioners with many missions, have become more immersed in the world around them as well as with each other. Increasingly psychiatrists have been concerned with their own identity and the extension of knowledge within this medical-psychological-social field and its application to the problems of the psychiatrically ill and to the provision of meaningful service. A somewhat angry reaction to the diffuse, ethereal, and misty concept of mental health, the politicization of the plight of the mentally disabled and the multiply handicapped, and institutional control by the naive has grown within the profession. This feeling of professional militancy has permeated Assembly endeavor and expression. In these perilous times, it was not inappropriate that a group of our brethren, the American Academy of Psychiatry and the Law, devoted its annual meeting to the topic of "Psychiatry Under Seige." Psychiatrists have increasingly attempted to speak out on behalf of their patients and themselves; as Abraham Lincoln stated, "To sin by silence when they should protest makes cowards of men."

One does not achieve acceptance and popularity by pusillanimous acquiescence and servility. On the other hand, one does not achieve professional status in a pluralistic society in the absence of sheer power, which we do not have, without communication with others and accountability for our own acts and procedures. The medical scientist is not well suited by virtue of his professionalism to compete in the marketplace of ideas; the scientific aspect of medical practice does not achieve its veracity through the media of public relations, power politics, or judicial declarations. Unfortunately, decision-makers are overwhelmed by the cacophony of self-asserted competence and the misuse of the democratic concept by those who proclaim that all ideas are created equal.

As a very complex, inherently multidisciplinary group, psychiatrists have been forced to recognize that the family of psychiatry houses many siblings and that a commune is not necessarily a community. We cannot dissipate our energies focusing on mythologies when the realities themselves tax our capacities. In this effort to unify without regimentation, all levels of organized psychiatry have worked more closely in this last year. The hydra-headed monster of Central Administration, Board of Trustees, district branches, councils and committees, legislative network, and Assembly may yet turn out to have one head and one body.

The most significant effort in the last year has been the attempt to restructure representative authority within the Association. Members of the Assembly and of the Board have worked laboriously and lengthily to evolve a slimmer and more efficient system of governance. The Assembly in its fall meeting reviewed in extended discussion proposed constitutional changes and cautiously rejected a number of plans while continuing the process of negotiation and review. This hesitance bespeaks well of a maturity that treads delicately between impulsivity and paralysis.

The Assembly has not restricted itself to the production of gaseous vapors. A significant event has been the limited representation made available for members of certain defined minority or underrepresented groups on a trial or limited time basis. Black, Hispanic-speaking, women, American Indian, and Asian-American psychiatrists have been provided with a forum within the Assembly. The Assembly authorized its Executive Committee to structure a five-year program, recognizing that the ultimate goal is participation by all our members in and through the district branches and a truly adequate representational system for psychiatrists whose prime identity will be that of their profession.

Special Assembly task forces have dealt with two major issues of the day. One group has provided a stormy liaison with the *DSM-III* committee. Although disagreements are rampant and voices strident, the prime purpose of continuing review and input at the Assembly and Board levels has been met. An evolving issue is the sense of dissatisfaction with the community mental health center movement and an attempt to appraise its function and its accomplishment, particularly as some have sought to impose this as the solitary model of mental health delivery, a concept psychiatrists increasingly see as inadequate to its purported goals. Psychiatrists are concerned both about the increasing restrictions on private health care and the collapse of state services in many parts of the United States. While the scenario remains to be written, many are working on the script.

The Assembly has increasingly reflected the wish of organized psychiatry to march forward as a medical specialty. Perhaps the Assembly was in the forefront in this regard which is now so clearly and proudly proclaimed by our training institutes and numerous other spokesmen. The Assembly has recognized that psychiatrists will have to work with greater diligence in this medically competitive world to continue to attract the best and the brightest into our profession. We have recognized that our numerous practitioners reflect a plethora of philosophies, cultures, life-styles, races, sexes, and many other categorizations that ordinarily and unfortunately are used to divide mankind, not to unite it.

If we are to be sensitive to the demands of our governments and to the needs of our patients, then indeed we must be exquisitely attuned to those factors which promote psychiatric knowledge and practice. The work of professionalism ethically applied is a noble task. We need not be defensive about proclaiming virtue where it is merited while keeping in mind the words of the Chinese philosopher, Lao-Tse, who said, "There is no greater injury to one's character than practicing virtue with motivation." As psychiatrists we can accept the fact that appropriate motivation is not a vice and that passive acquiescence to the demands of the ill-informed is certainly no virtue.

This is an edited version of the report presented by the Speaker-Elect of the Assembly of District Branches to the annual business meeting in Toronto, Ont., Canada, May 3, 1977.

Accomplishment in medicine or organizational structure is not dramatic. Our medical revolutions in retrospect have generally been the manifestation of tediously obtained, unexciting accretions of expanding knowledge cautiously applied. Perhaps the concept of the cautious revolution can best characterize the progress of the Assembly and the American Psychiatric Association in this last year as we have inched forward through the swirling maelstrom and chaos of the seventies to what we hope is a better tomorrow.

IRWIN N. PERR, M.D., J.D.

Report of the Speaker-Elect

THE ASSEMBLY OF DISTRICT BRANCHES of the American Psychiatric Association moves at an irregular pace and in an irregular pattern. The turbulence of the 1960s precipitated attention to our own governance. We have agonized at length over becoming more democratic and involving all groups in the decision-making process. Despite the appointment of committees and commissions, achieving the objectives agreed upon at the Key Conference has proven elusive. Much, however, has been accomplished. Our President, Speaker, and Medical Director have articulated an extensive list of accomplishments during the past year. Many changes have occurred, especially the implementation of recommendations originating from the Key Conference. We have thus far not presented a proposed revision of our Constitution and By-Laws, despite our extensive efforts. A revision "by petition" is in the works and may produce the hoped-for changes and improvements. A good plan implemented now is better than an excellent plan ready next year.

During the next year I look forward to the following:

1. An orientation session for executive secretaries of district branches. District branch staff can benefit from the expertise of the Central Office staff by working closely with them.

2. A strengthening of our area council efforts and activities. The APA Central Office is studying the feasibility of assisting area councils with such items as agenda, mailing, budget planning and expenditure control, and closer liaison with Central Office activities. As area councils become more active, we must face the issue of uniform funding. Some area councils assess their members as much as $25 per year to finance council activities.

3. A revision and update of the guidelines for district branch activities. A "how-to-do-it" manual can do much to complement our executive secretaries' orientation.

4. The development of some type of reference committee; this is necessary because of the volume of Assembly business. Matters must be considered by smaller groups to prevent our becoming a discussion group or committee of the whole.

Several additional activities can be started during the next year but will probably take more than a year to complete.

1. A closer relationship between the Assembly and the councils can be initiated at the Fall Committee Meetings by scheduling meetings of the various Assembly representatives and the Speaker-Elect, who serves as a member of the Reference Committee.

2. The technical process of a membership survey was demonstrated several years ago when the members were surveyed on approximately 20 issues facing our organization. A nonbinding solicitation of member opinion via total membership response or a sample survey on 20 to 40 brief issues can be done at dues payment or election time and can alert the leadership to the opinions of the members.

3. Last year the Assembly revised the method of establishment of its district branches. Many of our district branches are too small to effectively manage the many problems that face them, and several are too large to effectively represent their many members. Paradoxically, some of our largest district branches have their officers serving in multiple roles. Each district branch needs to reassess itself in terms of the newly established procedure for district branch formation.

I have heard much over the past year about who has the power in APA. Is it the Board of Trustees or the Assembly of District Branches? It is neither. The power of the American Psychiatric Association rests with its members. Power is discretionary authority over decision making. This power is exercised through a system of representation and delegated authority, periodically renewed, i.e., the democratic process. We have a strong, viable organization; we have a large and increasing membership of talented, able, and competent members. Their leaders must make it possible for them to contribute these talents and abilities to the greatest benefit of all of us.

DANIEL A. GRABSKI, M.D.

This is an edited version of the report presented by the Speaker of the Assembly of District Branches to the annual business meeting in Toronto, Ont., Canada, May 3, 1977.

Report of the Committee of Tellers

THE COMMITTEE MET on March 9, 1977, at APA headquarters to certify the results of the 1977 election. In accordance with the timetable for elections approved by the Board of Trustees at its December 1976 meeting, ballots were mailed on February 1, 1977, to 21,386 eligible voting members. Fifty-seven undeliverable ballots were deducted from that number; the final number of eligible voting members was 21,329; 10,475 ballots were returned and included in the final vote tally (49.1% of the eligible voting membership).

The Tellers acted on questionable ballots held by staff pending the Tellers' decision on their inclusion. The Tellers also verified that all candidates for the 1977 election had certified the accuracy of their submitted biographical information and that all candidates had submitted the required statements of compliance with election guidelines, in accordance with guidelines approved by the Board at its December meeting.

The Committee of Tellers certified the following candidates as elected: Jules H. Masserman, M.D., President-Elect; Donald G. Langsley, M.D., Vice-President; H. Keith H. Brodie, M.D., Secretary; Mary Ann Bartusis, M.D., Trustee-at-Large; Abram M. Hostetter, M.D., Area III Trustee; and Robert A. Moore, M.D., Area VI Trustee. Because of the closeness of the races for Trustee-at-Large, Area III Trustee, and Area VI Trustee, the committee called for a hand count of the ballots; the results were the same.

The Committee of Tellers certified that all amendments to the Constitution and By-Laws and the amendment authorization had passed.

The Committee of Tellers' report was presented to the Board of Trustees on April 1, 1977, and the following recommendations were approved: 1) to accept the results of the 1977 election; 2) to include in the *Operations Manual* the statement that official election envelopes will contain only one ballot; and 3) to refer the issue of accepting ballots postmarked with the deadline date of receipt to the Ad Hoc Committee on Election Procedures.

The Board delayed disposing of the ballots until after the annual meeting in Toronto. Election costs to date, excluding costs of keypunching and tallying the ballots, are $9,281.

LOUIS F. RITTELMEYER, M.D.

This is an edited version of the report presented by the Committee of Tellers to the annual business meeting in Toronto, Ont., Canada, May 3, 1977.

Report of the Membership Committee

THE APA MEMBERSHIP COMMITTEE met October 27–28, 1976, to consider policy and procedural issues and to process applications for various changes in membership status. On October 29, 1976, the committee met jointly with the Assembly Membership Committee to discuss and act on policy items referred by the Board of Trustees to both the committees for joint consideration.

Because the Board of Trustees on several recent occasions has expressed concern about the process and validity of the selection of Fellows, considerable discussion was devoted to this matter.

Processing nominations for Fellowship is a difficult and sensitive task. A very narrow line must be maintained between setting standards that are unfair to the nominee because they are too strict and letting standards fall so low that attaining the status of Fellow becomes meaningless. Unfortunately, the care with which the various district branches screen their nominees is quite variable, and the information available to the Membership Committee is frequently less than complete. Although the committee attempts to be fair at all costs, it tends to be lenient when truly in doubt. District branches and members frequently criticize our failure to approve Fellowship for certain members; on the other hand, to my knowledge, we have never received thanks from those marginal members who are approved.

The committee reviewed the entire process carefully, looking for possible improvements. Among alternatives discussed were more specific definition of categories for significant contributions, differential weighting of the categories, and the addition of other categories. While it was recognized that the membership needs a better explanation of the process, major changes were rejected.

The Membership Committee continues to believe that Fellowship should be reserved for outstanding APA members whose contributions have both breadth and depth; therefore, the committee requires significant and continued contributions in at least four of the seven identified categories: certification; activities in APA, district branches, and other medical societies; non-job-related community service, civic, and social activities; clinical and administrative appointments; teaching activities; research activities; and publications. In addition, there is an "other" category in which special awards, honors, activities, etc., can be noted.

The Membership Committee continuously attempts to evaluate each member's application in the perspective of that member's geographic location and opportunities.

It has been stated that Board certification is over-emphasized under the present system. The Membership Committee continues to believe that Board certification and subspecialty degrees and certifications are important mechanisms to demonstrate a member's general competence and excellence. In addition, Board examinations can be taken by any member who would be eligible for consideration for Fellowship. A review of this year's actions showed that Board certification is helpful but not essential in attaining Fellowship

This is an edited version of the report presented by the Membership Committee to the annual business meeting in Toronto, Ont., Canada, May 3, 1977.

TABLE 1
Membership Transactions

Status	1975–1976 (12-Month Period)	1976 (9-Month Period)
New (N=1500)		
Members-in-Training		655
Associate Members		25
General Members		733
Corresponding Fellows	2	1
General Members-at-Large	5	3
Corresponding Members	10	6
Advancements (N=627)		
From Associate Member or Member-in-Training to General Member		625
From Member-in-Training to General Member		2
Resignations and drops (N=291)		
Resignations from APA		121
Drops for nonpayment of APA dues		126
Resignations and drops from a district branch and consequently dropped by APA		44
Life Fellows, Life Members, 50-Year Life Members (N=269)		
Life Fellows, 1977		195
Life Members, 1977		57
50-year Life Fellows and Life Members		17
Transfers in membership status (N=383)		
General Member to Fellow	206	145
Fellow to Corresponding Fellow	1	1
Corresponding Member to Corresponding Fellow	1	1
Member to Corresponding Member	7	10
Member-in Training/ Associate Member to General Member-at-Large	5	6
Inactive (N=76)	36	40
Honorary and Distinguished Fellows (N=5)		
New Honorary		2
Transfer from Corresponding Fellow to Distinguished Fellow		3
Membership Committee deferral actions (N=163)		
Transfer of General Member to Fellow	107	53
New Corresponding Member		2
Request for waiver denied, transfer of General Member to Fellow		1
Deceased (N=182)		182

status. Over 75% of nominees were Board certified, many with multiple certifications; 81% of those with and 47% without certification were approved.

The question of equal weights for all categories has also been raised. At the beginning of each session, the committee adopts a numerical system for judging all applications to establish interrater reliability within the categories. Different committee members then review different sets of nominees. Those members obviously deserving Fellowship or obviously failing to do so are then easily identified. All near-fails or near-passes are then more carefully reviewed by the entire committee until a consensus can be reached. During this process the individual's total contributions are considered. When contributions are exceptional but activities are narrow (e.g., certain researchers), or if publications are a direct result of research activities already given adequate consideration rather than other types of scholarly professional writing, the necessary adjustments are made. More "credit" is given for community activities that are not requirements for maintaining a practice or appointment on a hospital staff, since these latter activities are recognized under different categories.

In summary, the Membership Committee attempts to use good judgment, tact, and sensitivity in reviewing its APA peers. We feel a more mechanistic system would be detrimental to the exercise of these qualities of review. We recommend that the committee continue to have a balanced representation of minorities, both sexes, different types of practice, and various levels of academic interest.

Finally, it must be noted that Fellowship is a national APA honor with national standards. Difficult situations arise wherein a member does yeoman local work but cannot meet the standards of Fellowship. For these individuals we feel that special recognition at the district branch level would be more appropriate than nomination for Fellowship.

The committee recommends more care on the part of the district branches in screening potential nominees, assembling the necessary information, and forwarding only those truly thought to meet Fellowship standards. We feel that, where possible, the nomination should be initiated by the district branch rather than by the member. The variability among district branches makes the establishment of policies and procedures for nomination very difficult. However, progress has been made over the past year, as reflected in the improved percentage of approvals versus submissions.

Table 1 presents a breakdown of the 3,496 membership transactions during 1976, and table 2 shows a numerical comparison of the 1976 membership and the 1977 membership.

TABLE 2
APA Membership

Classification	As of January 1, 1976 (N=22,205)	As of January 1, 1977 (N=23,301)
General Members	12,844	13,548
Fellows	4,439	4,387
Members-in-Training	1,377	1,495
Associate Members	989	844
Life Members	319	422
Life Fellows	1,244	1,535
Corresponding Members	129	156
Corresponding Fellows	207	207
Inactive Members	598	648
Distinguished Fellows	26	26
Honorary Fellows	33	33

JOHN R. ELPERS, M.D.

Report of the Committee on Constitution and By-Laws

THIS ASSOCIATION, under the title, "Association of Medical Superintendents of American Institutions for the Insane," adopted its first constitution in 1844. This basic document has been modified many times to keep pace with the changes in organizational structure required by the growth of the society. Prior to 1950, these changes were simply accomplished by the process of voting by members in attendance at the annual meeting. Amendments adopted in 1950 changed the process to allow for a mail ballot, considerably widening membership participation in the amendment process.

The Joint Conference Committee on the Key Conference has been working on changes in the Constitution and By-Laws for the past year. However, until the process of comment by the Board, the Assembly, and the district branches, the Committee on Constitution and By-Laws will not review the proposed changes. We do expect this to be a major part of our work during the coming year.

In addition to the annual reports, there are also petitions to amend the Constitution and By-Laws, submitted by petitioners in accordance with provisions in the CBL. Because of the extreme complexity of the process, the committee would like to review the procedure for submission of petitions to amend the Constitution and By-Laws:

1. Any voting member may initiate a petition, signed by 50 or more voting members, to amend the Constitution and By-Laws. The sponsors have until 60 days before the opening of the annual meeting to file the signed petition with the Secretary. If this is done and the petition has the required number of valid signatures, the proposal is presented to the membership at the annual meeting. You now have the petitions. No action is to be taken on the proposal. In addition, the proposal must be disseminated in full text to the entire membership, and this will be done by publication in *Psychiatric News*.

2. Any nonsubstantive changes may be made in the petitions, up to the time of printing. Ballots are usually sent to the printer in early January. Nonsubstantive changes might include changes in the wording to make a meaning clearer or to spell out an intent already indicated.

3. Petitions may be withdrawn up to the time of printing. Thus, although documents to amend the Constitution and By-Laws may be presented to the membership during the annual meeting and printed in *Psychiatric News*, they need not appear on the ballot if they are withdrawn by the petitioners.

The Committee of Tellers has already reported that all of the amendments to the Constitution and By-Laws, voted on by the membership in 1977, were approved and certified by the Tellers. There were several very important changes. Article II of the Constitution outlines the Objectives and Stipulations of the Association, and the membership voted to add an advocacy clause encompassing all who may utilize mental health services: "The objectives of the Association are . . . to promote the best interests of patients and those actually or potentially making use of mental health services." In the present climate of consumerism, it appears to be important to have this clause in our Constitution, explicitly spelled out, although this has been one of the prime objectives of the profession for many years.

Article III of the Constitution establishes a new category of Provisional Member, defined further in Chapter One, Section 4, as physicians in their first year of approved psychiatric residency or in approved internships preparatory to entering approved psychiatric residency. The Assembly proposed this new category to the committee, and we are pleased that the Association has opened its doors to first-year residents and to certain interns as we attempt to provide greater involvement of all young people in APA.

Chapter Two, Section I, now provides for denial of membership in a district branch based on criteria of ethical and professional suitability. An appeal mechanism will be available to those who are so denied membership, through the Recorder of the Assembly.

One important matter that the committee did consider was Article II, Section 2, of the Constitution, which states that no substantial part of the activities of the Association shall be the carrying on of propaganda or otherwise attempting to influence legislation. The APA Executive Committee asked the committee to consider the following question: If a substantial portion of a district branch's budget is spent on legislative matters, is the district branch in violation of the APA Constitution? The committee felt that legislative matters are not necessarily propaganda, lobbying, and/or attempting to influence legislation. However, they may be if they fall outside the objectives of the Association. If a district branch spends a substantial portion of its budget on legislative matters, it is not necessarily in violation of Article II, Section 2, of the Constitution as the question depends on exactly what the legislative matters include.

WALTER H. WELLBORN, JR., M.D.

This is an edited version of the report presented by the Committee on Constitution and By-Laws to the annual business meeting in Toronto, Ont., Canada, May 3, 1977.

1976 Annual Report of the American Board of Psychiatry and Neurology, Inc.

OFFICERS AND MEMBERS OF THE BOARD

AT ITS ANNUAL MEETING on October 17, 1976, the Board elected the following officers: Dr. Thomas W. Farmer, President; Dr. Marc H. Hollender, Vice-President; Dr. Chester M. Pierce, Secretary; and Dr. Melvin D. Yahr, Treasurer.

Dr. Richard I. Shader was elected to succeed Dr. Milton Greenblatt, who had completed his second term of four years in December 1976. Dr. John R. Calverley was elected to succeed Dr. David B. Clark, who had completed his second term of four years in December 1976.

The 1977 Directors of the American Board of Psychiatry and Neurology, Inc., are as follows:

Nominated by the American Medical Association: Dr. Robert J. Joynt* (term expires December 1976), Dr. Richard M. Steinhilber (term expires December 1977), Dr. Thomas R. Johns (term expires December 1978), Dr. James N. Sussex (term expires December 1978), and Dr. S. Mouchly Small (term expires December 1979).

Nominated by the American Neurological Association: Dr. Thomas W. Farmer** (term expires December 1977), Dr. Melvin D. Yahr (term expires December 1978), Dr. Patrick F. Bray (term expires December 1979), and Dr. John R. Calverley (term expires December 1980).

Nominated by the American Psychiatric Association: Dr. Marc H. Hollender* (term expires December 1976), Dr. Chester M. Pierce** (term expires December 1978), Dr. Alfred M. Freedman (term expires December 1978), Dr. Donald G. Langsley (term expires December 1979), and Dr. Richard I. Shader (term expires December 1980).

Nominated by the American Academy of Neurology: Dr. Audrey S. Penn (term expires December 1978) and Dr. Dewey K. Ziegler (term expires December 1978).

EXAMINATIONS

Part I

At the Part I (written) examination on April 19, 1976, 1,730 psychiatrists were tested; 1,473 (85.1%) passed, 257 (14.9%) failed. A total of 461 neurologists were examined; 392 (85%) passed, 69 (15%) failed.

For the 1977 Part I examination, 1,819 psychiatrists and 391 neurologists have accepted assignment. (See table 1 for the number of candidates declared eligible and notified for Part I in 1976 and table 2 for a comparison of these figures with other years.)

This is an edited version of the report presented by the American Board of Psychiatry and Neurology, Inc., to the American Psychiatric Association, April 2, 1977.

*These Directors have been elected to a second term.

**These Directors are serving their second term of four years and are not eligible for reappointment.

Part II

Statistics on Part II examinations administered in 1976 and 1977 to date are listed in tables 3–6.

The Board's most recent Part II examination was administered February 8–9, 1977, in Seattle, Wash., to a total of 333 candidates. Statistics on this examination are not yet available.

The Part II examination schedule for the remainder of 1977 included examinations in Washington, D.C., on April 18–19; Kansas City, Mo., on June 6–7; and Philadelphia on October 17–18. The 1977 Child Psychiatry examinations were held in Chicago on March 14–15.

CHILD PSYCHIATRY

Members of the 1977 Committee on Certification in Child Psychiatry are Dr. John F. McDermott, Chairperson; Dr. Jeanne Spurlock, Vice-Chairperson; Dr. Irving Philips, Secretary; Dr. Richard S. Ward; Dr. Joseph M. Green; and Dr. Norman Bernstein.

The Committee on Certification in Child Psychiatry examined 139 candidates, a record number, at its March 14–15, 1977, examinations in Chicago compared to 106 candidates in 1976. The committee continued to offer its "retake option" in the 1977 examination and extended this option to reexaminees as well as those candidates taking the examina-

TABLE 1
Number Declared Eligible and Notified for Part I Written Examination April 19, 1976

Item	Psychiatry	Neurology	Total
Number declared eligible and notified	2,530	583	3,113
Number who declined or withdrew	631	91	722
Number remaining for written examination as of March 19, 1976	1,899	492	2,391

TABLE 2
Five-Year Comparison of Number Declared Eligible and Notified for Part I Examination

Item	1972	1973	1974	1975	1976
Total eligible	1,552	1,999	2,098	3,146	3,113
Total who accepted	1,119	1,381	1,821	2,408	2,391
Total who actually appeared	965	1,205	1,627	2,240	2,191

TABLE 3
Analysis of Performance of Group Who Took Part II Examination in Minneapolis, Minn., June 21–22, 1976

Item	Psychiatry		Neurology		Child Neurology		Total	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Candidates	285	69.2	122	29.6	5	1.2	412	100.0
Results								
Pass	205	71.9	73	59.8	3	60.0	281	68.2
Fail	18	6.3	8	6.6	0		26	6.3
Fail (must reapply)	32	11.2	5	4.1	0		37	9.0
Condition	30	10.6	36	29.5	2	40.0	68	16.5

TABLE 4
Analysis of Performance of United States (N=334) and Foreign (N=78) Medical School Graduates Who Took Part II Examination in Minneapolis, Minn., June 21–22, 1976

Results	United States Graduates		Foreign Graduates	
	Number	Percent	Number	Percent
Pass	235	70.3	46	59.0
Fail	20	6.0	6	7.8
Fail (must reapply)	24	7.2	13	16.6
Condition	55	16.5	13	16.6

TABLE 5
Analysis of Performance of Group Who Took Part II Examination in Chicago, Ill., October 18–19, 1976

Item	Psychiatry		Neurology		Child Neurology		Total	
	Number	Percent	Number	Percent	Number	Percent	Number	Percent
Candidates	731	74.9	213	21.8	32	3.3	976	100.0
Results								
Pass	388	53.1	127	59.6	19	59.3	534	54.7
Fail	144	19.7	22	10.4	2	6.3	168	17.2
Fail (must reapply)	16	2.2	15	7.0	1	3.1	32	3.3
Condition	183	25.0	49	23.0	10	31.3	242	24.8

TABLE 6
Analysis of Performance of United States (N=619) and Foreign (N=357) Medical School Graduates Who Took Part II Examination in Chicago, Ill., October 18–19, 1976

Results	United States Graduates		Foreign Graduates	
	Number	Percent	Number	Percent
Pass	383	61.9	151	42.3
Fail	79	12.8	89	24.9
Fail (must reapply)	22	3.5	10	2.8
Condition	135	21.8	107	30.0

tion for the first time. This option allows candidates who feel they have performed badly in an oral section to repeat one oral section on the day of the examination. This option does not apply to the committee's written examination.

At a two-day retreat on March 28–29, 1977, in Tucson, Ariz., the following agenda items were discussed: 1) reconstruction of the grid for the committee's 1978 written examination; 2) revision of the Program for Special Training and Qualifications (PSTQ) guides; and 3) involvement of child psychiatry training directors in the certification process.

BOARD DECISIONS AND ITEMS OF INTEREST

The following major policy decisions were enacted by the Board in 1976:

1. Effective July 1, 1969, a passing grade on the Part I

examination dated any time since April 1, 1969, will be valid for seven years from the date of passing. All individuals holding such a grade may apply or reapply for the Part II examination.

2. Candidates who do not pay the Part II examination fee for their scheduled examination by the deadline specified will be removed from the roster of candidates for that examination. Such candidates will be required to submit a Part II application and the examination fee in order to be scheduled for a future Part II examination.

Candidates who pay their Part II examination fee but fail to appear at their scheduled examination will forfeit the fee. Such candidates will be required to submit a Part II application and a new examination fee in order to be scheduled for a future Part II examination.

Candidates submitting the Part II application may be required to wait one year, and possibly longer, before the Board will have sufficient examination space to schedule them.

Candidates submitting the Part II application will be billed for the Part II examination at the time they are scheduled. See appendix 1 and appendix 2 for additional policy changes.

At its June 19–20, 1976, business meeting in Minneapolis, Minn., the Board took the following significant actions:

1. Went on record in favor of greater regionalization of Part II examiners. Adopted guidelines which specify that the majority of examiners come from the region in which the examination is being conducted.

2. Decided on a new scoring system for the Part I examination. Discontinued use of the American-Canadian reference group, and substituted a standard deviation approach.

3. Agreed to substitute one videotaped examination for one live patient examination in Psychiatry and Neurology, beginning with the October 1977 Part II examination in Chicago. Under the new examination format, each Psychiatry candidate will take three hours of examination: one hour interviewing a psychiatric patient, one hour viewing a videotaped interview with a psychiatric patient and then being quizzed by an examiner, and one hour interviewing a neurological patient. The Neurology candidates will take a four-hour examination: one hour interviewing a neurological patient, one hour viewing a videotaped interview with a neurological patient and then being quizzed by an examiner, one hour interviewing a psychiatric patient, and one hour interviewing a child neurology patient.

4. Agreed to deny a proposal that ABPN join with the American Board of Physical Medicine and Rehabilitation to establish a joint certification program in EEG.

5. Agreed to explore with interested neurologists the possibility of developing a proposal, under the aegis of ABPN, regarding standards for special competence and training in electromyography (EMG) for the ABMS with requirements acceptable to ABPN.

At its November 18–20, 1976, policy meeting, the Board took the following actions:

1. Reviewed the results of a study done by Dr. Donald Langsley on the Chicago Part II examination (October 18–19, 1976). The study involved nearly 1,000 candidates and compared their performances on the live patient and audiovisual examinations. Between live patient and audiovisual Psychiatry and between live patient and audiovisual Neurology the concurrence was 84% and 86%, respectively. Dr. Langsley also presented evidence that the audiovisual examination was well accepted by Psychiatry and Neurology candidates and examiners.

2. Reaffirmed its earlier decision not to assume responsibility for certification in Administrative Psychiatry.

3. Approved the following resolution: "The ABPN recommends that only specialist site visitors be used to evaluate programs in Psychiatry and Neurology. The present system does not provide the necessary information for proper evaluation of training programs." It was agreed that the above resolution should be sent to the Residency Review Committee and promulgated to the ABMS, the Coordinating Council on Medical Education, and the Board's sponsoring societies.

4. Agreed that Dr. Marc Hollender be granted funds and authorization to invite certain psychiatrists and neurologists to develop questions for a written examination to go with the Part II audiovisual examination.

5. Agreed to revise its Part I information booklet. The new booklet includes far more detailed information on the categories for questions in Psychiatry and Neurology and sample questions from the NBME pool.

At its business meeting on February 6, 1977, in Seattle, Wash., the Board took the following actions:

1. Agreed to revise the material for Part II examiners' orientation.

2. Decided that the Credentials Committee should hold a special meeting to consider credit for the first postgraduate year abroad, the PSTQ plan, credit for psychiatry candidates with two years of training in general psychiatry and one year of training in child psychiatry, D.O. internships, and interpretation of the requirement for four months in a primary care specialty.

3. Agreed that for the 1977–1978 Part II examination cycle, beginning in October 1977, the following examinations would be authorized and added to the format currently being used: one audiovisual examination in Child Neurology for child neurology candidates, and one audiovisual examination in Neurology for psychiatrists to include, if possible, a written examination.

4. Decided to enlarge the Steering Committee on Recertification to include two representatives from each of the Board's constituent organizations—the Steering Committee for Psychiatry, chaired by Dr. S. Mouchly Small, and a Subcommittee for Neurology, cochaired by Dr. Dewey Ziegler and Dr. Jerry Chutkow. The steering committee agreed that the recertification procedure would be devised to test competence in the major specialty. Any tests for competence in the minor specialty would include only material that was relevant and necessary for the optimal clinical care of patients in the major specialty. The Steering Committee has not yet set a date for recertification but plans to do so at its next meeting.

APPENDIX 1
Policy for Retaking of Part II (Oral) Examination*

FOR PSYCHIATRY CANDIDATES

1. A candidate for certification in psychiatry who passes both Psychiatry sections of his/her Part II examination need

*This policy applies to all candidates who have passed the Part I (written) examination since June 1969 or later. Candidates who have previously failed the Part II examination and who do not now have an active Part II application on file must submit a Part II application to the Executive Office of the American Board of Psychiatry and Neurology, Inc., in order to be scheduled for a Part II examination under the new policy. The candidate's most recent pass on the Part I examination will be considered the effective date for the beginning of the seven-year period.

not repeat this part of the examination within seven years following the date of successful completion of the Part I examination. To pass the Psychiatry portion of the Part II examination, the candidate must achieve final grades of 75 or above on both Psychiatry sections of the examination.

2. A psychiatry candidate who fails one or both sections of the Psychiatry examination must take two hours of examination in Psychiatry at each subsequent examination until he/she passes both sections or until the expiration of the seven-year period following successful completion of the Part I examination.

3. Any candidate for certification in psychiatry who passes the Neurology portion of his/her Part II examination need not repeat the Neurology portion of the examination within a period of seven years following the successful completion of the Part I examination.

FOR ADULT NEUROLOGY CANDIDATES

1. A candidate for certification in adult neurology who passes both sections of his/her Adult Neurology portion of the Part II examination need not repeat this part of the examination within a period of seven years following the date of successful completion of the Part I examination. To pass the Adult Neurology portion of the Part II examination, the candidate must achieve final grades of 75 or above on both Adult Neurology sections of the examination.

2. A candidate in adult neurology who fails one or both sections of the Part II Adult Neurology examination must take two hours of Adult Neurology at all subsequent Part II examinations until he/she passes both sections or until the expiration of the seven-year period following successful completion of the Part I examination.

3. A candidate for certification in adult neurology who passes the Psychiatry portion of the Part II examination need not repeat this part of the examination within a seven-year period following successful completion of the Part I examination. The same provision applies to adult neurology candidates who pass the Child Neurology portion of the examination.

FOR NEUROLOGY WITH SPECIAL COMPETENCE IN CHILD NEUROLOGY CANDIDATES

1. A candidate in child neurology who passes both sections of his/her Child Neurology portion of the Part II examination need not repeat this part of the examination within a period of seven years following the date of successful completion of the Part I examination. To pass the Child Neurology portion of the Part II examination the candidate must achieve final grades of 75 or above on both Child Neurology sections of the examination.

2. A candidate in child neurology who fails one or both sections of the Part II Child Neurology examination must take two hours of Child Neurology at all subsequent Part II examinations until he/she passes both sections or until the expiration of the seven-year period following successful completion of the Part I examination.

3. A candidate for certification in Child Neurology who passes the Psychiatry portion of the Part II examination need not repeat this part of the examination within a seven-year period following successful completion of the Part I examination. The same provision applies to Neurology with Special Competence in Child Neurology candidates who pass the Adult Neurology portion of the examination.

APPENDIX 2
Current Regulations for Conditioning or Failing the Part II Examination*

FOR CERTIFICATION IN PSYCHIATRY

2 hours of Clinical Psychiatry (major)
1 hour of Clinical Neurology (minor)
Failure**=failing 1 hour major and 1 hour minor
Condition***=failing 1 or 2 hours major or 1 hour minor

FOR CERTIFICATION IN NEUROLOGY

2 hours Clinical Neurology (major)
1 hour Clinical Psychiatry (minor)
1 hour Child Neurology (minor)
Failure**=failing 1 hour major and 2 hours minor
Condition***=failing 1 or 2 hours major and/or 1 hour minor or 2 hours minor

FOR CERTIFICATION IN NEUROLOGY WITH SPECIAL COMPETENCE IN CHILD NEUROLOGY

2 hours Child Neurology (major)
1 hour Clinical Psychiatry (minor)
1 hour Clinical Neurology (minor)
Failure**=failing 1 hour major and 2 hours minor
Condition***=failing 1 or 2 hours major and/or 1 hour minor or 2 hours minor

FEE SCHEDULE FOR REEXAMINATION

Failure=$190.00
Condition of major=$190.00
Condition of minor=$140.00

*A candidate must pass both hours of his/her major at the same Part II examination. If one hour of the major is conditioned, both hours of the major must be repeated.
**A candidate who fails his/her initial Part II examination must repeat and pass the entire Part II examination.
***A candidate who conditions his/her initial Part II examination must repeat and pass those subjects he/she failed originally.

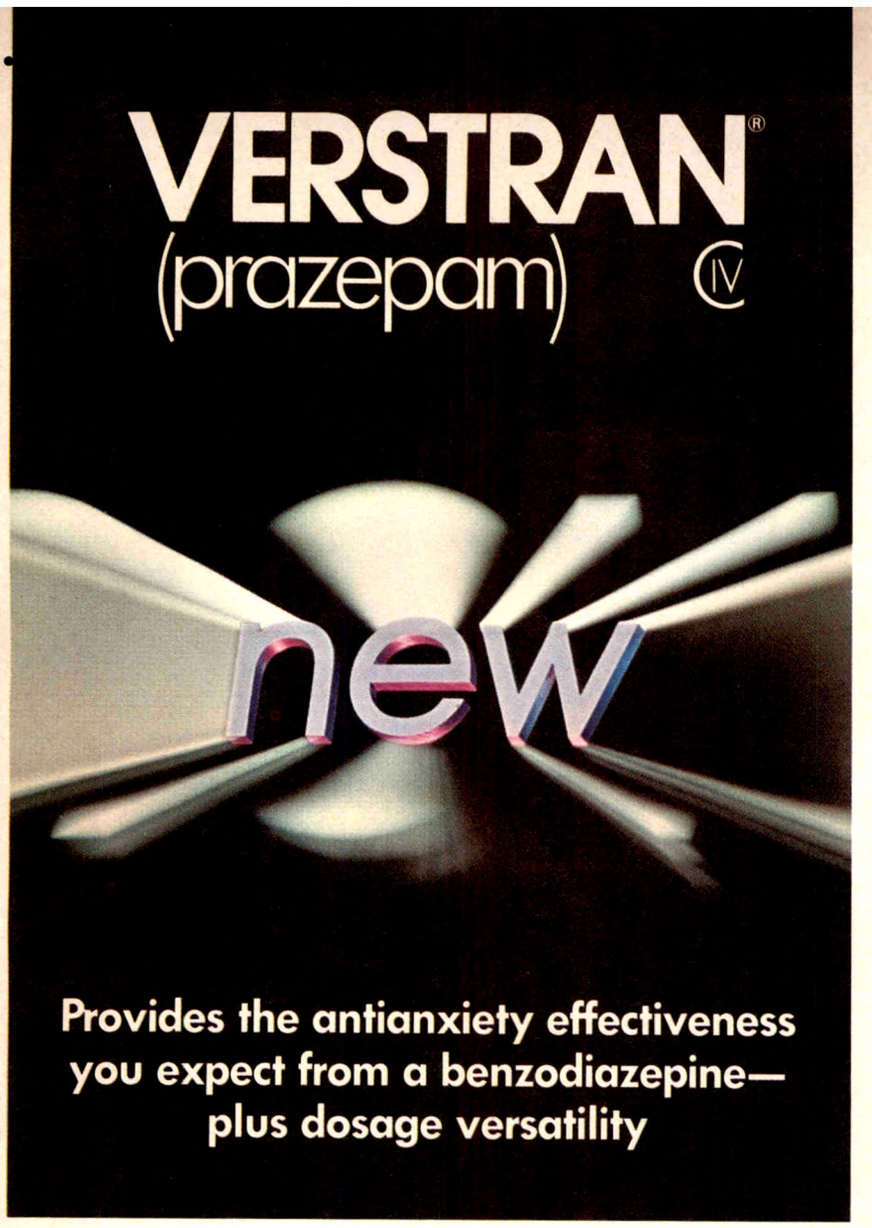
LESTER H. RUDY, M.D.
Executive Director

Warner/Chilcott Introduces



VERSTRAN®
(prazepam) C IV

A new addition for the treatment of anxiety



Provides the antianxiety effectiveness you expect from a benzodiazepine—plus dosage versatility

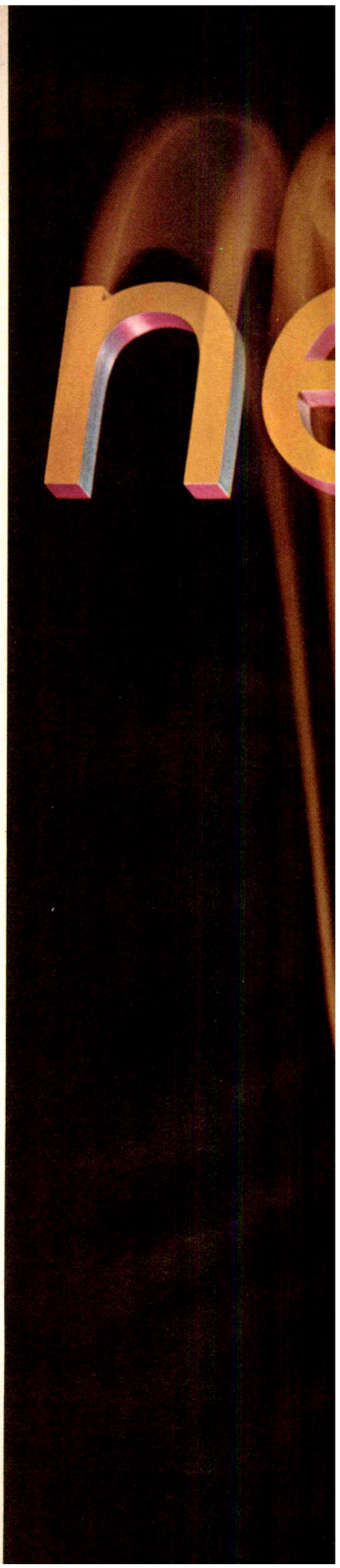
New Verstran can be administered in daily divided doses or once a day at bedtime

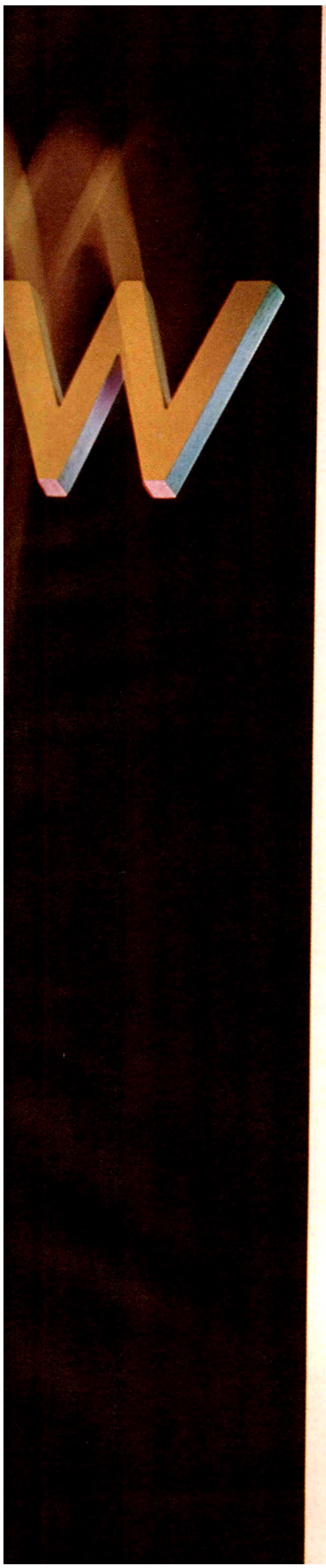
Option 1: A daily divided dose

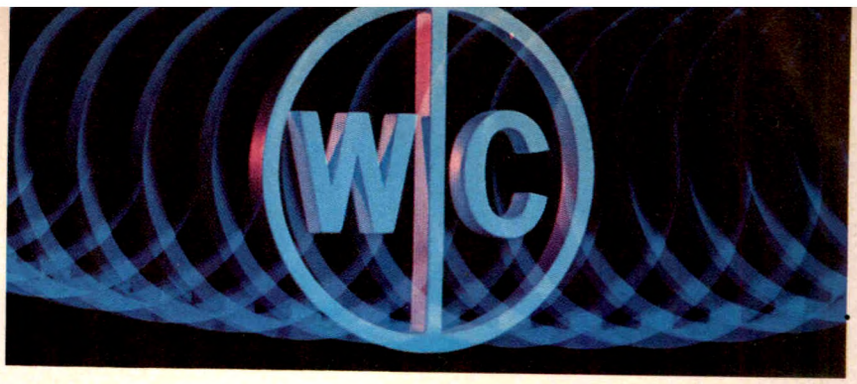
For those anxious patients who need the reassurance of multiple daily doses, the recommended starting dose is 10 mg B.I.D. This dose may be titrated gradually within a range of 20 to 60 mg per day in accordance with response.

Option 2: A once-a-day dose at bedtime

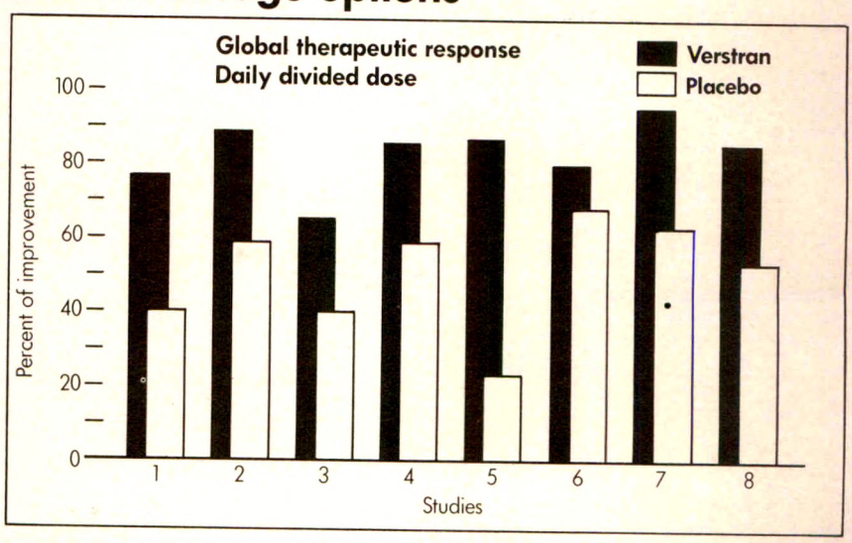
Taken in a single daily dose at bedtime, Verstran helps reduce the symptoms of anxiety—whether psychic or somatic in origin—throughout the following day. The usual starting dose for this regimen is 20 mg (two 10 mg tablets). Optimum dosage usually ranges from 20 to 40 mg daily.





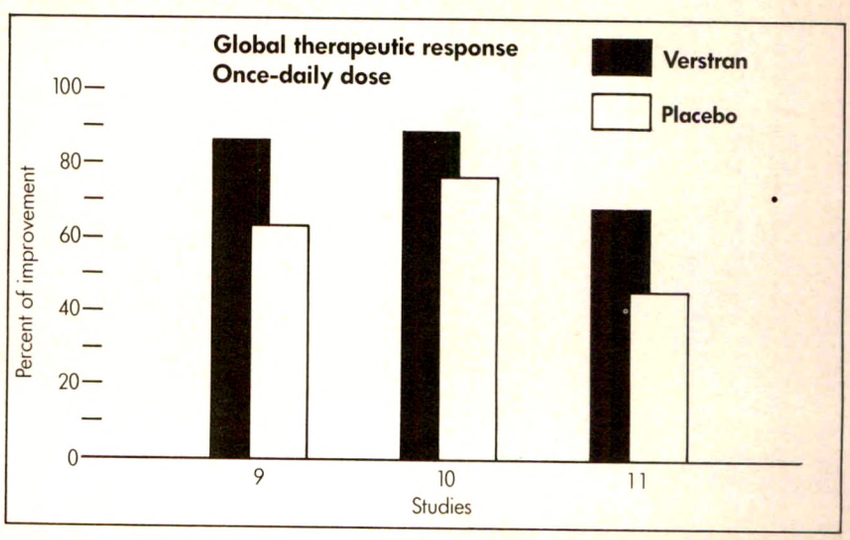


Eleven double-blind studies totaling 560 patients confirm the clinical efficacy of both dosage options*†



Four hundred and four patients suffering nonpsychotic clinical anxiety were given Verstran (228 patients) or placebo (176 patients) in a daily divided dose according to a randomized, double-blind design. Dosage of Verstran was within the recommended limits.

Target symptoms rated include: anxiety, depressive mood, irritability/hostility, tension and insomnia. Global Improvement Ratings were made at week 2, 3, or 4.



One hundred and fifty-six patients suffering nonpsychotic clinical anxiety were given Verstran (70 patients) or placebo (86 patients) once daily at bedtime according to a randomized, double-blind design. Dosage of Verstran was within the recommended limits.

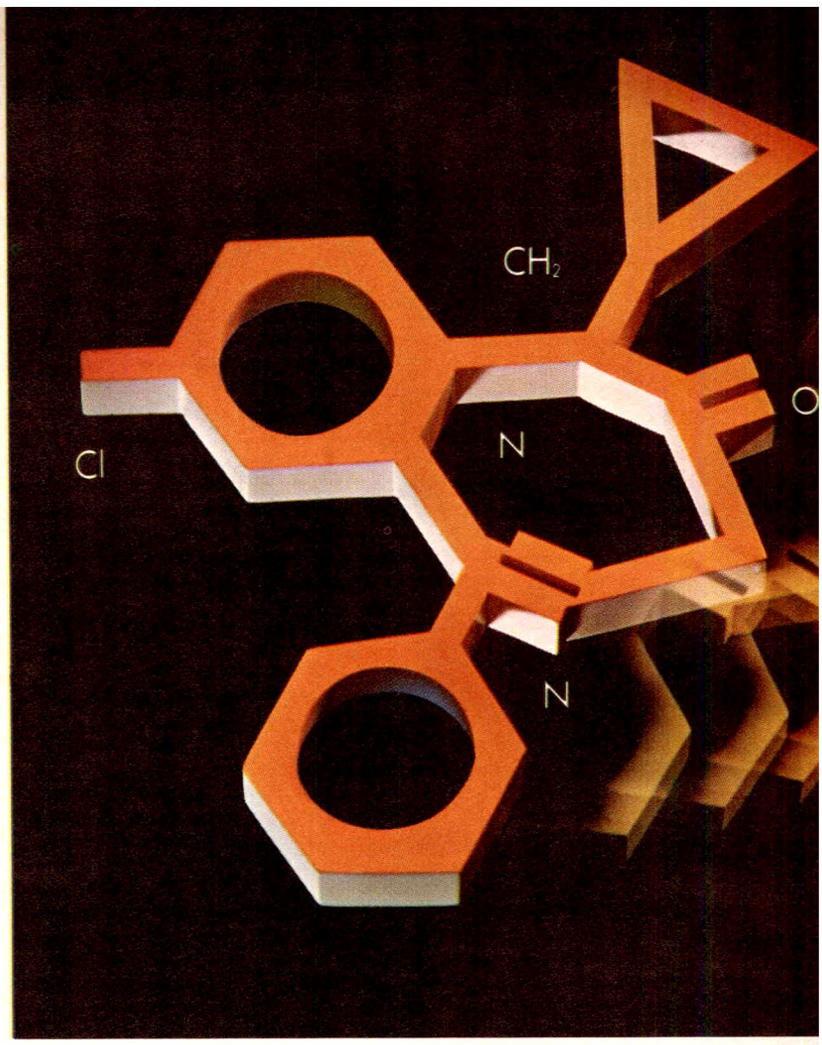
Target symptoms rated include: anxiety, depressive mood, irritability/hostility, tension and insomnia. Global Improvement Ratings were made at week 3 or 4.

*Data on file, Warner/Chilcott Medical Department.

†The number of patients cited represents those evaluated at final observation.

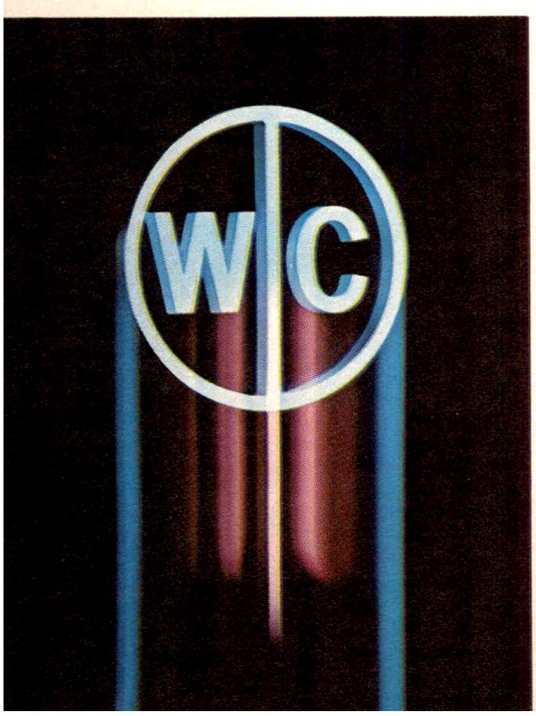
Please see last page of this advertisement for full product information.





New Verstran provides the simplicity of a single 10 mg tablet strength

Verstran is available in one 10 mg tablet strength only. Titration is simplified, and dosage is easy for the patient to remember. Scored tablets facilitate precise titration.



■ **New Verstran.**
Effective for the symptomatic relief of a wide range of anxiety conditions — whether psychic or somatic in origin.

■ **New Verstran.**
The option of fully effective divided daily or single nighttime dose.

■ **New Verstran.**
The simplicity of a single tablet strengt

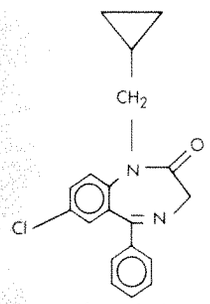
■ **New Verstran.**
Side effects are characteristic of benzodiazepine drugs.*

*Please see last page of this advertisement for warnings, precautions and adverse reactions.

Verstran® (prazepam) C IV

Caution: Federal law prohibits dispensing without prescription.

Description: Verstran (prazepam), a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1,4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows:



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (± 15 s.d.) hours before and 70 (± 10 s.d.) hours after multiple dosing—a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:

Use in Pregnancy and Lactation:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible aberrations of liver function tests have been reported, as have been slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though unlikely, may be controlled with Levophed® (levarterenol bitartrate) or Aramine® (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection, USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

How Supplied: Verstran (prazepam) 10 mg light blue, scored tablets in bottles of 100 (N 0047-0276-51).

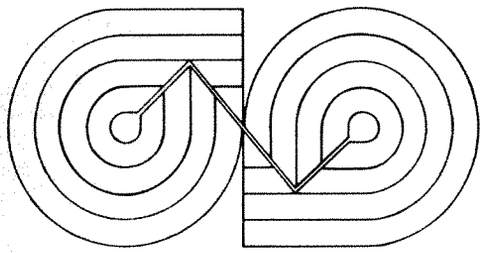
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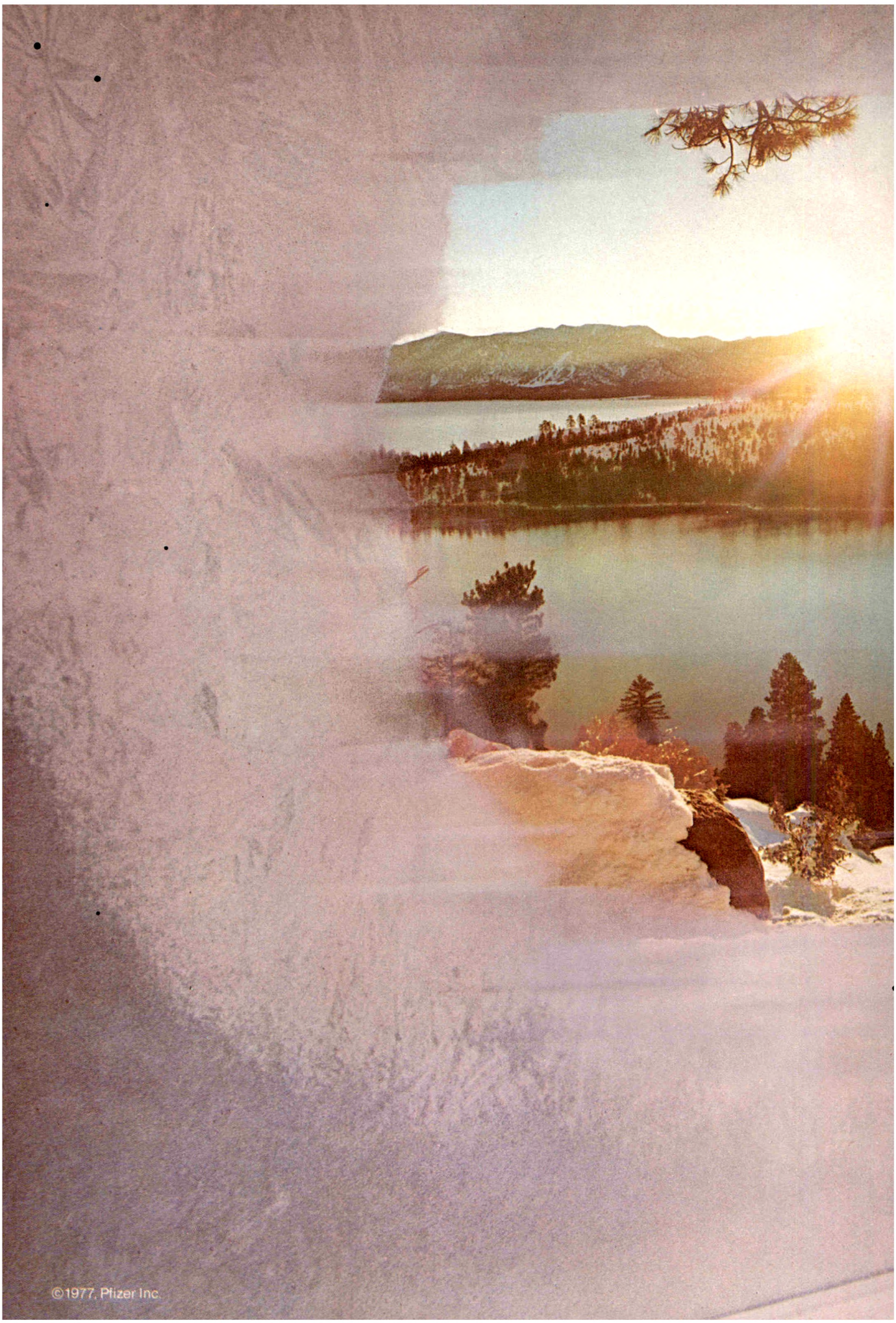
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INTRODUCING
NEW
SINEQUAN® 75 mg
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ANTIDEPRESSANT EFFECTIVENESS...
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*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.
See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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EXTENDS THE RANGE OF DOSAGE STRENGTHS

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which may improve patient compliance. The tota daily dosage, up to 150 mg per day, may be giver on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY GOOD TOLERATION

At doses up to 150 mg per day, Sinequan does no generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have occasionally been reported. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug. Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects. Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, starting dose of 25 mg t.i.d. is recommended. Dosage may subsequently be increased c decreased at appropriate intervals and according to individual response. The usual optimu dose range is 75 mg/day to 150 mg/day.

In more severely ill patients an initial dose of 50 mg t.i.d. may be required with subsequer gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to b obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organ disease, lower doses may suffice. Some of these patients have been controlled on doses as lo as 25-50 mg/day.

As an alternative regimen, the total daily dosage, up to 150 mg, may be given on a once-a-da schedule without loss of effectiveness. This dose may be given at bedtime.

Anti-anxiety effect is apparent before the antidepressant effect. Optimal antidepressant effe may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.

2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias an tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.

2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressiv supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions t prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbe The use of activated charcoal has been recommended, as has been continuous gastric lavag with saline for 24 hours or more. An adequate airway should be established in comatose patient and assisted ventilation used if necessary. EKG monitoring may be required for several day since relapse after apparent recovery has been reported. Arrhythmias should be treated with th appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CN symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intr venous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine rapidly metabolized, the dosage should be repeated as required. Convulsions may respond t standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depre sion. Dialysis and forced diuresis generally are not of value in the management of overdosag due to high tissue and protein binding of SINEQUAN.

Supply. Available as capsules containing doxepin HCl equivalent to 10 mg, 25 mg, 50 m 75 mg, and 100 mg doxepin in bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's SINEQUAN 25 mg and 50 mg are also available in bottles of 5000. SINEQUAN Oral Concentrat (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 m 10 mg, 15 mg, 20 mg, and 25 mg. Just prior to administration, SINEQUAN Oral Concentrat should be diluted with approximately 120 ml of water, whole or skimmed milk, or orang grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physical compatible with a number of carbonated beverages. For those patients requiring antidepressar therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadon syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, c water; but not with grape juice. Preparation and storage of bulk dilutions is not recommende

More detailed professional information available on request.

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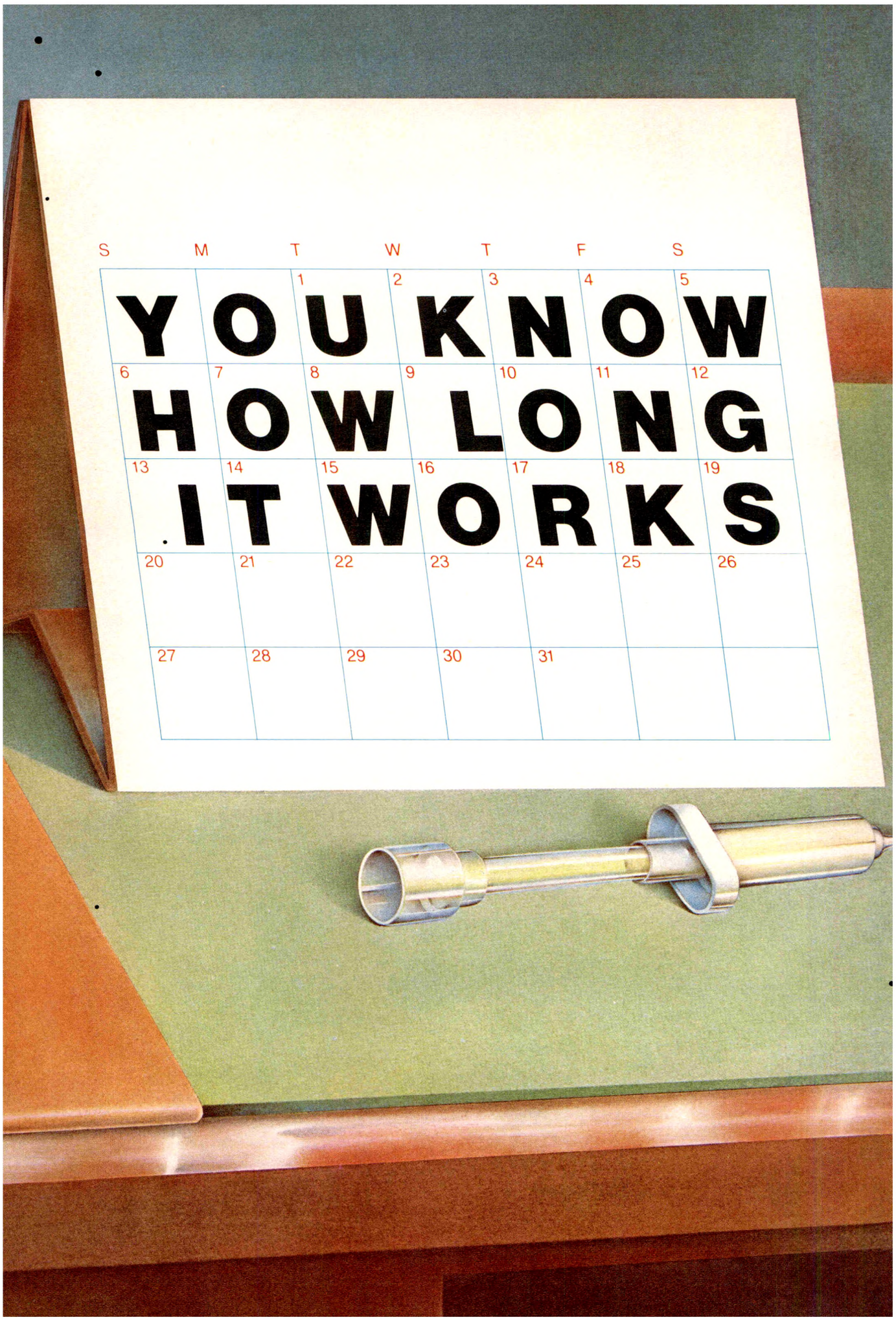
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HERE'S HOW WELL IT WORKS

Rapidly effective in acute schizophrenia[1]

Average length of hospitalization was 6.1 days (range 1 to 8) for 13 acutely psychotic schizophrenic patients treated with Prolixin Decanoate using the rapid neuroleptization technique. Dosage the first 72 hours averaged 73.2 mg.

Psychotic symptoms diminished rapidly during the first week and continued to abate throughout the 2-month study.

Statistically significant improvement in 13 target symptoms of the Brief Psychiatric Rating Scale, including disorientation, hostility and uncooperativeness, occurred between pretreatment and final evaluation.

Often works where others fail...[2]

Of 52 chronic schizophrenic patients, hospitalized an average 17.5 years, 71% showed clinical improvement after Prolixin Decanoate therapy (25 mg every 7 to 14 days). 32 patients were refractory to prior therapy, including haloperidol combined with chlorpromazine or thioridazine.

General mobilization of the patients was the most significant effect. 7 patients could be discharged from the hospital. In 30 patients refractory to other medication, Prolixin Decanoate tended to inhibit withdrawal symptoms.

...at a fraction of the cost

Prolixin Decanoate therapy at a maintenance dose of 25 mg. every 28 days can cost from 51% to 69% less than the lowest maintenance dosages of several oral medications (see table below).

Comparison of estimated annual hospital costs for oral and injectable therapy, given the following examples:

Drug	Assumed adult dosage*	Form and potency	Cost per unit	Annual cost
Prolixin® Decanoate	12.5-100 mg every 28 days	5 ml vials 25 mg/ml	8.5¢† per mg	$ 13.81 – 110.50
Thorazine®(SKF brand of chlorpromazine)	300-600 mg per day	100 mg tablets bottles of 1000	5.1¢ per tab.**	55.84 – 111.69
Mellaril®(Sandoz brand of thioridazine)	200-800 mg per day	100 mg tablets bottles of 1000	12¢ per tab.**	87.60 – 350.40
Haldol®(McNeil brand of haloperidol)	5-50 mg per day	5 mg tablets bottles of 1000	17.1¢ per tab.**	62.42 – 624.15
Navane®(Roerig brand of thiothixene)	20-60 mg per day	20 mg tablets bottles of 500	24.7¢ per tab.**	90.16 – 270.47

*Dosage level and interval should be determined on an individual basis, in accordance with patient's response to the particular drug as well as the manufacturer's specific recommendations for the use of the product. **Based on prices listed in 1977 Red Book.†Based on Squibb list price of $10.65 per 5 ml vial.

1. Donlon PT, Axelrad AD, Tupin JP and Chien C-p: Comp Psychiat 17:369-376, 1976.
2. Christodoulidis H and Frangos H: Curr Ther Res 18:193-198, 1975.

PROLIXIN® DECANOATE
Fluphenazine Decanoate Injection

See following page for brief summary.

PROLIXIN DECANOATE®

Fluphenazine Decanoate Injection

Often works where others fail... at a fraction of the cost

Prolixin Decanoate (Fluphenazine Decanoate Injection) provides 25 mg. fluphenazine decanoate per ml. in a sesame oil vehicle with 1.2% (w/v) benzyl alcohol as a preservative.

CONTRAINDICATIONS: In presence of suspected or established subcortical brain damage. In patients who have a blood dyscrasia or liver damage, or who are receiving large doses of hypnotics, or who are comatose or severely depressed. In patients who have shown hypersensitivity to fluphenazine; cross-sensitivity to phenothiazine derivatives may occur.

Not intended for use in children under 12.

WARNINGS: Mental and physical abilities required for driving a car or operating heavy machinery may be impaired by use of this drug. Physicians should be alert to the possibility that severe adverse reactions may occur which require immediate medical attention. Potentiation of effects of alcohol may occur. Safety and efficacy in children have not been established because of inadequate experience in use in children.

Usage in Pregnancy: Safety for use during pregnancy has not been established; weigh possible hazards against potential benefits if administering this drug to pregnant patients.

PRECAUTIONS: Caution must be exercised if another phenothiazine compound caused cholestatic jaundice, dermatoses or other allergic reactions because of the possibility of cross-sensitivity. When psychotic patients on large doses of a phenothiazine drug are to undergo surgery, hypotensive phenomena should be watched for; less anesthetics or central nervous system depressants may be required. Because of added anticholinergic effects, fluphenazine may potentiate the effects of atropine.

Use fluphenazine decanoate cautiously in patients exposed to extreme heat or phosphorus insecticides; in patients with a history of convulsive disorders since grand mal convulsions have occurred; and in patients with special medical disorders such as mitral insufficiency or other cardiovascular diseases, and pheochromocytoma. Bear in mind that with prolonged therapy there is the possibility of liver damage, pigmentary retinopathy, lenticular and corneal deposits, and development of irreversible dyskinesia.

Fluphenazine decanoate should be administered under the direction of a physician experienced in the clinical use of psychotropic drugs. Periodic checking of hepatic and renal functions and blood picture should be done. Renal function of patients on long-term therapy should be monitored; if BUN becomes abnormal, treatment should be discontinued. "Silent pneumonias" are possible.

ADVERSE REACTIONS: *Central Nervous System*—Extrapyramidal symptoms are most frequently reported. These include pseudoparkinsonism, dystonia, dyskinesia, akathisia, oculogyric crises, opisthotonos, and hyperreflexia; most often these are reversible, but they may be persistent. One can expect a higher incidence of such reactions with fluphenazine decanoate than with less potent piperazine derivatives or straight-chain phenothiazines. The incidence and severity will depend more on individual patient sensitivity, but dosage level and patient age are also determinants. As these reactions may be alarming, the patient should be forewarned and reassured. These reactions can usually be controlled by administration of antiparkinsonian drugs such as benztropine mesylate or intravenous Caffeine and Sodium Benzoate Injection U.S.P., and by subsequent reduction in dosage.

Persistent Tardive Dyskinesia: As with all antipsychotic agents, persistent and sometimes irreversible tardive dyskinesia may appear in some patients on long-term therapy or may occur after discontinuation of drug. The risk seems greater in elderly patients, especially females, on high dosages. The syndrome is characterized by rhythmical involuntary movements of tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and may be accompanied by involuntary movements of extremities. There is no known effective therapy for tardive dyskinesia; usually the symptoms are not alleviated by antiparkinsonism agents. If the symptoms appear, discontinuation of all antipsychotic agents is suggested. The syndrome may be masked if treatment is reinstituted, or drug dosage increased, or a different antipsychotic agent used. Reports are that fine vermicular movements of the tongue may be an early sign of the syndrome which may not develop if medication is stopped at that time.

Phenothiazine derivatives have been known to cause restlessness, excitement, or bizarre dreams; reactivation or aggravation of psychotic processes may be encountered. If drowsiness or lethargy occur, the dosage may have to be reduced. Dosages, far in excess of the recommended amounts, may induce a catatonic-like state.

Autonomic Nervous System—Hypertension and fluctuations in blood pressure have been reported. Although hypotension is rarely a problem, patients with pheochromocytoma, cerebral vascular or renal insufficiency or severe cardiac reserve deficiency such as mitral insufficiency appear to be particularly prone to this reaction and should be observed carefully. Supportive measures including intravenous vasopressor drugs should be instituted immediately should severe hypotension occur; Levarterenol Bitartrate Injection U.S.P. is the most suitable drug; *epinephrine should not be used* since phenothiazine derivatives have been found to reverse its action. Nausea, loss of appetite, salivation, polyuria, perspiration, dry mouth, headache and constipation may occur. Reducing or temporarily discontinuing the dosage will usually control these effects. Blurred vision, glaucoma, bladder paralysis, fecal impaction, paralytic ileus, tachycardia, or nasal congestion have occurred in some patients on phenothiazine derivatives.

Metabolic and Endocrine—Weight change, peripheral edema, abnormal lactation, gynecomastia, menstrual irregularities, false results on pregnancy tests, impotency in men and increased libido in women have occurred in some patients on phenothiazine therapy.

Allergic Reactions—Itching, erythema, urticaria, seborrhea, photosensitivity, eczema and exfoliative dermatitis have been reported with phenothiazines. The possibility of anaphylactoid reactions should be borne in mind.

Hematologic—Blood dyscrasias including leukopenia, agranulocytosis, thrombocytopenic or nonthrombocytopenic purpura, eosinophilia, and pancytopenia have been observed with phenothiazines. If soreness of the mouth, gums or throat or any symptoms of upper respiratory infection occur and confirmatory leukocyte count indicates cellular depression, therapy should be discontinued and other appropriate measures instituted immediately.

Hepatic—Liver damage manifested by cholestatic jaundice, particularly during the first months of therapy, may occur; treatment should be discontinued. A cephalin flocculation increase, sometimes accompanied by alterations in other liver function tests, has been reported in patients who have had no clinical evidence of liver damage.

Others—Sudden deaths have been reported in hospitalized patients on phenothiazines. Previous brain damage or seizures may be predisposing factors. High doses should be avoided in known seizure patients. Shortly before death, several patients showed flare-ups of psychotic behavior patterns. Autopsy findings have usually revealed acute fulminating pneumonia or pneumonitis, aspiration of gastric contents, or intramyocardial lesions. Although not a general feature of fluphenazine, potentiation of central nervous system depressants such as opiates, analgesics, antihistamines, barbiturates, and alcohol may occur.

Systemic lupus erythematosus-like syndrome, hypotension severe enough to cause fatal cardiac arrest, altered electrocardiographic and electroencephalographic tracings, altered cerebrospinal fluid proteins, cerebral edema, asthma, laryngeal edema, and angioneurotic edema; with long-term use, skin pigmentation and lenticular and corneal opacities have occurred with phenothiazines. Local tissue reactions occur only rarely with injections of fluphenazine decanoate.

For full prescribing information, consult package insert.

HOW SUPPLIED: 1 ml. Unimatic® single dose preassembled syringes and cartridge-needle units, and 5 ml. vials.

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Basic tool of primitive psychiatry



Fertility protective statues: For pregnant women, the statues insured a healthy, beautiful child, and protected both mother and child from the "evil eye." For sterile women, the statues' magical power aided fertility. Ashanti, Ghana.

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Basic tool of modern psychiatry

Thorazine®

brand of **chlorpromazine**

Tablets: 25 and 50 mg of the HCl

- Effective control of psychotic symptoms
- Especially useful in agitated, violent or anxious schizophrenic patients
- Unsurpassed clinical experience
- 18 convenient dosage forms and strengths

Before prescribing, see complete prescribing information in SK&F literature or *PDR*. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings:

Caution is recommended when 'Thorazine' is administered for the treatment of vomiting in children.

Antiemetics are not recommended to treat uncomplicated short-term vomiting in children or vomiting of unknown etiology.

The possibility of extrapyramidal reactions from 'Thorazine' may confuse the diagnosis of Reye's syndrome or other encephalopathy. Although unconfirmed, some suspicion exists that centrally-acting antiemetics may contribute to or adversely affect the course of Reye's syndrome, and should be avoided in suspected cases.

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Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg., in bottles of 100; Single Unit Packages of 100 (intended for institutional use only). Spansule® capsules, 30 mg., 75 mg., 150 mg., 200 mg. and 300 mg., in bottles of 50; in Single Unit Packages of 100 (intended for institutional use only). Injection, 25 mg./ml.; Syrup, 10 mg./5 ml.; Suppositories, 25 mg. and 100 mg. Concentrate (intended for institutional use only), 30 mg./ml. and 100 mg./ml.

Smith Kline &French Laboratories
Philadelphia, Pa.

SK&F
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Helps Return Patients to Reality

in clinically significant depression...

penetrating the symptom barrier to productive psychotherapy

symptoms often interfere with therapy

In clinically significant depression, the patient often becomes barricaded behind his own symptoms during psychotherapy. Because these symptoms can be so sharply defined and somatic in expression, he may reject the fact that they are psychic in origin. Sleep disturbance, gastrointestinal complaints, anorexia, chronic fatigue, and other psychosomatic symptoms may so preoccupy—or overwhelm—the depressed patient that truly productive therapeutic sessions are difficult to achieve. Instead of being able to concentrate on the core emotional problems of the depression itself, he may for example go on at length about how broken his sleep is or keep reiterating (while the therapeutic hour slips by) how physically miserable he feels.

ELAVIL®
(AMITRIPTYLINE HCl, MSD)
to help penetrate the symptom barrier in depression requiring medication

ELAVIL, a highly effective tricyclic antidepressant, will often alleviate these symptoms. With the help of ELAVIL, sleep disturbance—so common in clinically depressed patients and often the symptom that distresses them most—as



well as other "barrier symptoms" may be lessened to the point where they no longer come between you and the patient.

psychotherapy takes direction

As these preoccupying complaints are lifted, psychotherapy sessions can become more productive. Patients are enabled to respond more fruitfully during therapeutic hours and to resume or pursue more efficiently the necessary activities in their personal lives.

meeting therapeutic goals

And as the antidepressant activity of ELAVIL (Amitriptyline HCl, MSD) takes hold, it helps relieve the depressive condition itself. Sometimes only a minimum of actual psychotherapy is required. The patient may experience a more rapid recovery—while you conserve valuable time.

Prescribe ELAVIL then, to help lighten the patient's burden in depression—and yours in its management.



ELAVIL should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks; or in children under 12. Patients with cardiovascular disorders should be watched closely. Safe use during pregnancy and lactation has not been established. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Hospitalize as soon as possible any patient suspected of having taken an overdose.

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, 100 mg, and 150 mg
INJECTION: 10 mg per ml

ELAVIL®

(AMITRIPTYLINE HCl | MSD)

an antidepressant with an anxiety-reducing sedative component to its action

MSD
MERCK SHARP & DOHME

For a brief summary of prescribing information, please see following page.

MSD
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addendum

To further simplify once-daily therapy for many depressed patients

Tablets

ELAVIL®

(AMITRIPTYLINE HCl | MSD)

ONE TABLET, ONCE DAILY

—an appropriate way to prescribe for many depressed adult outpatients. Because of its simplicity, this regimen helps improve patient compliance. Of course, ELAVIL may also be prescribed in divided daily doses.

ONCE-DAILY DOSAGE SCHEDULE FOR ADULT OUTPATIENTS:

A single 75-mg tablet
Usual starting dosage



A single 100-mg tablet
Maximum starting dosage



A single 50-mg tablet
Minimum starting dosage



Dosage may be increased by 25 or 50 mg as necessary until a total of 150 mg per day is reached.

A single 150-mg tablet
Maximum daily dosage



ELAVIL MAY ALSO BE PRESCRIBED IN DIVIDED DAILY DOSES

The 25-mg tablet



This strength may prove useful when therapy is initiated with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime dose.

The 10-mg tablet



This strength may prove useful for patients who require lower doses, e.g., adolescent and elderly patients. For these patients who can not tolerate higher doses, 10 mg three times a day with 20 mg at bedtime may be satisfactory.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.
When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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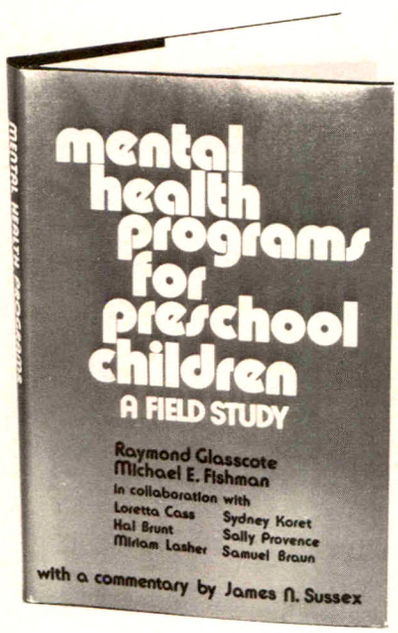
ELAVIL®
(AMITRIPTYLINE HCl, MSD)

MSD
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A Timely New Book from The Joint Information Service

This latest Joint Information Service field study provides a concise, comprehensive overview of the complex matter of the very young American child and his mental health, and then describes in intimate detail seven unique programs for pre-school children with problems—from North Hollywood's Dubnoff Center, emphasizing education as its primary therapeutic modality, to Baltimore's Martin Luther King, Jr. Parent-Child Center, serving two housing projects, served by Johns Hopkins, and largely governed by its own people. All different in treatment approach and philosophy, the programs described are uniform in their dedication to innovation in improving service to the emotionally disturbed very young child, and, equally, to the emotional well being of all young children.

This thoughtful and perceptive description of what has been created by some of the best informed, most experienced and thoughtful specialists in mental health services for very young children should be welcome to all involved in the mental health of children and a casebook for those dealing with child and family mental health.



An articulate exposition of the immense problems and encouraging accomplishments in a particularly difficult area of endeavor . . . . Beyond their concern with the manifestly ill child, the authors intriguingly explore what may be needed to enhance the mental good health of all young children . . . . A significant contribution.

James N. Sussex, M.D.
President, American Association of Psychiatric Services for Children

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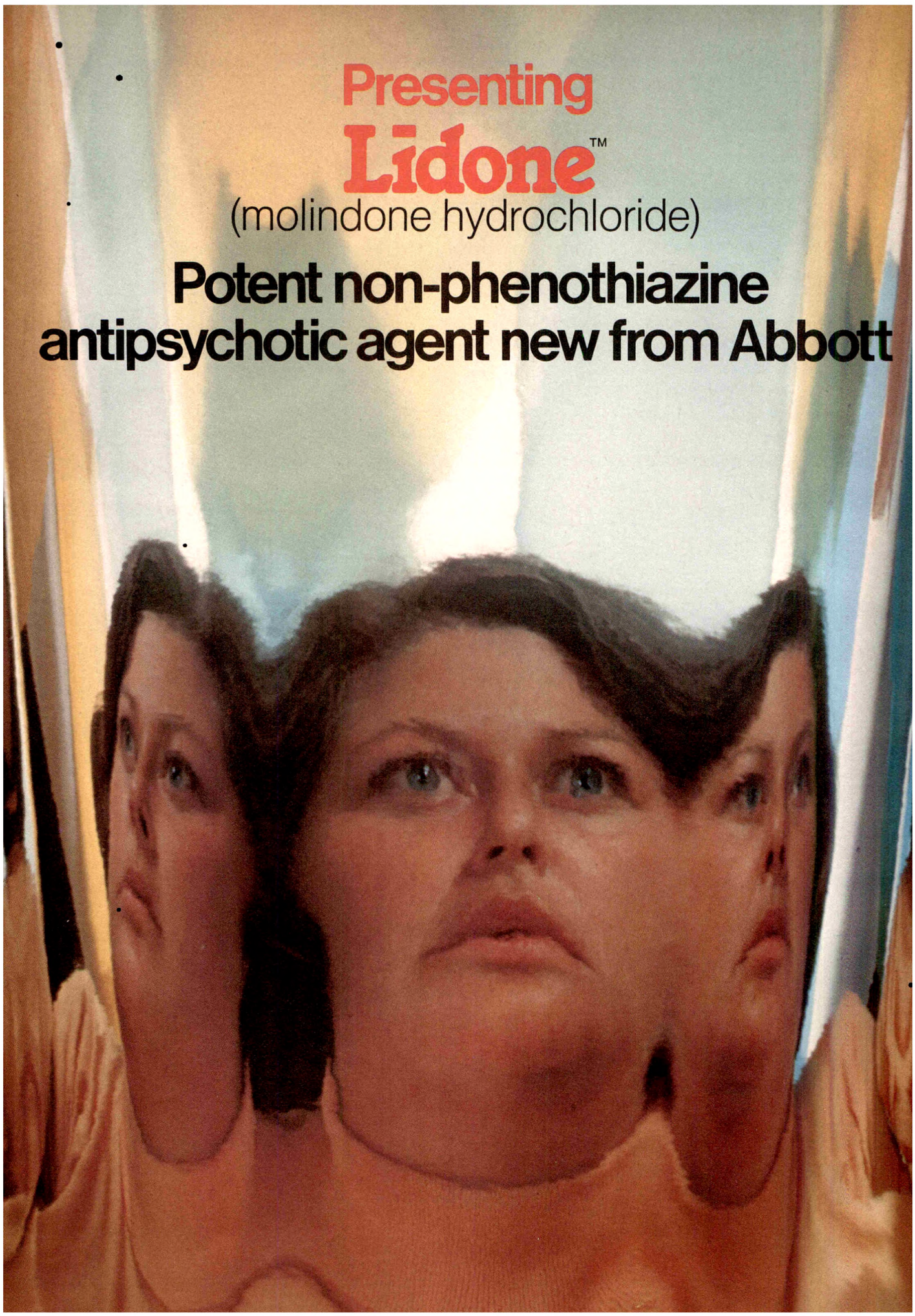
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Presenting
Lidone™
(molindone hydrochloride)
Potent non-phenothiazine
antipsychotic agent new from Abbott

(molindone
hydrochloride)

Lidone™ treats the psychosis with little likelihood of causing excessive weight gain or problem sedation

Lidone is molindone hydrochloride, a potent antipsychotic not related to other agents. It is effective against the manifestations of schizophrenia. Yet it is largely free of certain common problems.

Normalizing weight trend seen

Unlike some potent agents, Lidone has not caused excessive weight gain. Though both weight gain and loss have been seen, the shift has typically been toward normal or ideal weight.

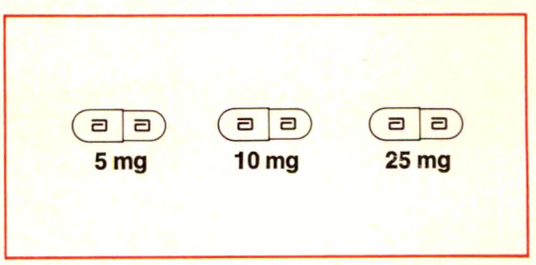
Benefits in alertness

Lidone is classed among the "alerting" or less-sedating group of major tranquilizers. If drowsiness does occur during initial therapy, it is usually transient. Patients on continuing Lidone therapy tend to stay alert; hence they can be more responsive to rehabilitation measures.

Improved social outlook

Patients on Lidone have shown good progress in social competence and interest. And side effects such as sexual dysfunction or weight gain, which might disrupt family or interpersonal relationships, have not been reported.

Please contact your Abbott Representative to arrange for a clinical evaluation.



Lidone™ (molindone hydrochloride)

See overleaf for prescribing information.

7083207

Brief summary of
prescribing information for

Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2 to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose —
20 mg/kg/day — 2 weeks, no adverse effect
40 mg/kg/day — 2 weeks, no adverse effect

Pregnant Mice oral dose —
20 mg/kg/day — 9 days, slight increase resorptions
40 mg/kg/day — 9 days, slight increase resorptions

Pregnant Rabbits oral dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to l greater in elderly patients on high-dose therap especially females. The symptoms are persiste and in some patients appear to be irreversible. Th syndrome is characterized by rhythmical involu tary movements of the tongue, face, mouth or ja (e.g., protrusion of tongue, puffing of cheek puckering of mouth, chewing movements). The may be involuntary movements of extremities.

There is no known effective treatment of tardi dyskinesia; antiparkinsonism agents usually not alleviate the symptoms of this syndrome. It suggested that all antipsychotic agents be disco tinued if these symptoms appear. Should it be ne essary to reinstitute treatment, or increase th dosage of the agent, or switch to a different an psychotic agent, the syndrome may be masked. has been reported that fine vermicular movemen of the tongue may be an early sign of the syndron and if the medication is stopped at that time th syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial ar maintenance doses of LIDONE (molindone hydr chloride) should be individualized, and th minimal effective dose should be employed. Elder and debilitated patients should be started on low dosage.

DOSAGE SCHEDULE, BASED ON SEVERITY OF SYMPTOMATOLOGY

1. Mild — 5 mg three or four times a day; an i crease to 15 mg three or four times a day may l required.
2. Moderate — 10 mg three or four times a day; a increase to 25 mg three or four times a day ma be required.
3. Severe — daily dosage as high as 225 mg may l required.

DRUG INTERACTIONS — Potentiation of dru administered concurrently with LIDONE (molin done hydrochloride) has not been reported. Add tionally, animal studies have not shown increase toxicity when LIDONE is given concurrently wit representative members of three classes of dru (i.e., barbiturates, chloral hydrate and antiparki son drugs).

MANAGEMENT OF OVERDOSAGE — Sympt matic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction absorption of LIDONE (molindone hydrochlorid which is freely soluble in water.

Since the adsorption of LIDONE (molindone h drochloride) by activated charcoal has not bee determined, the use of this antidote must be co sidered of theoretical value.

Emesis in a comatose patient is contraindicate Additionally, while the emetic effect apomorphine is blocked by LIDONE in animals, th blocking effect has not been determined humans.

A significant increase in the rate of removal unmetabolized LIDONE from the body by force diuresis, peritoneal or renal dialysis would not expected. (Only 2% of a single ingested dose LIDONE is excreted unmetabolized in the urine However, poor response of the patient may justi use of these procedures.

While the use of laxatives or enemas might b based on general principles, the amount of u metabolized LIDONE in feces is less than 1%. E trapyramidal symptoms have responded to the u of diphenhydramine (Benadryl*) and the sy thetic anticholinergic antiparkinson agents (i Artane*, Cogentin*, Akineton*).

HOW SUPPLIED — LIDONE (molindone h drochloride) capsules are supplied in bottles of 1 in the following dosage strengths and colors:

5 mg (NDC 0074-5542-13) blue and cream
10 mg (NDC 0074-5543-13) red and cream
25 mg (NDC 0074-5544-13) brown and cream.

TM — Trademark
*Benadryl — Trademark, Parke Davis and Co.
*Artane — Trademark, Lederle Laboratories
*Cogentin — Trademark, Merck Sharp & Dohme
*Akineton — Trademark, Knoll Pharmaceutical Co.

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Progressive community mental health center in central Arkansas needs two innovative and energetic community-oriented psychiatrists. Responsibilities include: working in small, short-term inpatient unit; providing pre- and aftercare services; consulting with staff and local medical community; and carrying a selected outpatient case load.
Excellent fringe benefits; salaries open. Interested parties contact Dr. Alan G. Tuft, Human Services Center, P. O. Box 1398, Russellville, Arkansas 72801.
The Human Services Center is an equal opportunity employer.

THE UNIVERSITY OF VERMONT COLLEGE OF MEDICINE

Department of Psychiatry

offers

a comprehensive, eclectic 4 year residency training program in psychiatry to medical graduates. The first year includes 4 months medicine/3 months psychiatry/2 months neurology and an elective. The second year, (available also to those who have completed the first postgraduate year elsewhere), includes an inpatient experience at the Medical Center Hospital of Vermont. During this year, the resident will begin longterm psychotherapy under intensive supervision. The third year emphasizes ambulatory experiences, including child and adolescent psychiatry. Rotations in consultation-liaison, community mental health and selective experiences complete the program.

Child psychiatry and psychosomatic fellowships are available.

For further information and application materials write: Barry Nurcombe, M.D., Director of Residency Training, UVM Department of Psychiatry, Medical Alumni Building, Burlington, Vermont 05401.

IMMEDIATE OPENINGS FOR PSYCHIATRIST AT RICHARD H. HUTCHINGS PSYCHIATRIC CENTER

THE RICHARD H. HUTCHINGS PSYCHIATRIC CENTER IS A PROGRESSIVE COMMUNITY MENTAL HEALTH CENTER LOCATED IN SYRACUSE IN CENTRAL NEW YORK. THE CENTER SERVES A FIVE COUNTY REGION AND PROVIDES PROGRAMS FOR ADOLESCENTS AND ADULTS. THE NEWLY CONSTRUCTED HOSPITAL OFFERS A WIDE VARIETY OF PATIENT SERVICES. OPPORTUNITIES INCLUDE:

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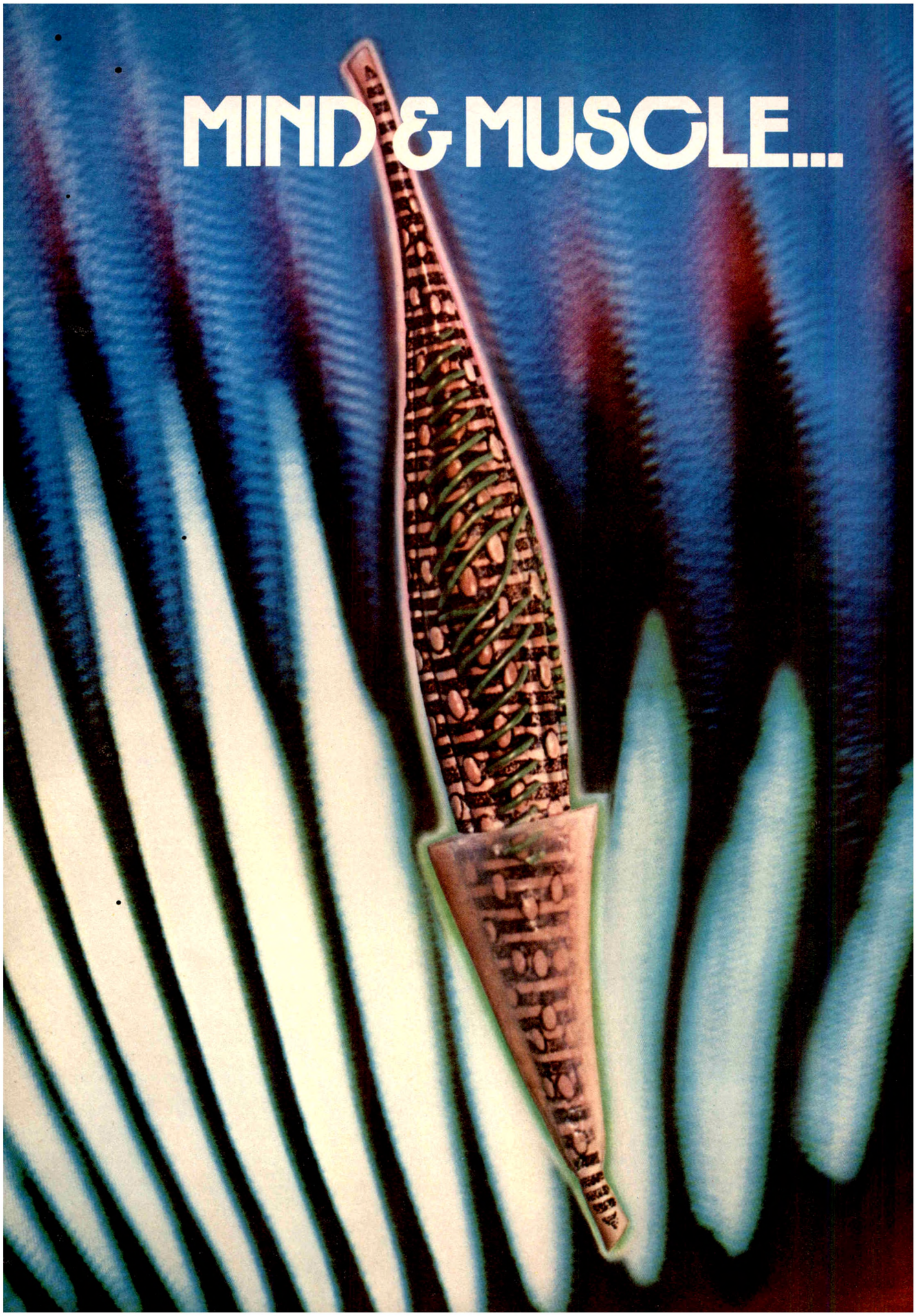
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FOR MORE INFORMATION CONTACT:
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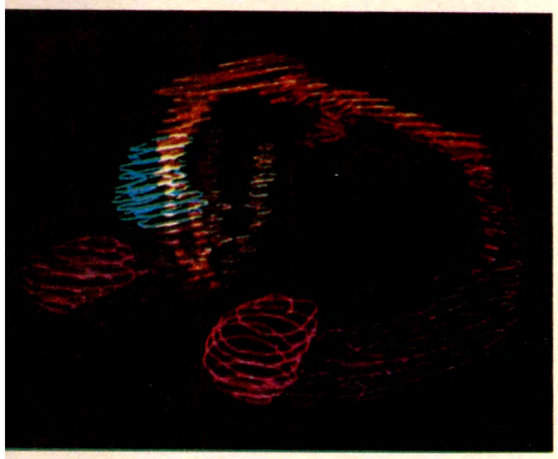
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MIND & MUSCLE...

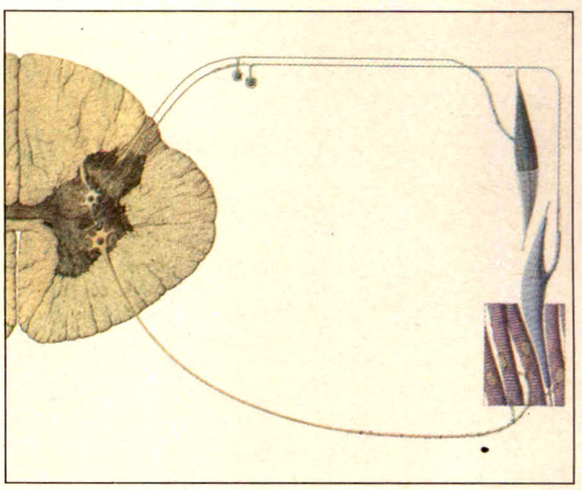
ONLY VALIUM (diazepam) HAS THESE TWO DISTINCT EFFECTS...

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alium is useful for the relief of undue sychic tension and anxiety whether seen one or associated with organic or functonal disorders, or with secondary depressive symptoms. Valium provides a quality response you know, want and trust. A esponse which has a pronounced effect n the somatic symptoms of anxiety.



MUSCLE: SKELETAL MUSCLE RELAXANT



Preliminary studies in both animals and humans have suggested that Valium (diazepam) may also work at the spinal level by enhancing presynaptic inhibition, a mechanism believed to diminish spasm in skeletal muscle. Clinically, adjunctive Valium has significant skeletal muscle relaxant properties.

VALIUM® (diazepam) IV

2-MG, 5-MG, 10-MG SCORED TABLETS

FOR THE RESPONSE YOU KNOW, WANT AND TRUST

Before prescribing, please see following page for a summary of product information.

ROCHE

A75

VALIUM®
(diazepam)

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology, spasticity caused by upper motor neuron disorders, athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

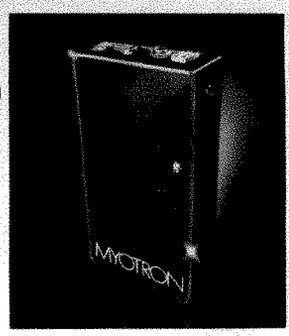
Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

ROCHE

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The first complete pocketsize system to monitor muscle activity.

Immediate visual feedback: three light-emitting diodes.

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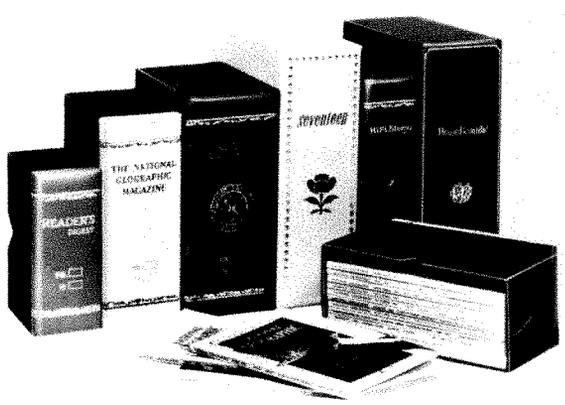
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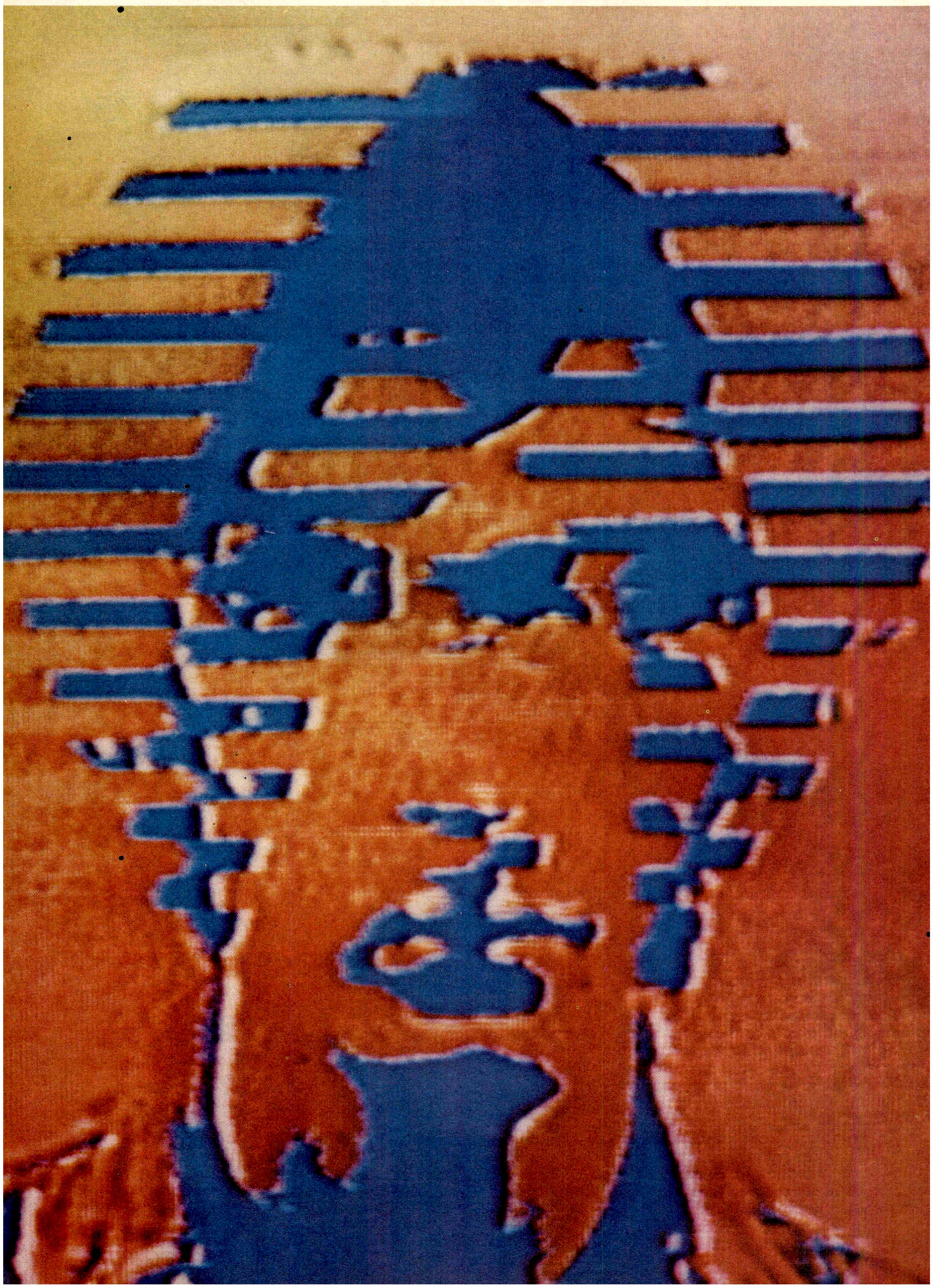
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MOBAN®
molindone HCl

- an effective antipsychotic agent
- not a phenothiazine

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NO incidence	Tolerance development Hepatotoxic changes Ophthalmological changes Excessive weight gain Impotence Decreased libido Skin pigmentation Thyroid function changes Persistent tardive dyskinesia
LOW incidence	Sedation Adverse drug interactions Hypotension Hematological changes Lactation Amenorrhea Autonomic changes Skin rash Nonpersistent symptoms of tardive dyskinesia
MODERATE incidence	Extrapyramidal reactions

*This table compiled from clinical and field reports, according to the best available information

Further advantages with MOBAN®

The pharmacological effect from a single oral dose persists for 24-36 hours, permitting *h.s.* dosage, once the daily dosage level is established.

Several clinicians have reported that obese patients on MOBAN® frequently experienced a marked reduction in weight toward normal weight.

How to use MOBAN®

The usual starting dose is 50-75 mg/day. You can increase to 100 mg/day in 3-4 days. Titrate individually up or down depending on patient response. Some chronic, treatment-resistant patients may require an increase to 400 mg/day. (However, the long-term safety of 400 mg/day has not been established.) Please refer to complete prescribing information for further dosage instructions.

For complete prescribing information, please see next page.

Endo Laboratories, Inc.
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Garden City, New York 11530

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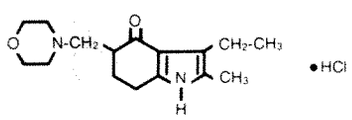
MOBAN®

5 mg	10 mg	25 mg

molindone HCl

DESCRIPTION MOBAN® (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

MOBAN® is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4 (5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.



MOLINDONE HYDROCHLORIDE

ACTIONS MOBAN® (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, MOBAN® antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, MOBAN® exerts its effect on the ascending reticular activating system.

Human metabolic studies show MOBAN® (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24-36 hours. There are 36 recognized metabolites with less than 2-3% unmetabolized MOBAN® being excreted in urine and feces.

INDICATIONS MOBAN® (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS MOBAN® (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS Usage in Pregnancy: Studies in the pregnant patient have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose— 20 mg/kg/day—10 days
no adverse effect
40 mg/kg/day—10 days
no adverse effect

Pregnant Mice oral dose— 20 mg/kg/day—10 days
slight increase resorptions
40 mg/kg/day—10 days
slight increase resorptions

Pregnant Rabbits oral dose— 5 mg/kg/day—12 days
no adverse effect
10 mg/kg/day—12 days
no adverse effect
20 mg/kg/day—12 days
no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of MOBAN® (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of MOBAN® (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS Some patients receiving MOBAN® (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving MOBAN® Caution should be exercised where increased activity may be harmful.

MOBAN® does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

The physician should be aware that this tablet preparation contains calcium sulfate as an excipient and that calcium ions may interfere with the absorption of preparations containing phenytoin sodium and tetracyclines.

MOBAN® has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS CNS EFFECTS The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological

Extrapyramidal Reactions

Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia

Motor restlessness may occur early.

Parkinson Syndrome

Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome

Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System

Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological

There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with MOBAN® may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects

Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with MOBAN® therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with MOBAN®.

Cardiovascular

Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological

Lens opacities and pigmentary retinopathy have not been reported where patients have received MOBAN® (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with MOBAN®.

Skin

Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with MOBAN® usage alone.

MOBAN® (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when MOBAN® is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia

Although rarely reported with MOBAN® (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). There may be involuntary movements of extremities.

There is no known effective treatment of tardive dyskinesia; antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear. Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked. It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time the syndrome may not develop.

DOSAGE AND ADMINISTRATION Initial and maintenance doses of MOBAN® (molindone hydrochloride) should be individualized.

Initial Dosage Schedule

The usual starting dosage is 50-75 mg/day.

—Increase to 100 mg/day in 3 or 4 days.

—Based on severity of symptomatology, dosage may be titrated up or down depending on individual patient response.

—An increase to 225 mg/day may be required in patients with severe symptomatology.

—Some chronic, treatment resistant patients, may require up to 400 mg/day (however, the long term safety of 400 mg/day has not been established).

Elderly and debilitated patients should be started on lower dosage.

Maintenance Dosage Schedule

1. Mild—5 mg-15 mg three or four times a day.
2. Moderate—10 mg-25 mg three or four times a day.
3. Severe—225 mg/day may be required.

Dosages may be administered once a day.

DRUG INTERACTIONS Potentiation of drugs administered concurrently with MOBAN® (molindone hydrochloride) has not been reported. Additionally, animal studies have not shown increased toxicity when MOBAN® is given concurrently with representative members of three classes of drugs (i.e., barbiturates, chloral hydrate and antiparkinson drugs).

MANAGEMENT OF OVERDOSAGE Symptomatic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction of absorption of MOBAN® (molindone hydrochloride) which is freely soluble in water.

Since the adsorption of MOBAN® (molindone hydrochloride) by activated charcoal has not been determined, the use of this antidot must be considered of theoretical value.

Emesis in a comatose patient is contraindicated. Additionally, whil the emetic effect of apomorphine is blocked by MOBAN® in animals this blocking effect has not been determined in humans.

A significant increase in the rate of removal of unmetabolized MOBAN® from the body by forced diuresis, peritoneal or renal dialysis would not be expected. (Only 2% of a single ingested dose of MOBAN® is excreted unmetabolized in the urine.)

However, poor response of the patient may justify use of these procedures. While the use of laxatives or enemas might be based o general principles, the amount of unmetabolized MOBAN® in feces is less than 1%. Extrapyramidal symptoms have responded to the use of diphenhydramine (Benadryl*) and the synthetic anticholinergic antiparkinson agents (i.e., Artane*, Cogentin*, Akineton*).

HOW SUPPLIED As tablets in bottles of 100 and 1000 with potencie and colors as follows: 5 mg orange, 10 mg lavender, 25 mg light green.

*Benadryl—Trademark, Parke Davis and Co.
*Artane—Trademark, Lederle Laboratories
*Cogentin—Trademark, Merck Sharp & Dohme
*Akineton—Trademark, Knoll Pharmaceutical Co.

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DUPONT

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Tranxene® (IV)

CAPSULES
SINGLE DOSE TABLETS

CLORAZEPATE DIPOTASSIUM 4306CB **Brief Summary**

ACTIONS—Pharmacologically, **TRANXENE** (clorazepate dipotassium) has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine.

INDICATIONS—TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE for long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS—TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS—TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS—In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS—The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION—

For symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg TRANXENE initially, followed by 30 to 60 mg in divided doses; 2nd 24 hours, 45 to 90 mg in divided doses; 3rd 24 hours, 22.5 to 45 mg in divided doses; 4th day, 15 to 30 mg in divided doses. Thereafter gradually reduce to 7.5 to 15 mg daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS—If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE (clorazepate dipotassium) is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE—Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed, since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY—Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption–Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED—TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

7.5 mg. capsules (gray with maroon cap) in bottles of 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3418-11).

15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3419-11).

TRANXENE-SD single dose tablets are supplied in two dosage strengths:

TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 100 (NDC 0074-2699-13).

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In this issue

The Neurobiological Origins of Psychoanalytic Dream Theory
By Robert W. McCarley and J. Allan Hobson

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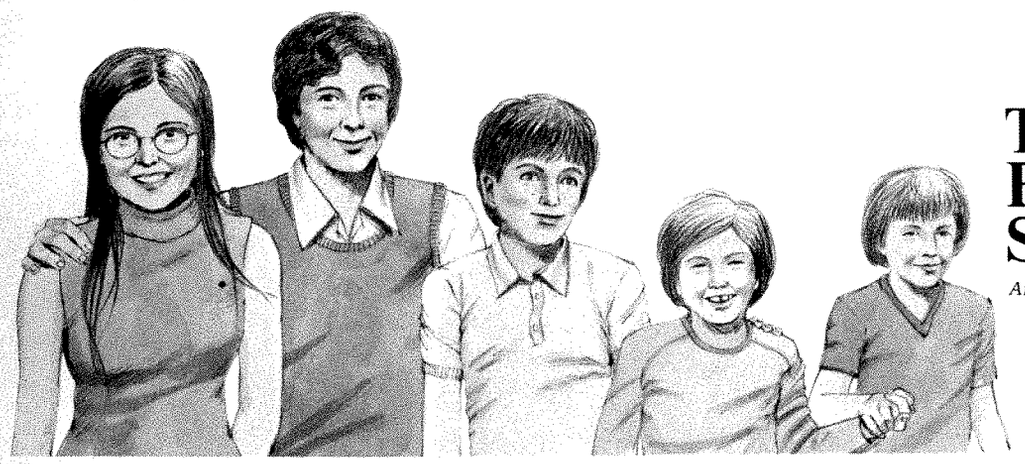
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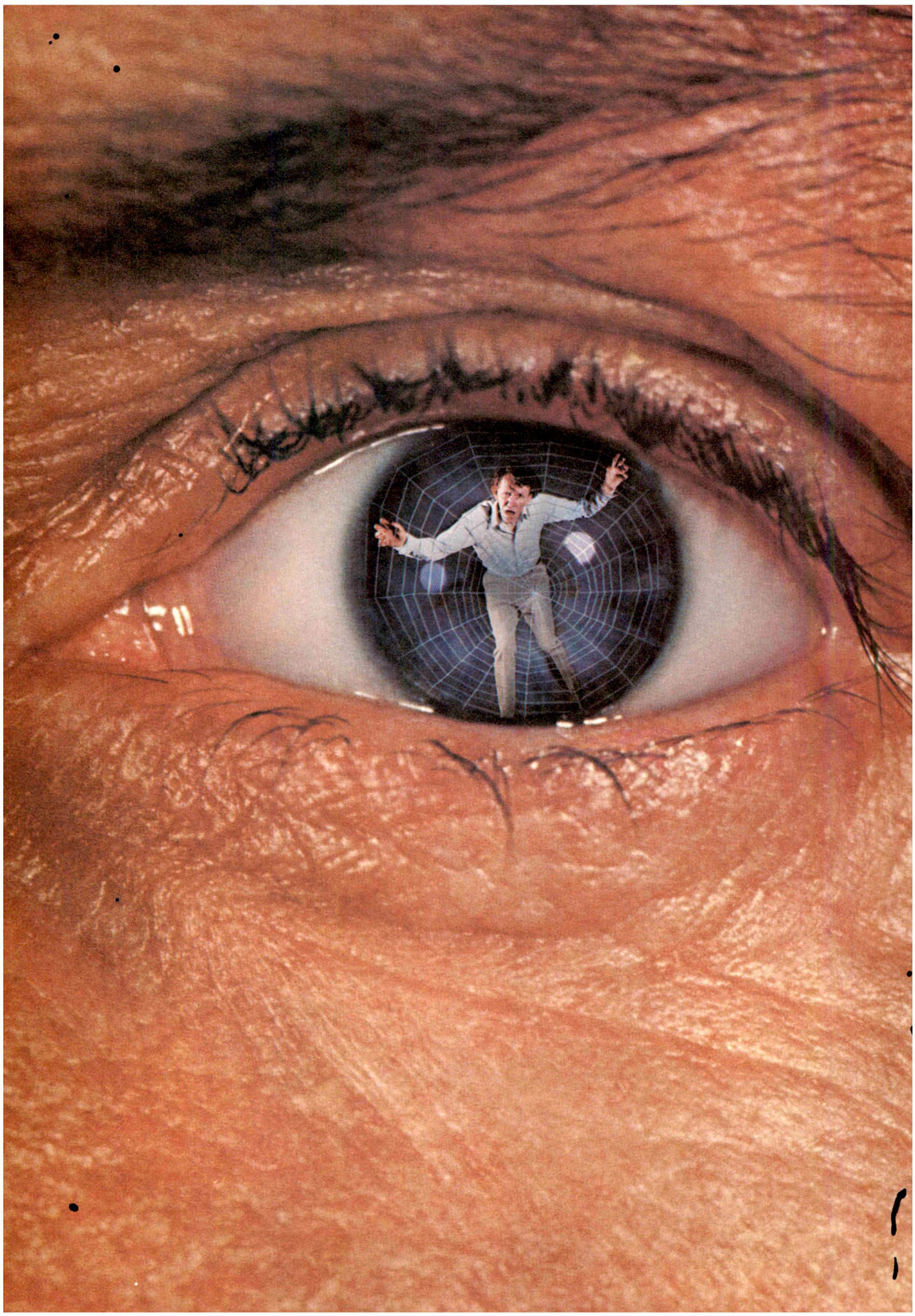
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Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy* – Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children–The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration – As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate–In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg. of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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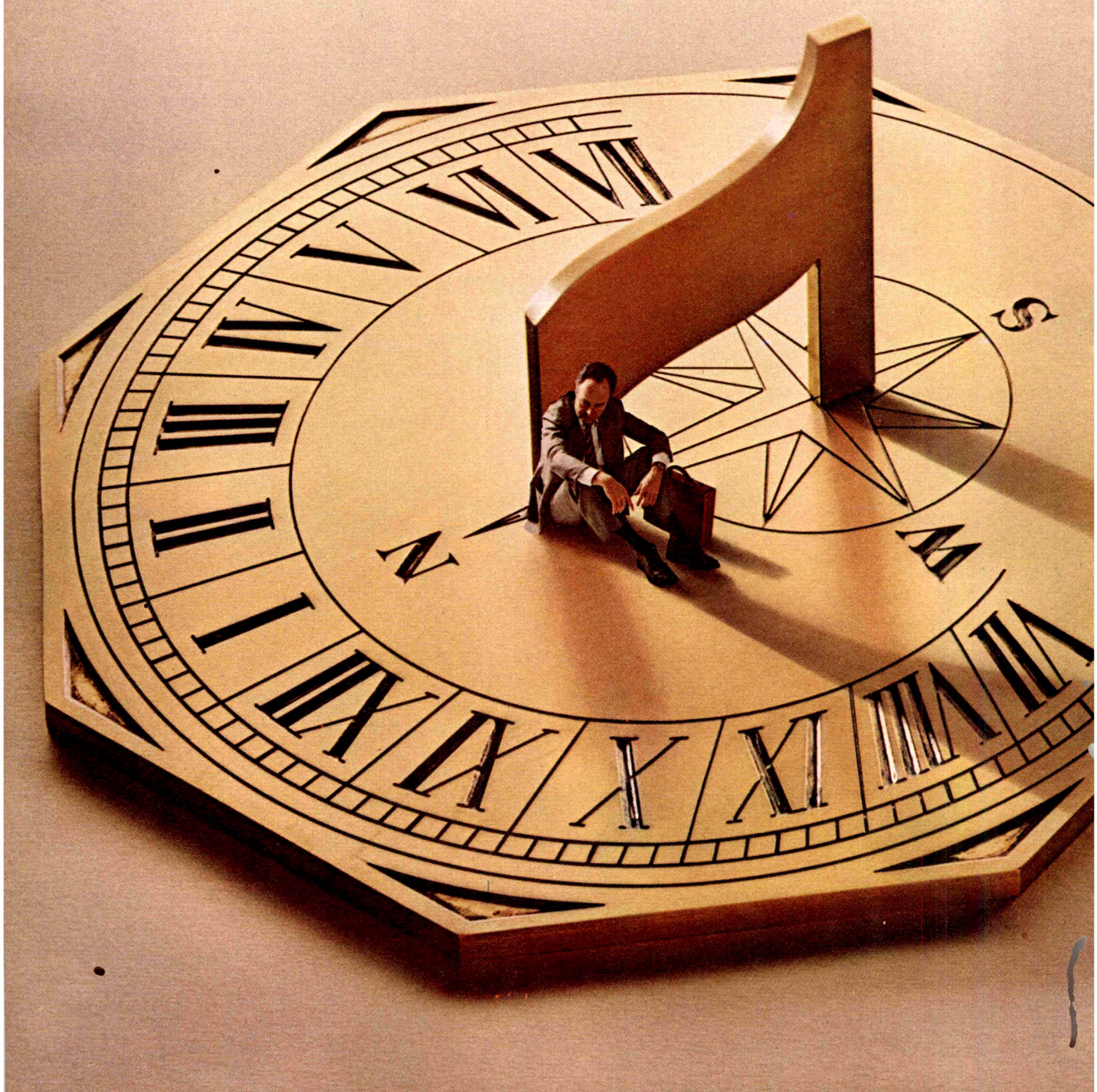
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Brief Summary

Indications: Norpramin (desipramine hydrochloride tablets NF) is indicated for the relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Desipramine hydrochloride should not be given within two weeks of treatment with a monoamine oxidase inhibitor. Contraindications include the acute recovery period following myocardial infarction and hypersensitivity to the drug. Cross sensitivity with other dibenzazepines is a possibility.

Warnings: 1. Extreme caution should be used in patients: (a) with cardiovascular disease, (b) with a history of urinary retention or glaucoma, (c) with thyroid disease or those on thyroid medication, (d) with a history of seizure disorder. 2. This drug is capable of blocking the antihypertensive effect of guanethidine and similarly acting compounds. 3. *Use in Pregnancy:* Safe use during pregnancy and lactation has not been established. 4. *Use in Children:* Norpramin is not recommended for use in children. 5. This drug may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery. Therefore, the patient should be cautioned accordingly. 6. In patients who may use alcohol excessively, it should be borne in mind that the potentiation may increase the danger inherent in any suicide attempt or overdosage.

Precautions: This drug should be dispensed in the least possible quantities to depressed outpatients, since suicide has been accomplished with drugs of this class. If possible, dispense in child-resistant containers. It should be kept out of reach of children. Reduce dosage, or alter treatment, if serious adverse effects occur. Norpramin therapy in patients with manic-depressive illness may induce a hypomanic state after the depressive phase terminates and may cause exacerbation of psychosis in schizophrenic patients. Use cautiously with anticholinergic or sympathomimetic drugs. Response to alcoholic beverages may be exaggerated. In the concurrent administration of ECT and antidepressant drugs one should consider the possibility of increased risk relative to benefits. The sedative effects of Norpramin and benzodiazepines (e.g., chlordiazepoxide or diazepam) are additive. Both the sedative and anticholinergic effects of the major tranquilizers are additive to those of Norpramin. Discontinue as soon as possible prior to elective surgery because of possible cardiovascular effects. Hypertensive episodes have been observed during surgery in patients on desipramine hydrochloride. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is neutropenia.

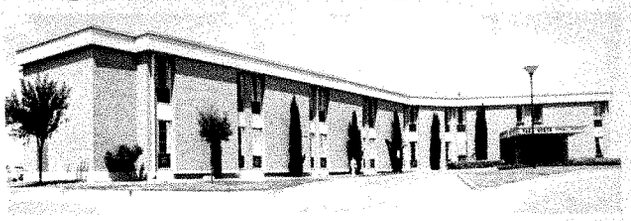
Adverse Reactions: *Cardiovascular:* hypotension, hypertension, tachycardia, palpitation, arrhythmias, heart block, myocardial infarction, stroke. *Psychiatric:* confusional states (especially in the elderly), hallucinations, disorientation, delusions, anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis. *Neurological:* numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures; alteration in EEG patterns; tinnitus. *Anticholinergic:* dry mouth, and rarely associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, hypotonic bladder. *Allergic:* skin rash, petechiae, urticaria, itching, photosensitization, edema (of face and tongue or general), drug fever, cross sensitivity with other tricyclic drugs. *Hematologic:* bone marrow depressions including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* anorexia, nausea and vomiting, epigastric distress, peculiar taste, abdominal cramps, diarrhea, stomatitis, black tongue. *Endocrine:* gynecomastia, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence, testicular swelling; elevation or depression of blood sugar levels. *Other:* jaundice (simulating obstructive); altered liver function; weight gain or loss, perspiration, flushing; urinary frequency, nocturia; parotid swelling; drowsiness, dizziness, weakness and fatigue, headache; alopecia. *Withdrawal symptoms:* Though not indicative of addiction, abrupt cessation after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: *Usual adult dose:* 100 mg. to 200 mg. per day. Dosages above 200 mg. per day are not recommended. *Initial therapy:* Should be administered at a low level and increased according to tolerance and response. It may be administered in divided doses or a single daily dose. *Maintenance:* Lower adequate dose once daily to maintain remission. *Adolescent and geriatric patient dose:* 25 mg. to 100 mg. per day, in single or divided doses. Dosages above 100 mg. are not recommended.

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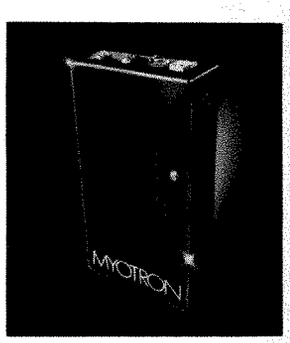
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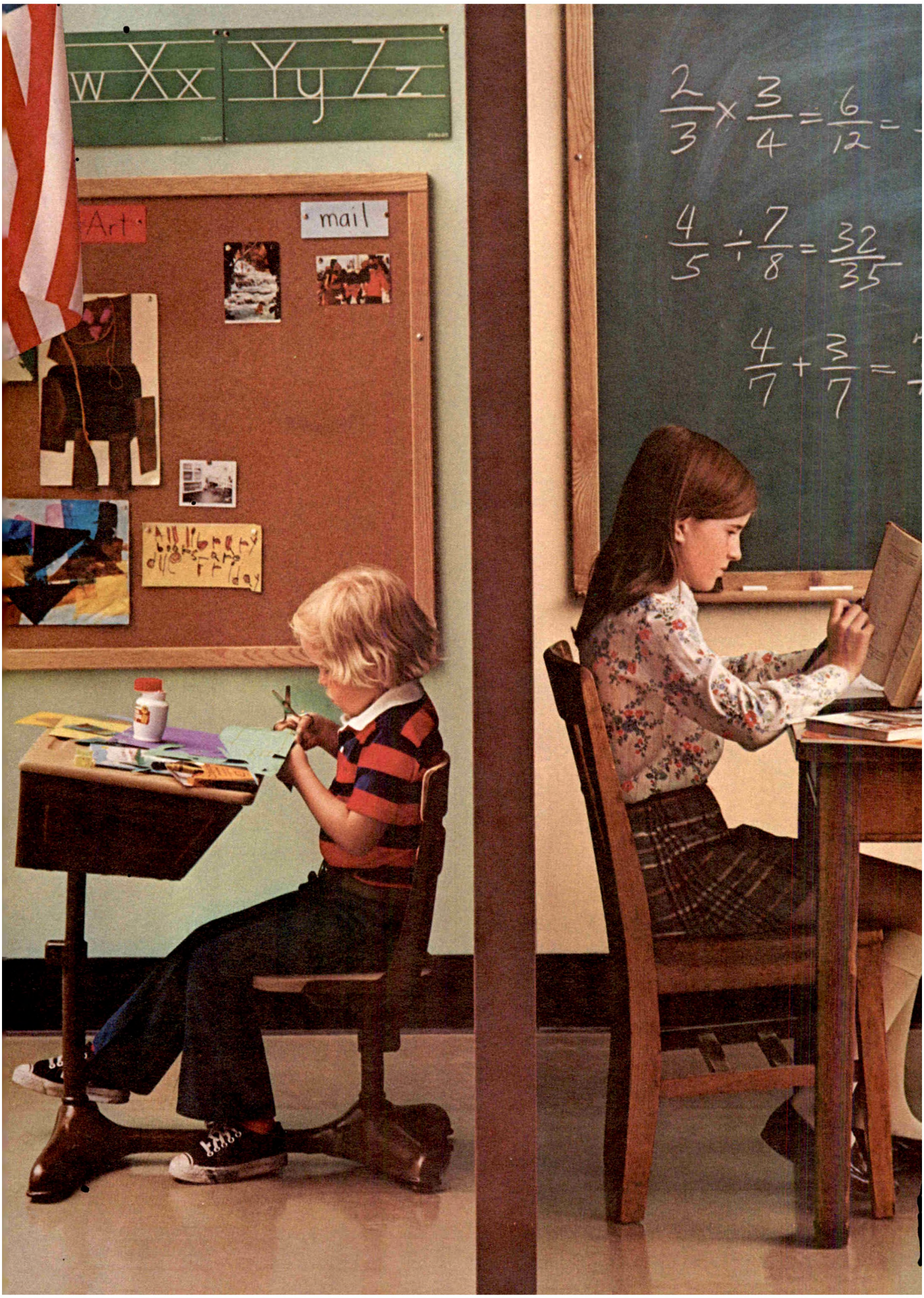
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The vulnerable ages

The first epileptic seizure is most likely to occur during early childhood and at the onset of puberty

About 9 out of 10 epileptics experience their first seizure before the age of 20—with the highest incidence between 5 and 7, when children start school, and at the onset of puberty, a time of physiological and psychic turmoil.[1] The most common type, grand mal, occurs in approximately 75% of epileptic children,[1] and more than 50% of patients who suffer initially from petit mal develop grand mal seizures before they reach the age of 16.[2]

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At the onset and afterwards — used alone or as concomitant therapy, MYSOLINE may reduce the frequency and severity of major motor seizures—or even eliminate them. *Excellent* for control of grand mal. Valuable for control of psychomotor[1,3,4] and focal epilepsy as well.[5]

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BRIEF SUMMARY
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ACTIONS: MYSOLINE acts on the central nervous system to raise seizure threshold or alter seizure pattern. The mechanism(s) of action of anticonvulsant drugs is not known.

Primidone has anticonvulsant activity *per se*. In addition, its two metabolites possess anticonvulsant qualities. The major metabolite is phenylethylmalonamide (PEMA); the other is phenobarbital. In addition to its own anticonvulsant potential, PEMA potentiates phenobarbital.

INDICATIONS: MYSOLINE, either alone or used concomitantly with other anticonvulsants, is indicated in the control of grand mal, psychomotor, and focal epileptic seizures. It may control grand mal seizures refractory to other anticonvulsant therapy.

CONTRAINDICATIONS: Primidone is contraindicated in: 1) patients with porphyria and 2) patients who are hypersensitive to phenobarbital (see ACTIONS).

WARNINGS: The abrupt withdrawal of antiepileptic medication may precipitate status epilepticus.

The therapeutic efficacy of a dosage regimen takes several days before it can be assessed.

Use in pregnancy: Recent reports strongly suggest an association between the use of anticonvulsant drugs by women with epilepsy and an elevated incidence of birth defects in children born to these women. Reference has been made to primidone in several cases in which it was used *in combination* with other anticonvulsants; but its teratogenicity has not been conclusively demonstrated. The possibility exists that other factors, *e.g.*, genetic factors or the epileptic condition, may contribute to the higher incidence of birth defects. The data also indicate that the great majority of mothers receiving anticonvulsant medication deliver normal infants.

Anticonvulsant drugs should not be discontinued in patients in whom the drug is administered to prevent major seizures because of the strong possibility of precipitating status epilepticus with attendant hypoxia and risk to both mother and the unborn child.

When the nature, frequency, and severity of the seizures do not pose a clear threat to the patient, good medical practice requires that the physician weigh the expected therapeutic benefit of anticonvulsant therapy against possible risk on an individual basis.

Neonatal hemorrhage, with a coagulation defect resembling vitamin K deficiency, has been described in newborns whose mothers were taking primidone and other anticonvulsants. Pregnant women under anticonvulsant therapy should receive prophylactic vitamin K_1 therapy for one month prior to, and during, delivery.

The physician should weigh all of the foregoing considerations when treating and counseling epileptic women of childbearing potential.

PRECAUTIONS: The total daily dosage should not exceed 2 Gm. Since MYSOLINE therapy generally extends over prolonged periods, a complete blood count and a sequential multiple analysis-12 (SMA-12) test should be made every six months.

In nursing mothers: There is evidence that in mothers treated with primidone, the drug appears in the milk in substantial quantities. Since tests for the presence of primidone in biological fluids are too complex to be carried out in the average clinical laboratory, it is suggested that the presence of undue somnolence and drowsiness in nursing newborns of MYSOLINE-treated mothers be taken as an indication that nursing should be discontinued.

ADVERSE REACTIONS: The most frequently occurring early side effects are ataxia and vertigo. These tend to disappear with continued therapy, or with reduction of initial dosage. Occasionally, the following have been reported: nausea, anorexia, vomiting, fatigue, hyperirritability, emotional disturbances, sexual impotency, diplopia, nystagmus, drowsiness, and morbilliform skin eruptions. Occasionally, persistent or severe side effects may necessitate withdrawal of the drug. Megaloblastic anemia may occur as a rare idiosyncrasy to MYSOLINE and to other anticonvulsants. The anemia responds to folic acid, 15 mg. daily, without necessity of discontinuir medication.

DOSAGE AND ADMINISTRATION: The avera adult dose is 0.75 to 1.5 Gm. per day. The initial dose is 250 n Increments of 250 mg. are added, usually at weekly interva to tolerance, or therapeutic effectiveness, up to daily doses n exceeding 2.0 Gm. A typical dosage schedule for the introdu tion of MYSOLINE (primidone) is as follows:

Adults and Children Over 8 Years of Age

1st Week 250 mg. daily at bedtime	*2nd Week* 250 mg. b.i.d.
3rd Week 250 mg. t.i.d.	*4th Week* 250 mg. q.i.d.

In children under 8 years of age maintenance levels are e tablished by a similar schedule, but at one-half the adult dosa It is best to begin with 125 mg., with gradual weekly increas of 125 mg. a day, to a daily total usually between 500 mg. ar 750 mg.

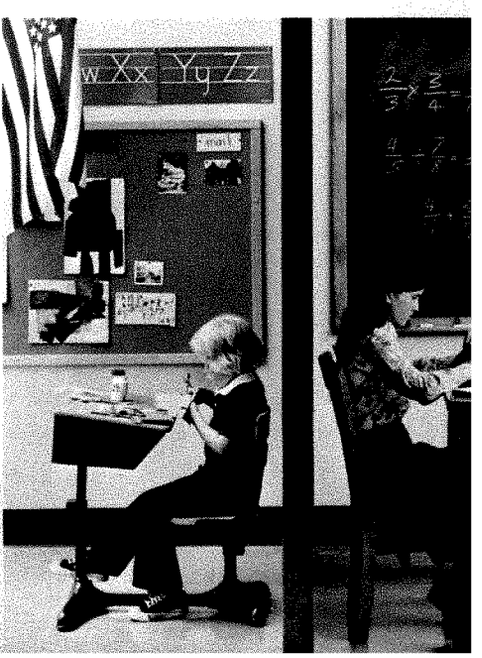
In patients already receiving other anticonvulsant MYSOLINE should be gradually increased as dosage of tl other drug(s) is maintained or gradually decreased. This re men should be continued until satisfactory dosage level achieved for combination, or the other medication is complete withdrawn. When therapy with this product alone the objective, the transition should not be completed in le than two weeks.

MYSOLINE 50 mg. Tablet can be used to practical advanta when small fractional adjustments (upward or downwar may be required, as in the following circumstances:

- for initiation of combination therapy
- during "transfer" therapy
- for added protection in periods of stress or stressful sitt tions that are likely to precipitate seizures (menstruatio allergic episodes, holidays, etc.)

HOW SUPPLIED: MYSOLINE *Tablets*—No. 430—Eac tablet contains 250 mg. of primidone (scored), in bottles 100 and 1,000. Also in unit dose package of 100. No. 431—Eac tablet contains 50 mg. of primidone (scored), in bottles of 10 and 500. MYSOLINE *Suspension*—No. 3850—Each 5 cc. (t spoonful) contains 250 mg. of primidone, in bottles of 8 flui ounces.

References: 1. Livingston, S.: Comprehensive Managemer of Epilepsy in Infancy, Childhood and Adolescence, Springfie Ill., Charles C Thomas, 1972, pp. 6, 7, 584. 2. Grossman, H.J Ill. Med. J. *135*:260 (Mar.) 1969. 3. Scholl, M.L., in Con H.E.: Current Therapy 1973, Philadelphia, Saunders, 197 pp. 675-7. 4. Metrick, S.: C.M.D. 37:49 (Jan.) 1970. 5. Forst F.M.: Med. Clin. North Am. 47:1579 (Nov.) 1970. 6. Whi P.T.: Wis. Med. J. 68:178 (Apr.) 1969. 7. Millichap, J.G Drug Ther. *1*:15 (Oct.) 1971.



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in clinically significant depression...

penetrating the symptom barrier to productive psychotherapy

symptoms often interfere with therapy

In clinically significant depression, the patient often becomes barricaded behind his own symptoms during psychotherapy. Because these symptoms can be so sharply defined and somatic in expression, he may reject the fact that they are psychic in origin. Sleep disturbance, gastrointestinal complaints, anorexia, chronic fatigue, and other psychosomatic symptoms may so preoccupy—or overwhelm—the depressed patient that truly productive therapeutic sessions are difficult to achieve. Instead of being able to concentrate on the core emotional problems of the depression itself, he may for example go on at length about how broken his sleep is or keep reiterating (while the therapeutic hour slips by) how physically miserable he feels.

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to help penetrate the symptom barrier in depression requiring medication

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well as other "barrier symptoms" may be lessened to the point where they no longer come between you and the patient.

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As these preoccupying complaints are lifted, psychotherapy sessions can become more productive. Patients are enabled to respond more fruitfully during therapeutic hours and to resume or pursue more efficiently the necessary activities in their personal lives.

meeting therapeutic goals

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ELAVIL should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks; or in children under 12. Patients with cardiovascular disorders should be watched closely. Safe use during pregnancy and lactation has not been established. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Hospitalize as soon as possible any patient suspected of having taken an overdose.

TABLETS: 10 mg, 25 mg, 50 mg, 75 mg, 100 mg, and 150 mg
INJECTION: 10 mg per ml

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Usual starting dosage



A single 100-mg tablet
Maximum starting dosage

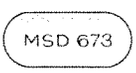


A single 50-mg tablet
Minimum starting dosage



Dosage may be increased by 25 or 50 mg as necessary until a total of 150 mg per day is reached.

A single 150-mg tablet
Maximum daily dosage



ELAVIL MAY ALSO BE PRESCRIBED IN DIVIDED DAILY DOSES

The 25-mg tablet



This strength may prove useful when therapy is initiated with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime dose.

The 10-mg tablet



This strength may prove useful for patients who require lower doses, e.g., adolescent and elderly patients. For these patients who can not tolerate higher doses, 10 mg three times a day with 20 mg at bedtime may be satisfactory.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

Contraindications: Known hypersensitivity. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.
When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice). *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

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BASIC APPROACHES TO GROUP PSYCHOTHERAPY AND GROUP COUNSELING (2nd Ed., 2nd Ptg.) *edited by* **George M. Gazda,** *Univ. of Georgia, Athens. (20 Contributors)* As with the First Edition, this Second Edition provides a convenient reference to basic theories for practitioners and students of group psychotherapy and group counseling. The amount of material covered in this new edition has increased markedly due to the growing popularity of the treatment and subsequent addition of several new theoretical positions.

Each chapter follows an outline that enables the reader to compare and contrast each position according to the following aspects: definition, theoretical foundation, related research, treatment goals, group selection and composition, group settings, group size, frequency and duration of group sessions, media employed, therapist/counselor qualifications, ethical considerations, limitations of the treatment, and suggested readings.

The fundamental psychotherapeutic positions summarized include psychodrama, psychoanalysis, existential group psychotherapy, behavioral group therapy, client-centered group therapy, reality therapy, transactional analysis, gestalt therapy, rational-emotive therapy, conjoint family therapy and group therapy with children. An eclectic position, a behavioral position and a teleoanalytic position are the three basic group counseling positions that are discussed. Each is represented by a leading proponent of the position, and references to more comprehensive sources for each theoretical model are cited.

This Second Edition also includes a revised chapter on the history and development of group psychotherapy and group counseling, a new chapter on the analysis of research and a new chapter with guidelines for ethical practice in group work.

Personnel and Guidance Journal cited this book as ". . . a readable, informative, and comprehensive collection of theoretical approaches to group work written by a distinguished group of contributors. . . . The major strength of the book lies in the fact that the writers do not simply present their theoretical positions; they push toward translating theory into practice, thereby providing a valuable resource book for students and practitioners of group work." *'77, 560 pp., 9 il., $16.75*

LEISURE COUNSELING: An Aspect of Leisure Education *edited by* **Arlin Epperson,** *Univ. of Missouri, Columbia;* **Peter A. Witt,** *Univ. of Ottawa, Ottawa, Ontario, Canada; and* **Gerald Hitzhusen,** *Univ. of Missouri, Columbia. (31 Contributors)* This book is designed to aid counselors, therapists, and park and recreation personnel in both institutional and community settings who wish to help their clients use free time more effectively. Personality and needs assessment, values clarification, and special problems of alcoholics, parolees, the mentally retarded, psychiatric patients, and retirees are examined. The focus is placed on the value of activities in achieving self-expression and self-actualization. Over 300 bibliographical references to authoritative literature in the field complement the text. *'77, 392 pp., 16 il., 22 tables, $18.00*

ACUTE GRIEF AND THE FUNERAL *edited by* **Vanderlyn R. Pine,** *State Univ. of New York, New Paltz;* **Austin H. Kutscher** *and* **David Peretz,** *both of Columbia Univ., New York City;* **Robert C. Slater,** *Univ. of Minnesota, Minneapolis;* **Robert DeBellis,** *Columbia Univ., New York City;* **Robert J. Volk,** *Burlington, Vermont; and* **Daniel J. Cherico,** *Columbia Univ., New York City. (47 Contributors)* This volume comprehensively describes the psychological and social impact of bereavement during the period of acute grief. Of particular interest are chapters on explaining death to children, the impact of social neglect on the elderly, and the roles of the psychiatrist, funeral director, educator and minister in giving counsel to the bereaved. *'76, 328 pp., 1 il., 26 tables, $16.50*

THE CLINICAL APPLICATION OF PROJECTIVE DRAWINGS (4th Ptg.) *edited by* **Emanuel F. Hammer,** *New York Univ., New York City. (14 Contributors)* A classic in this field, this book provides fundamentals of projective drawings, and goes on to considerations of differential diagnosis, appraisal of psychodynamics, conflict and defense, psychological resources as treatment potentials, and projective drawing usage in therapy. In addition to Buck's H-T-P Technique and Machover's Draw-A-Person Test, the book also includes the Draw-A-Family Procedure, Harrower's Unpleasant Concept Test, Kinget's Drawing Completion Test, and the Eight Card Redrawing Test. *'75, 688 pp., 360 il., $18.50*

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RAPE: Offenders and Their Victims (2nd Ptg.) *by* **John M. Macdonald,** *Univ. of Colorado, Denver. Chapter by* **Hunter S. Thompson.** Provided in this book is a study in depth from the viewpoint of the offender, his victim, the physician, psychiatrist, police officer, and lawyer. Why do men commit this crime? What are their methods of committing the crime? How do they select their victims? What steps can a woman take to protect herself? These and many other aspects are reviewed, including homicide and rape and the incidence of pregnancy and venereal disease following. Special attention is paid to the psychology of the offender, criminal investigation, and the problem of false accusations of rape. *'75, 360 pp. (6 3/4 x 9 3/4), 6 tables, $14.50*

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Today, direct coaching in appropriate social interaction, role playing, and encounter techniques are being used to help hyperactive children overcome disruptive social behavior patterns that often cause them to become objects of ridicule to their peers.

Ritalin, when indicated, can also play a useful role in this respect, as improving social interaction in the MBD child often involves reducing hyperactivity. Indeed, as one investigator[1] has reported, with Ritalin therapy, the MBD child can be less aggressive, less impulsive, and better able to interact with his peers.

Other studies also report improved social behavior of MBD children on Ritalin.[2,3]

A drug of choice when treatment of MBD is indicated,[4,5] controlled studies and clinical experience have demonstrated the ability of Ritalin to effectively reduce hyperactivity,[1,2] distractibility,[1,3] and disorganized behavior[1,3,6-8] in the MBD child.

And these symptomatic improvements achieved with Ritalin often result in the child being more responsive to the nonpharmacological measures of the MBD management team.[8]

Although side effects – insomnia and appetite loss – are occasionally seen with Ritalin, they are less frequent or severe than with dextroamphetamine.[4,10]

Therapy with Ritalin should be undertaken only after a medical diagnosis of MBD has been made. Dosage should be periodically interrupted. Often these interruptions reveal some "stabilization" in the child's behavior even without medication. In some MBD children they permit a reduction in dosage and eventual discontinuance of drug therapy.

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TABLETS

INDICATION

Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)

Special Diagnostic Considerations

Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.

Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS

Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS

Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy

Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS

Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS

Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION

Children with Minimal Brain Dysfunction (6 years and over).

Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.

If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.

Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED

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Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-Pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500, and 1000.

Consult complete product literature before prescribing.

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Based on a review of this drug by the National Academy of Sciences — National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

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'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

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Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudoparkinsonism, persistent tardive dyskinesia.

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Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions; peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

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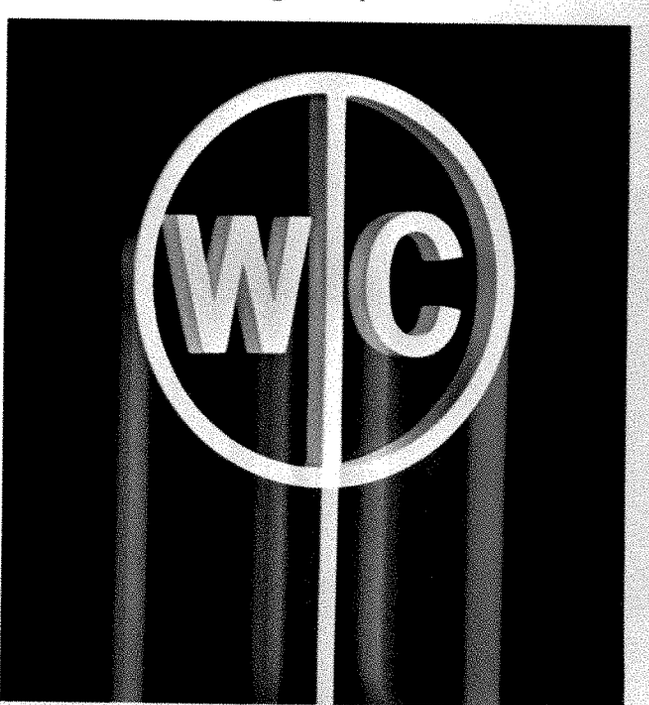
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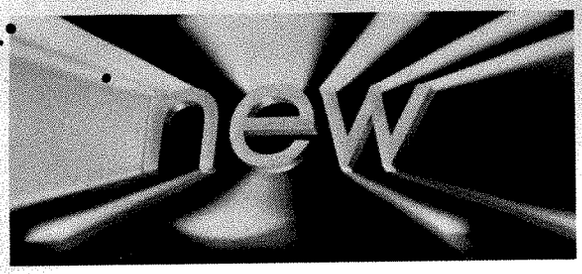
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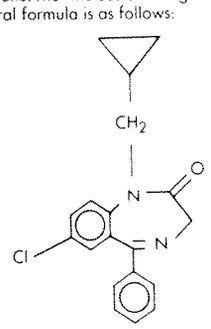
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Description: Verstran (prazepam), a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1, 4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows:



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (± 15 s.d.) hours before and 70 (± 10 s.d.) hours after multiple dosing—a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:
Use in Pregnancy and Lactation:
An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use:
Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (5.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible aberrations of liver function tests have been reported, as have slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though unlikely, may be controlled with Levophed® (levarterenol bitartrate) or Aramine® (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection, USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

How Supplied: Verstran (prazepam) 10 mg light blue, scored tablets in bottles of 100 (N 0047-0276-51).

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*Please see this page for warnings, precautions and adverse reactions.

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The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

2. THE WORKING PAPERS OF THE 1975 CONFERENCE ON EDUCATION OF PSYCHIATRISTS Paper bound, 432 pages, $12.50.

Sixty two leading psychiatric educators served on seven preparatory commissions, one for each of seven topic areas, and prepared advance working papers as a basis for Conference deliberations. The topics and Commission Chairpersons were: Social Issues and Needs (James N. Sussex, M.D.); Goals and Objectives (Morton F. Reiser, M.D.); The Resident (David R. Hawkins, M.D.); Content, Methodology, Faculty and Environment (Milton Greenblatt, M.D.); Research (Daniel X. Freedman, M.D.); Economics (Herzl Spiro, M.D.); Evaluation and Quality Control (Eric Pfeiffer, M.D.). The Chairpersons, together with the Conference Chairperson, served as the Editorial Board for this volume.

Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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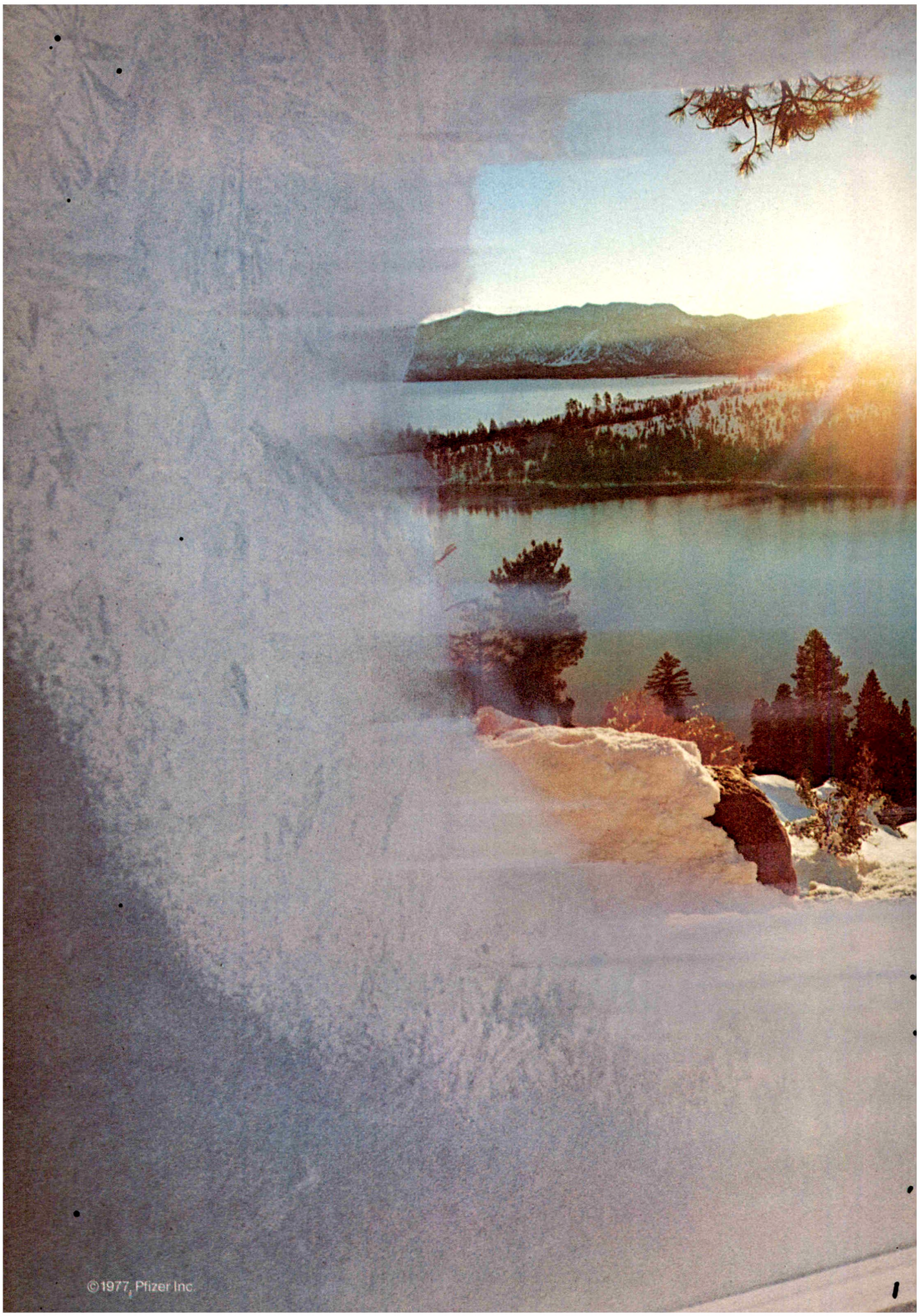
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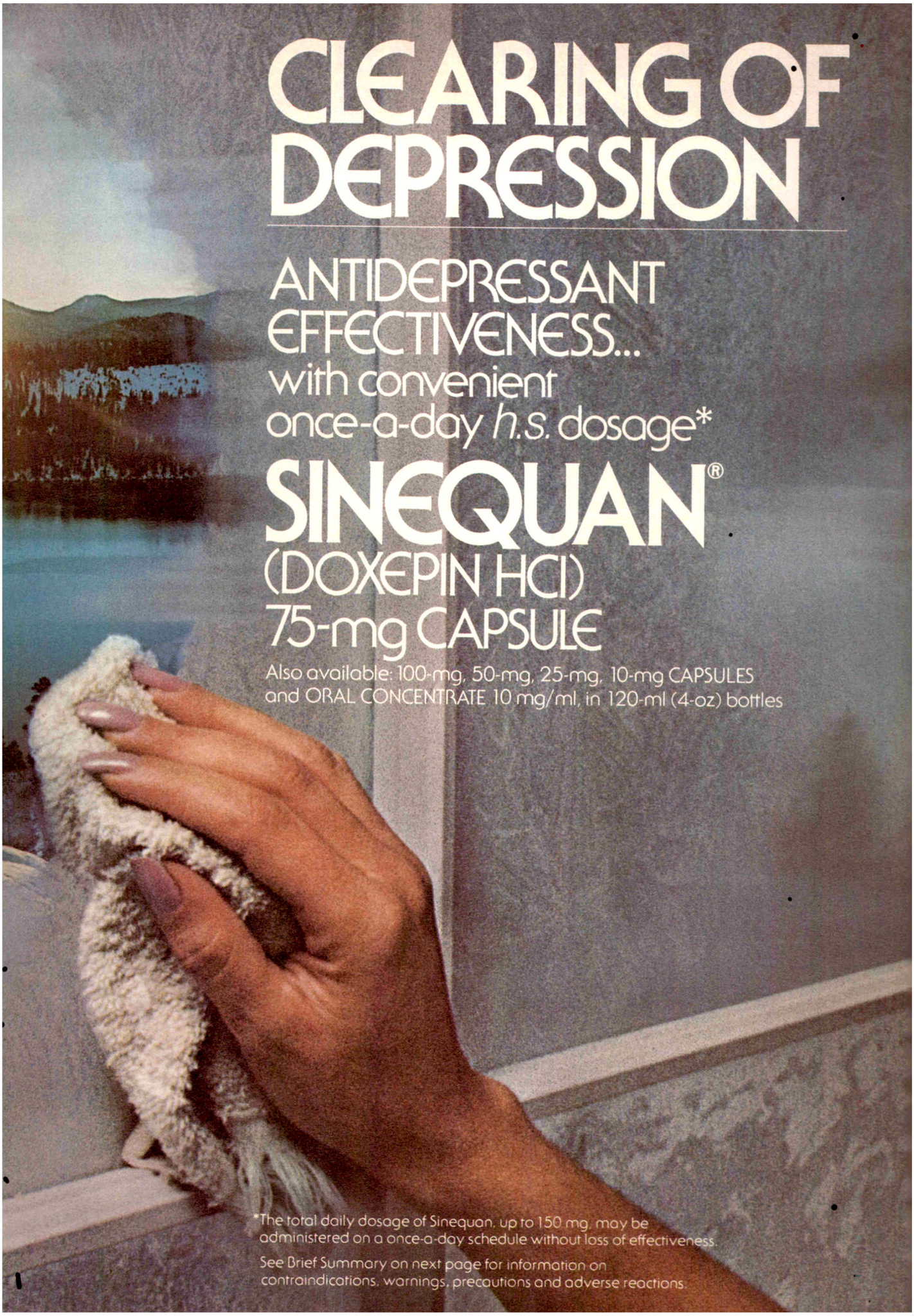
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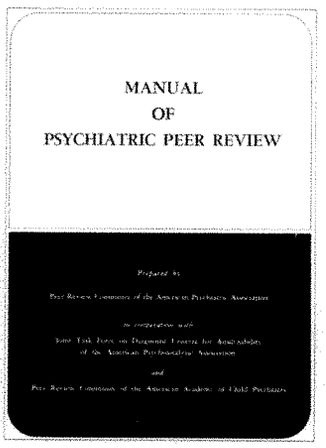
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Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.
Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.
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Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.
Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.
Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.
Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.
Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.
Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.
Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.
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Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)
Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.
Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.
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1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.
Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus) hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.
B. Management and Treatment
1. Mild: Observation and supportive therapy is all that is usually necessary.
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MANUAL OF PSYCHIATRIC PEER REVIEW

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"This Manual provides a set of guidelines for district branches of APA and their peer review committees which should be adapted and modified in terms of local criteria, standards, and practices. The guidelines are not intended to be hard and fast rules. They are screening criteria, and not regulations to control practice. They are so designed that they can be utilized by non-physicians to identify those cases that can profitably be reviewed by psychiatrists serving on peer review committees. This document should be viewed as transitional, subject to revision as experience is gained, and continually updated in response to advances in psychiatric knowledge and changes in patterns of practice. . . . The application of these standards, and refinement through experience must rest with the peer review committees of district branches. . . . It should help them to move ahead in the development of a peer review for American psychiatry."

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President

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Part II titled PSYCHOANALYTIC PEER REVIEW has sections on Steps in Peer Review for Psychoanalysis, Guidelines for Model Criteria Sets, Pilot Application of Guidelines for Model Criteria Sets, Sample Peer Review Form, Sample data from Pilot Applications of Guidelines, and Glossary of Terms Used.

Part III titled PEER REVIEW AND CHILD PSYCHIATRY contains sections of Special Considerations, Legal Rights of Children, Issue of Confidentiality, Conclusions, References, and Appendices on Model Screen Criteria Format for Intermediate Care, for Acute Care in a Short Stay Facilities, and for Partial Hospitalization, Outpatient Diagnostic Evaluation Procedures, and Outpatient Treatment for Children and Adolescents.

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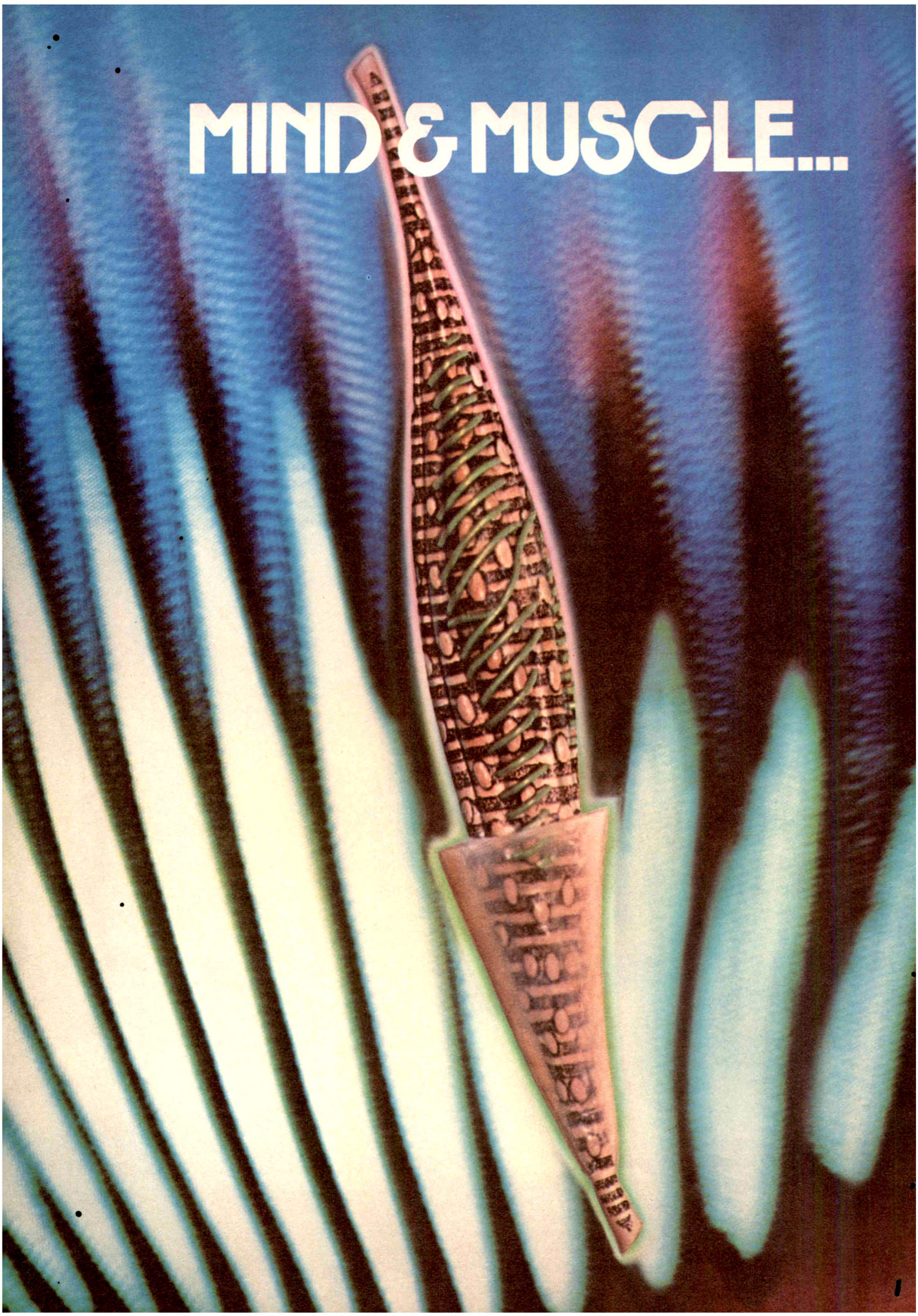
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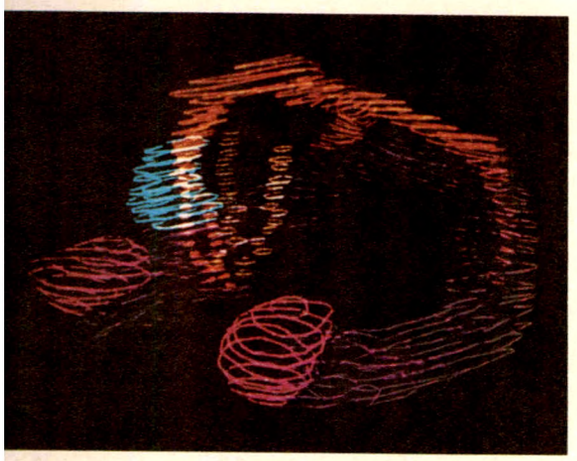
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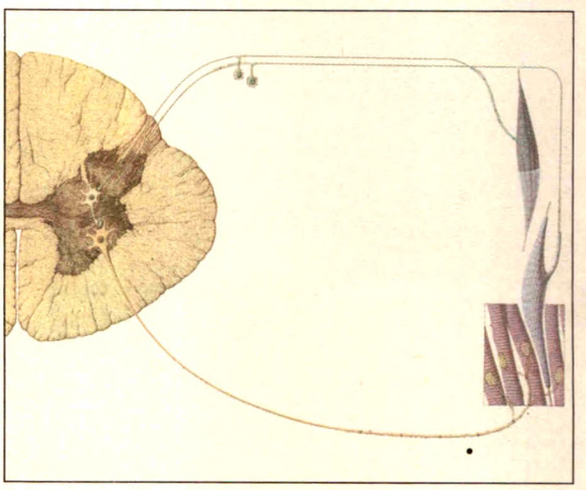
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Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

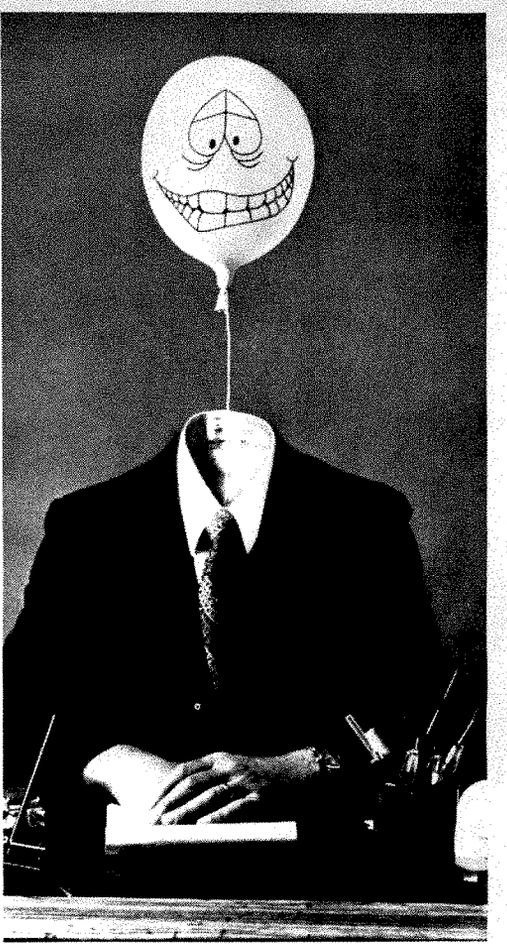
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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Contraindications: Severe central nervous system depression, comatose states from any cause, hypertensive or hypotensive heart disease of extreme degree.

Warnings: Administer cautiously to patients who have previously exhibited a hypersensitivity reaction (e.g., blood dyscrasias, jaundice) to phenothiazines. Phenothiazines are capable of potentiating central nervous system depressants (e.g., anesthetics, opiates, alcohol, etc.) as well as atropine and phosphorus insecticides; carefully consider benefit versus risk in less severe disorders. During pregnancy, administer only when the potential benefits exceed the possible risks to mother and fetus.

Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

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THE AMERICAN JOURNAL OF PSYCHIATRY

The Neurobiological Origins of Psychoanalytic Dream Theory

BY ROBERT W. MCCARLEY, M.D., AND J. ALLAN HOBSON, M.D.

Freud built his model of the mind and his hypotheses about dreaming directly on the structure of his neurobiological model of the brain, which was developed in the "Project for a Scientific Psychology," written in 1895. Among the concepts modeled in this work were ego, somatic drives as motivationally critical, cathexes of psychic energy, wish fulfillment, and primary and secondary process. From the vantage point of more than 80 years later, the authors indicate the areas in which many of Freud's neurobiological assumptions are inaccurate. Revisions are needed in the neurobiologically derived psychoanalytic concepts, especially those of Freud's wish fulfillment-disguise theory of dreams.

THE MOST celebrated theory of dreams is that of Sigmund Freud. Today Freud's idea that certain memories actually *cause* dreams is so widely accepted that it constitutes a major article of belief in American popular and literary culture, not to mention its pervasive influence within clinical psychiatry.

Less well appreciated is the extent of Freud's fascination with biology and the extent to which Freud's psychological theories, including those of "The Interpretation of Dreams" (1, 2), are the offspring of his long experience in neurobiology. In this paper we will examine the ghosts of antique neurobiology that still haunt Freud's dream theory, attempt to refurbish the neurobiology of sleep in accord with today's understanding, and then examine the consequences for psychoanalytic theory in general and dream theory in particular.

The two principal divisions of this paper will develop aspects of this theme. We shall first discuss the neurobiology of the 1890s to allow an understanding of what was known and in vogue when Freud was working on a model of the brain. We shall then show that Freud's psychology was rooted in his neurobiology, rooted deeply in an explicit model of the brain, and comment on what modern sleep neurophysiology may have to say about the accuracy of Freud's theories. A related article (3) will explore the extent to which an alternative, but still biologically oriented, theory of dream experience can be developed.

In both papers we will use the phrase "mind-body isomorphism" to describe a particular conceptualization of the relationship between body and mind, one that encompasses the most fundamental assumption of Freud, and, indeed, of many theorists of the 1890s. Freud believed that the *same* rules of description and functioning might be applied to both body and mind and that both had the same structures, the same basic operating principles, and the same rules of energy transfer, or dynamics. Whether one talked about physiological or psychological data or concepts seemed to be a matter of convenience and ease, for there were simple rules for mapping the concepts and operations of one domain onto the other.

We indicate this hypothesis of similarity by the term "isomorphism," which means literally "of the same form." It also has a quite exact usage in mathematics to indicate when there are precise rules for conversion between two logical structures or algebraic systems composed of objects and operations upon these objects (4). Although we think the precise aspects of the isomorphism concept may ultimately prove useful, this is not the place to discuss its possible direct applicability, and in this paper we will use the term in a less exact sense.

The term "isomorphism" will also serve to indicate the extent to which Freud believed that his model of

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the brain could lead to inferences about a model of the mind and vice versa. We view Freud's approach, the search for linkages between brain and mind models and data, as important and fruitful; we ourselves are concerned with such a search and use modern knowledge of neuronal and dream physiology as a basis for connections with dream psychology.

THE NEUROBIOLOGY OF THE 1890s

There are historical parallels between the great explosion of knowledge occurring in the field of neurobiology today and in the 1890s. In 1889 Santiago Ramon y Cajal made a victorious anabasis to Berlin where he presented to the leading scientists of the German-speaking world his dogma that the brain is composed of individual cells that are separated from each other and that the brain is not a reticulum or net as Golgi and others had believed (5). Cajal's exquisite materials persuaded even the mighty Kölliker, and the Germanic establishment gave the final stamp of accord in 1891 when Waldeyer spoke favorably of the "neuron theory." The study of cells and neurons was what was new and exciting, even though knowledge of pathways and functional localization was also quite advanced and certainly beyond that of most of today's first-year medical students. For example, to anticipate ourselves a bit, Freud had a certain fame as the neurologist whose powers of lesion localization were so sure that a pathological report was superfluous.

Compared with the neuroanatomy, the neurophysiology of the 1890s was not so far advanced, and we shall see that misconceptions in that area were to bedevil Freud's theory. At that time the precise role of electrical forces in nerve cells was unknown, although the knowledge of electrical changes associated with reflex conduction had made it apparent that electrical phenomena were important. It was Freud's misfortune to develop his theory of neuronal discharge and of the energy economy of the brain at this point of physiological uncertainty about the electrical and functional aspects of neurons.

A most important component of the spirit of that time was the sense of excitement. Old dogmas were falling, new edifices were being erected—in politics, the arts, biology, and physics. In biology, vitalism was being crushed by the new armamentarium of chemistry and physics. Biologists of Freud's generation were marked by two traits. First, they were loath to be mystical in any way; hard-nosed materialism—scientific method—was their credo and their weapon against the still lingering forces of vitalism. Yet they also had an audacity, an abundance of hope and faith in the new scientific method and the excitement of believing themselves on the verge of being scientifically able to describe that most difficult and elusive object of all, the human mind.

An extremely valuable source of information about the state of physiological and anatomical knowledge in Vienna in the 1890s comes from a book by Sigmund Exner. Exner was a co-worker of Freud's in the day when both were neurobiologists (6), and Exner went on to become the head of the department of physiology at the Royal University of Vienna. In scientific circles Exner was best known for his studies on reflexes and reflex conduction velocity (7, 8). Yet he also wrote the book *A Sketch of a Physiological Explanation of Psychic Events* (9). His book was published in 1894, just one year before the essence of Freud's theory of dreams was developed, and its title well conveys the audacity of that time, the soaring hope of a physiology of the mind.

Exner explained what he wanted to do in the introduction to the book, which was, appropriately for one speaking in both physiological and psychological tongues, completed on Pentecostal Sunday. With an eye on the vitalism of the past he wrote, "A path has been broken toward [the] conviction that life processes can be explained on the basis of chemical and physical processes. . . ." Looking toward the present and future and in a striking parallel to Freud's ambition, he noted that "the attempt to explain psychic events forms my assignment in life." As well as letting us see what was hoped for (the union of physiology and psychology), the book is most valuable from a documentary point of view because it allows us to scan the kind of physiological data and theory present in Vienna just before Freud's "The Interpretation of Dreams."

Exner used one of his own reflex experiments to illustrate the important concept of *Bahnung*, or, as Sherrington (8) translated this word, "facilitation." At certain stimulus intensities, neither a paw stimulus alone nor a cerebral cortical electrical stimulus alone will elicit the response of a paw twitch. However, when the two stimuli occur in close temporal contiguity a response results. The inference was that each stimulus served to facilitate the other and that a pathway (*Bahn* in German) had been formed so that the excitation from each stimulus could sum and produce a response. Freud was to make extensive use of this concept, as he did of the notion of temporal summation, or facilitation from repeated stimuli. Exner committed himself to the neuron theory and even went so far as to postulate that inhibition was an active mechanism served by its own neural pathways (9, p. 306; 10). This important but subtle concept of active inhibitory pathways is one that was not posited by many early neural theorists, including Freud (11). Exner's book may well have served as a stimulus for Freud's own attempts at a mind-body integration by combining his neurobiological training with his newly developing interest in the psyche.

FREUD'S "PROJECT"

Freud was a co-worker of Exner's at the University Physiological Institute in Vienna and is cited in Exner's work for his histological studies. Breuer, Freud's

collaborator in "Studies on Hysteria" (12), was Exner's family physician (13). Because of these relationships and interests it is quite likely that Freud knew about Exner's 1894 book, and Freud himself seems to have indicated that this was the case (14).

Freud spent most of his early career working in neurobiology. In 1876, at age 20, he began work in the University Physiological Institute. He obtained his doctorate in medicine in 1881, and in 1882, at age 26, he terminated his connection with the institute after 6 years of research work. Amacher (6) has examined the strong influence on Freud of the neurological doctrines and concepts of his teacher at the institute, Brücke, and of Meynert, Freud's teacher while Freud was a resident in the department of neurological diseases in 1884 (age 28) at Vienna General Hospital. In 1885, at age 29 and with a decade of training in neurobiology and neurology in Vienna behind him, Freud left the Vienna General Hospital and by 1886 had set up a practice as a neuropathologist. He retained an active interest in physiology, as evinced by his 1888 article in a medical encyclopedia on the brain (15) and by his 1891 volume on aphasia (16). Although he was developing interests in hypnosis, catharsis, and hysteria, he was still very much in the Viennese tradition of *Nervenartz* in 1895 when, at the age of 39, he wrote the document of particular interest to us.

This is the "Project for a Scientific Psychology" (14), or as we shall call it, the "Project." At the beginning, Freud told what he wished to do: "The intention is to furnish a psychology that shall be a natural science." Freud attempted to show the brain mechanisms of complex psychic phenomena, to develop a physiology of the mind. He occupied himself off and on with the "Project" from April 1895 to January 1896 and completed Part I, the part that is most important for our discussion, by the end of September 1895. This was a highly active period in Freud's life; he was also working out the ideas for "The Interpretation of Dreams" (1, 2), which he noted was essentially complete by the beginning of 1896. Indeed, Freud worked on both the "Project" and "The Interpretation of Dreams" during the summer of 1895 and it was at his summer house that he fancied later seeing this inscription: "In This House on July 24th, 1895, the Secret of Dreams was Revealed to Dr. Sigm. Freud." Freud described the "Irma dream" as the important catalyst, and this dream is referred to in both the "Project" and in "The Interpretation of Dreams."

There are several compelling reasons for focusing on the "Project"; the most important one is that the themes of the "Project" were the themes that would be dealt with throughout Freud's life. These ideas, derived directly from physiology, were the archaic mold for most of Freud's later theories of the mind. This is not to say that Freud's concepts were not clinically related. They were, but it must be remembered that what Freud was trying to do was to give a structure to the unconscious, by definition known only indirectly, and his ideas were strongly conditioned by what he knew from other sources, especially from his earlier work in neurobiology. This is particularly true for "The Interpretation of Dreams"; most of its theory was developed when Freud had analyzed only a *single* dream, his own "Irma dream" (1, 2, 14, 17).

Several writers have recognized and emphasized the close links between the neural model of the "Project" and the psychological model of Freud's later work. Jones, Freud's biographer, was strong in his affirmation of the "Project's" significance and documented this with a list of the 23 major topics of the "Project," 20 of which were to be discussed in detail in Freud's later writings (13). Strachey, Freud's translator and editor of the standard edition of his works in English, reported that the "Project," or rather its "invisible ghost, haunts the whole series of Freud's theoretical writings to the very end" (18). The contemporary psychoanalytic theorist, Holt, has used the "Project" as a basis of documentation of how "hidden biological assumptions . . . were retained . . . [in the] construction of an abstract psychological model" (19). Recently Pribram and Gill (20) have again emphasized that the "Project" contains, in their clearest form, the "explicit formulations and definitions of many central concepts and terms of that branch of psychoanalytic theory known as metapsychology, concepts and terms that Freud continued to use throughout his life."

For the reader whose introduction to Freud has been through the more clinically oriented papers, it is a great shock and revelation to read the "Project" since the embryos of most of Freud's major theoretical concepts are to be found here. Twenty years of concepts elaborated in clinical papers are collapsed into a few lines, and to add to the sense of wonder, they appear in a physiological paper. For the modern neurophysiologist the astonishment is of a different sort. So many of Freud's ideas about the function of neurons were simply and fundamentally wrong. We shall show that these erroneous physiological assumptions had important consequences for the formation of Freud's psychological theory.

The Elements of Freud's Model: The Neurons

In order to understand Freud's neural model it is first necessary to examine his conception of the constituent elements, the neurons, and the difficulties with many of his ideas that 80 succeeding years of neurophysiological research have made evident. The neurons in Freud's theory do appear structurally rather modern. They are discrete cells with points of contact between them, and Freud was quite correct in assuming some kind of structural specialization at the gaps between cell processes (which we call synapses). Physiology was the problem area, and Freud's principal ideas and the contrasting modern concepts about neurons can be grouped into four categories (see table 1).

1. *Neurons as serving for energy storage versus information transmission.* The modern view of neurons is that they are cells specialized for information trans-

TABLE 1
Contrasting Views of Neuronal Function

Concept	Freud's View	Modern View
Functional specialization of neuron.	Neuron as energy reservoir.	Neuron as signal element.
Mode of interaction of sensory neurons with external and somatic stimuli.	Energy conducted by neurons to the CNS.	Energy transduced by neurons.
Mode of neuronal activation.	"Passive" neurons: ultimate energy source, and stimulation to activity is always outside the CNS.	"Active" neurons: energy generated by neuronal metabolism with autochthonous regulation of neural activity.
Sign of neuronal interaction.	Excitation only.	Excitation *and* inhibition.

mission, for signaling. They use their own (metabolic) energy sources to maintain an electrical potential gradient between the outside and inside of the cell membrane (usually about −60 millivolts). A small lowering of the membrane potential sets into motion a positive feedback system leading to a "discharge" or complete depolarization of the membrane; only such "all or nothing" discharges propagating down the cell axon serve to communicate with distant cells by means of chemical transmitter release at the synapses.

The following tenets of modern electrophysiological theory contrast with Freud's ideas: 1) each cell generates its own "resting" polarization, 2) neurons influence each other by acting on this preexisting polarization, and 3) the actual amount of energy transfer between neurons needed to influence polarization and discharge frequency is quite small. These critical concepts remain valid even for other, more exotic forms of neuronal interaction, such as instances of electrical coupling and dendrodendritic synapses.

Freud, on the other hand, thought of neurons as passive energy reservoirs, able to be filled or "cathected" (*Besetzung*) with greater or lesser amounts of energy, with what he termed "nervous quantity" or *Qn*, as he abbreviated it. This stored energy, always derived from energy sources outside neurons, was passed on to other neurons in graded amounts. "Nervous quantity" is best thought of as electrical energy, although there were occasional uses of mechanical metaphors (energy of motion), as when Freud referred to neuronal "motion" or "vibration." This concept of neurons as passive reservoirs of energy derived elsewhere and of influencing other cells by passing on this energy is at variance with the modern notion of neurons as signaling elements. In electrical terms, Freud's energy dynamics would be modeled by "large signal analysis," the viewpoint appropriate to power supplies and transmission lines; the small amounts of energy involved in information transfer in the modern view suggest an analogy with "small signal analysis." Note that Freud's use of "discharge" was quite different from the modern meaning of a depolarization; for Freud it meant actual discharge of the quantity of nervous energy that was acquired, analogous to the discharge of a capacitor. It is, of course, not surprising that Freud was unable to guess the true nature of electrical forces in neurons since even the existence of the neuronal resting potential was not discovered until 1910.

2. *Neurons as conduits for energy transmission from outside into the CNS versus sensory receptor neurons as transducers*. The current neurophysiological conception of sensory receptor neurons is that they act as transducers, that is, they signal the presence of energy (e.g., light, sound, heat, and skin deformation) in the external world. They transduce this energy into a coded form of electrical signal. Similarly, from the internal world of the body, they transduce and signal information on joint angles, acceleration in space, distention of a viscus, and so forth. The important concept is that the signal indicates the presence of energy but does not conduct the energy itself into the CNS. An analogy is any physical transducer, such as a current meter, that signals the presence of current but does not itself conduct it.

In Freud's viewpoint neurons were characterized by an *actual transfer* of graded amounts of *Qn*, or nervous energy. It would be quite frightening to think of these neurons in one's periphery, for they would transmit the energy of, say, a lightning bolt directly into the CNS where this energy would have access to the whole pool of neurons. Freud used the term "excitation of neurons" (*Erregung*) to mean that they are *acquiring energy*, a notion very different from the modern concept that membrane potential is being altered in such a way as to increase the probability of discharge.

3. *Neurons as passive acceptors and donors of energy versus neurons as spontaneously active*. Freud held tenaciously to the idea of neurons as sources and sinks of energy ultimately derived from outside the brain; he on no occasion postulated that neurons had their own metabolic energy sources or that they formed self-regulatory networks. This assumption was of critical importance for Freud's neural model, and it led him to place the cause of dreaming outside the brain, since the ultimate sources of energy were also necessarily extracerebral. Dreams originated from somatic or external stimuli. This assumption further committed Freud to a reactive, essentially passive brain and to a model of the psyche that shared these characteristics. In sharply contrasting fact, the crucial biological rhythms such as respiration and, as we now know, dreaming sleep, imply the presence of endogenously regulated neural oscillations.

4. *Neurons as exclusively excitatory elements versus neurons also as inhibitory elements*. Freud's view of neurons as energy reservoirs probably blinded him to the possibility of inhibitory elements in the nervous system. We return to our analogy of modern neuronal theory as analogous to that of small signal analysis

(negligible amounts of energy) and to Freud's theory as that of large signal analysis or power generation and transmission theory. It is difficult to conceive of a neutralizing force for large signals (high energy), but easier to do so for small signals, for information. One suspects that Freud's strong histological training also may have played a role here; even Cajal never spoke of active inhibitory elements in the nervous system. It is likely that physiologists, accustomed to the obvious possibilities of active reflex inhibition, were more alert to the possibility of inhibitory elements in the CNS, as we have seen in Exner's prescient work.

For whatever reason, Freud did not include inhibitory elements in his neural model. Thus, for altering the pathways of neuronal excitation, he had only the mechanism of *diversion* of energy, or the formation of "side-paths." This diverted energy or excitation remained in the nervous system; it was immutable and persisted unless discharged in motoric activity. In contrast, modern concepts of neuronal function emphasize the possibility of *cancellation* of excitation (i.e., excitation in the modern sense of decreased membrane potential) by inhibition (through the opening of ionic channels that hyperpolarize the membrane). Freud's idea of diversion of excitation (versus the possibility of cancellation) was to have important influences on his later theoretical positions. For example, he conceived of diverted impulses or wishes (i.e., "repressed" wishes) as continually exerting a pressure toward discharge and often emerging in dreams. It is as intriguing to imagine the consequences for Freudian theory if the modern concepts of inhibition had been used as it is difficult to conceive of how modern physiologists would interpret data without them.

The Structure and Dynamics of Freud's Model

We are now ready to combine elements of Freud's model, the neurons, into the structures and paths of energy flow presented in his 1895 model of the nervous system. Figure 1 details this schema and should be consulted while reading this section; tables 2 and 3 summarize the links between Freud's neurobiology and his psychology.

In Freud's model, energy entered from the outside world, encountered a protective barrier or *Reizschutz* that prevented the full force of the energy from entering the first of Freud's systems of neurons, the ϕ or perceptual neurons. From here energy went into the main system of neurons, the ψ or psychic system. There the normal energy flow was toward the motoric system, where energy was dissipated in motoric discharge. Freud believed that primitive organisms functioned on this simple reflex model of stimulus and motoric discharge and that this model formed the basis for all of the more advanced organisms. Both neurons and organisms strived to get rid of energy. In fact, we now know that even in primitive organisms this kind of simple tension reduction or reflex model is inadequate (21).

For more complex organisms there is yet another set of neurons to be considered. Sketched above the other systems are the ω (omega) neurons, or the neurons of consciousness. The dotted lines in figure 1 indicate that these neurons have access to information from the perceptual system and also from the motor system. They have a further important function in Freud's model of sensing the level of psychic energy or *Qn* in the parts of the ψ system to which they have access. High levels of energy or charge are perceived consciously as "unpleasure," while energy discharge is perceived as pleasurable. Thus the higher organism has a strong conscious bias toward the discharge of

FIGURE 1
Freud's Model of the Nervous System

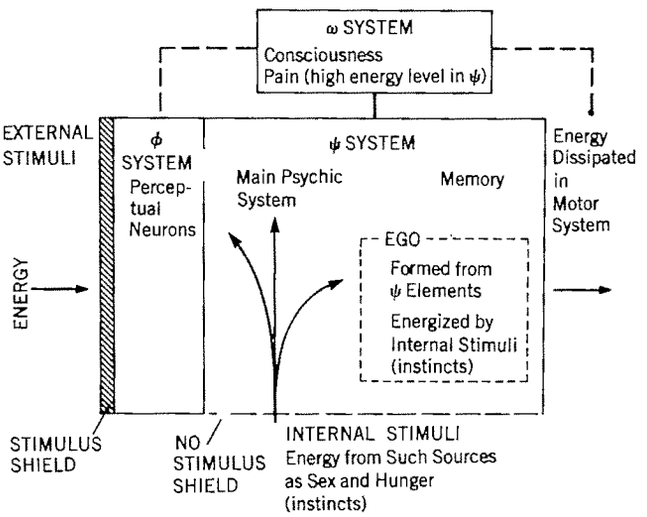


TABLE 2
Isomorphisms Between the Major Structural and Operational Concepts of Freud's Psychological Model and the Neuronal Model of the "Project"

Psychological Model	Physiological Model
Fundamental operating principle of organism is tension reduction, discharge of energy-nirvana principle.	Neurons divest themselves of nervous energy. Omega neurons sense pain as high energy level in the psychic system, pleasure as energy discharge-nirvana principle.
Primary direction of energy flow is from sensory input to motor discharge.	Direction of nervous energy flow is modeled on reflex physiology: sensory neurons → motor neurons.
Critical role of somatic drives for motivation.	Neurons receive *all* nervous energy from *outside* the brain. *Reizschutz* makes somatic input motivationally preeminent.
Lack of ego autonomy, corruptibility of ego processes by impulses.	No autonomous energy source for ego. Ego neurons derive nervous energy from somatic drives.
Chief difficulty of organism is in control of impulses, in discharging internal drives.	"Psi is at the mercy of Q." Immutability of nervous energy; no true inhibition and cancellation of nervous activation is possible. Motoric discharge is sole mode of energy release.

TABLE 3
Isomorphisms Between the Dynamic Processes of Freud's Psychological Model and the Neuronal Model of the "Project"

Psychological Process	Physiological Process
Association as principle of learning.	Reflex physiology: *Bahnungen* (pathways) between simultaneously activated neurons.
Regression in topographic sense.	Regressive direction of nervous energy flow is motor → sensory, the reverse of normal reflex flow.
Primary process thinking, hallucinatory wish fulfillment, psychoanalytic theory of psychosis (and dream imagery).	Overly strong cathexis of mnemic neurons of psychic system → hallucination. Energy is from somatic drive.
The "inhibiting ego" requires energy to divert wishes. Ego is limited in the amount of inhibition possible.	Ego neurons "inhibit" by *diverting* energy through energy requiring side-cathexes. No true inhibition or cancellation of neural activation in the modern sense.
Secondary process thinking.	Ego lessens somatic drive; overcathexis of mnemic neurons through attraction and diversion of the energy via side-cathexis.
Symptom substitution.	Immutability of nervous energy, no inhibitory elements. Blockage of energy discharge → buildup of energy in psychic neurons → tendency to emerge in another form of motoric discharge.

energy, toward what Freud called the "nirvana principle" in the "Project" and in his later works.

The importance of somatic drives—instincts—in Freud's model cannot be overemphasized. Unlike energy from the external world, there was no protective barrier or *Reizschutz* for these internal drives, and thus their full force had access to the entire psychic apparatus. Freud described the consequences in a passage that he underlined for emphasis: "ψ is at the mercy of Q (energy from somatic drives)." Thus Freud's neural model was constructed so that the somatic input of energy was motivationally critical. The multitudinous examples of the importance of the somatic drives (especially sex) in Freud's later writings hardly need emphasis. A plausible argument for why Freud selected the input from somatic drives as critical is that his model of "passive" neurons, which have no independent sources of energy, meant that the brain would of necessity have to receive energy from extracerebral sources and thus either from the external world or from the body. But, as noted in the "Project," the external world is a poor source of lasting motivational power, since painful external energy sources can usually be circumvented simply by moving away; on the other hand, internal sources of excitation, of energy, persist unless the particular drive, such as hunger or sex, is satisfied.

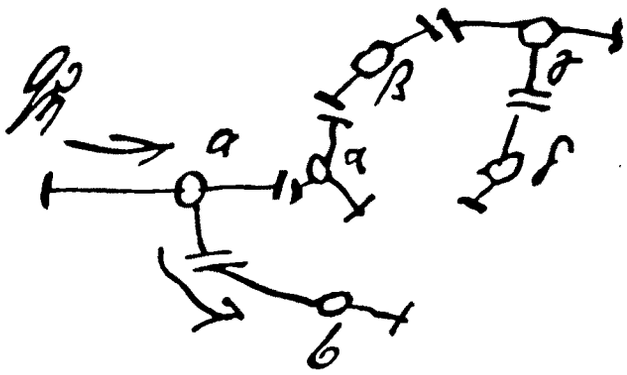
We chose the example used by Freud in the "Project," that of a hungry infant, to illustrate the dynamics of his neural model of the complex organism and the genesis of many familiar psychological concepts in the physiology of the "Project." When an infant becomes hungry, the energy from this somatic drive goes into the ψ system of neurons and the charge or cathexis is then dissipated in the motoric activity of crying. This crying acts as a signal to the mother, who provides a nipple for the infant, and the drive energy ceases with its satisfaction. Freud postulated that the neurons of the ψ system had memory, that the ease of passage across the "contact barriers" (Freud's term) or synapses was altered according to the frequency and amount of *Qn* or psychic energy passing through them. He further used the concepts of reflex physiology transposed to his neurons to postulate that "hot spots" of neuronal activity occurring simultaneously would be linked, that paths of facilitation (*Bahnungen*) would be formed and would remain, because of the memory of ψ neurons. Thus, in this example, the neurons storing the perception of the nipple and those cathected by the somatic energy from hunger would be linked. Freud put the locus of mnemic elements (or memory neurons) near the perceptual end of the psychic apparatus. The next time the infant becomes hungry energy flows from the soma, but because of the *Bahnung* it will now travel toward the mnemic neurons containing the perception of the nipple. These neurons will be very strongly cathected; this overly strong cathexis produces a hallucination rather than a memory.

At this point Freud introduced the term "regression," indicating that the flow of energy is "regressive" toward the perceptual side of the CNS and not the "progressive" and usual path toward motoric discharge. Here also is the origin of the concept of hallucinatory wish fulfillment and the basis of primary process thinking. As will be seen, regressive process also underlies Freud's theory of hallucinations in dreams and further forms the basis for the theory of psychotic hallucinations.

For the infant in the example, mere hallucination of the nipple cannot provide much satisfaction. Freud, not completely in jest, noted that one would have to be schizophrenic to cling to such a hallucination. But the difficulty in terms of the theory is how this neural modeler of the 1890s altered the course of energy away from the overcathected mnemic elements responsible for the unsatisfactory hallucination without having any inhibitory elements in his model of the nervous system.

Freud, in fact, was driven to postulate another principle of neuronal energy flow, one for which there was not (and is not) any experimental evidence. Figure 2 presents Freud's sketch of the normal direction of energy flow in a set of neurons. Freud attempted to deal with this dilemma by positing that a charge or cathexis in a postsynaptic neuron will attract *Qn* to it and thus can divert nervous energy from its normal path. Freud spoke of this diversion of energy as "inhibition," but his concept was distinctly different from the modern concept of inhibition and rested on inaccurate assumptions about neuronal functioning. The chief con-

FIGURE 2
Freud's Sketch of Neurons and the Flow of Neural Energy*



*This sketch illustrates Freud's concept of diversion of neural energy through a "side-cathexis." The normal flow of energy (arrow labeled *Qn* in Freud's script) is to neuron b; Freud postulated that a side-cathexis of neuron alpha would attract *Qn* and divert the flow from neuron b. Freud believed this postsynaptic attraction of energy or side-cathexis (for which there is no experimental support) to be the neuronal mechanism underlying repression.

sequence of Freud's concept of side-cathexis for his theory of the operation of the nervous system is that nervous energy can only be diverted, not cancelled, and it further increased the vulnerability of his model nervous system to an overload of energy.

In the example of the hungry infant, Freud said that the flow of energy is diverted away from the mnemic neurons by the use of such a side-cathexis. The lesser intensity of the cathexis of the mnemic elements produces a memory, not a hallucination, and the infant is able to compare this mnemic image with its perception—an example of *secondary process thinking*.

Freud believed that the "inhibitory" side-cathexes of this nature were made by a specialized set of neurons of the ψ system; he called this subsystem the "ego." This familiar psychological construct and even the psychoanalytically familiar phrase, "the inhibiting ego," had its origin at this point in the "Project."

A final comment on the dynamics of the neuronal model of the "Project" concerns the origin of the terms "repression" and "primary defense." Freud thought that perceptions of objects that had proved painful in the past were associated with a great deal of painful affect (i.e., excessive energy) because the perceptions were associatively linked to "key neurons" whose secretions (i.e., hormone-like activity) stimulated endogenous somatic sources of *Qn* and thus unpleasure. In fact, figure 2 represents such a neuron (labeled "b"), the "inhibition" of the course of energy toward the key neuron b is called primary defense, and the exact same mechanism of diversion of neuronal energy flow through ego side-cathexes is termed "repression" in the discussion of hysteria in the "Project."

Thus a recognizably Freudian theoretical system with its corpus of familiar clinical concepts is now evident in the *neural* model (see outline in tables 2 and 3). In fact, the entire psychic model and almost the entire theoretical structure of "The Interpretation of Dreams" and, indeed, much of Freud's later work is now comprehensible, since it is directly derived from the neurophysiological brain model of the "Project."

Overview of Freud's "Project"

This overview of Freud's "Project" will allow a discussion of the principal characteristics and difficulties of an organism constructed according to Freud's 80-year-old specifications. The most salient difficulty and unreality in Freud's model is an energy economy based on a demonstrably false conception of neurons and synapses. Using this false conception as a basis, Freud modeled an organism whose primary principle and goal is tension reduction, whose conscious elements register unpleasure when the energy level is too great, and who is subject to unshielded bombardment from internal drive energies—all three dubious assumptions.

Furthermore, the organizing, executive neurons (the ego) themselves do not have the possibility of direct inhibition of drives—as would be possible if this were a "small signal" instead of a "large signal nervous system" and if Freud had conceived of inhibitory elements. Energy can only be diverted or stored, but it retains its power, forever a threat to the organism's comfort, even though restrained for the moment.

We think it fair to say that one would expect the organism of the "Project" to be dominated by drives and to be in constant danger of being overwhelmed by them. It is not too great a caricature to call the "Project" a "cat on a hot tin roof" model, with the persistent internal drives generating blasts of energy that keep the ego and conscious system in frenzied movement.

It should be further noted that, since energy from drives can be diverted but not cancelled or discharged internally, suppression of one form of motoric dissipation inevitably leads to an internal energy buildup with the likelihood of flow into another motoric act. This is the primitive analogue of the doctrine of *symptom substitution* and is directly based on Freud's mistaken concepts of the immutability of nervous energy or excitation and the absence of inhibitory nervous elements.

The absence of self-generated activity and energy postulated by Freud's model for neuronal systems leads directly to the notion of lack of autonomy for psychological systems. The source of the postulate of ego dependence on drive energy originates in the physiology of the "Project," and one legacy of this inaccurate physiological model was the necessity of a long, difficult struggle by psychoanalytic ego psychologists to convince conservative analytic theorists that the ego should be regarded as autonomous (22, especially chapter 9).

It must be emphasized that there are also positive features for later psychological theory in the dynamism of Freud's CNS model. It is fluid in its postulates of facilitation and already embodies a rather specific, although ancient, learning theory: psychological asso-

ciation through temporal contiguity, represented neurally by facilitation between simultaneously occurring "hot spots" of neural activity. There is, further, the inclusion of the specific postulates of classical conditioning and a neuronal model of the mechanisms. This neuronal theory of learning provides a possible bridge to the concepts of what is today called learning theory. This link has not been sufficiently recognized and thus not used; even one of the classical works on conditioning and learning (23) fails to point out the common parenthood with psychoanalysis.

A few comments should be made about Freud's intellectual style and strategy as seen in the "Project" and in his letters about the "Project" (17). Because there was no final draft or publication of the "Project," the alternative and tentative approaches of Freud remain. They testify to his devotion to modeling and to theory and to his enchantment with logical coherence. He was elated when his model appeared to work and despaired when it ran into difficulty. He abandoned further formal work on the "Project" when, even after several revisions, he could not get his notion of the omega system (or consciousness system) to mesh satisfactorily with the other elements. However, what Freud kept was this notion of the basic principles of how the brain and mind work.

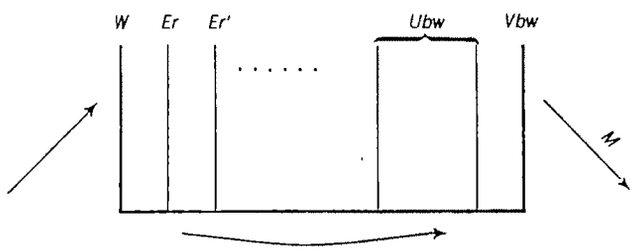
Amacher (24), in commenting recently on the extent of Freud's devotion to theory, emphasized that Freud's first impulse in cases of difficulty with empirical data was always to "save the theory," whether it was his theory of dreams or his theory of sexuality. This characteristic of Freud makes it understandable why the theories of the "Project" are of such critical importance for an understanding of Freud's later work. Examination of "The Interpretation of Dreams," the work Freud valued most highly, reveals many specific examples of theoretical similarities, its isomorphism with the "Project."

Freud's Dream Model

The seventh and most theoretical chapter of "The Interpretation of Dreams" (1, 2) outlines what Freud described as a psychological model of the psychic apparatus (see figure 3). However, it is in fact virtually the same model as that set out for the brain in the "Project" and should be compared with figure 2. Table 4 also summarizes some of the links between dream processes and the neuronal mechanisms of the "Project."

This "psychological model" of figure 3 has the same perceptual system as the "Project," although the abbreviation ϕ is not used. As in the "Project," excitation flows into a familiar second system. The psychic system of "The Interpretation of Dreams" includes three subsystems: mnemic elements; the unconscious, to be thought of as those psychic elements in contact with instincts and not open to the conscious system (the consciousness elements are not included in Freud's sketch); and, finally, the preconscious, composed of those psychic elements in close apposition to consciousness.

FIGURE 3
Freud's Model of the Psyche*



*This model appears in the seventh chapter of "The Interpretation of Dreams." W is the German abbreviation for the perceptual system. The constituent elements of the main or psychic system (not labeled as such in the original figure) are as follows: Er, memory; Ubw, unconscious; and Vbw, preconscious. M indicates the motoric system. The arrows indicate the normal direction of energy flow. Although Freud did not include as much detail in this drawing, the full model as outlined in his text is virtually identical to the brain model of the "Project" that is sketched in figure 1.

TABLE 4
Isomorphisms Between Freud's Dream Theory and the Neuronal Model of the "Project"

Dream Process	Neuronal Process
Sleep paralysis.	Withdrawal of ego cathexis from motor apparatus.
"Power supply" of dream is a wish, derived from somatic drive.	All neuronal energy comes from outside the brain; somatic energy has no stimulus barrier.
Dream process is a reaction to events *outside* the CNS, i.e., the combination of day residue with instinctual wish.	No autochthonous energy source or regulatory power for brain neurons. They are passive recipients of energy.
Regression of psychic energy after censor blocks access to consciousness.	Flow of nervous energy is toward perceptual end of apparatus.
Dream work, dream hallucinations. Dream guards sleep by disguising wish.	Overcathexis of mnemic neurons near sensory side of apparatus → hallucinations. *Bahnungen* (pathways) between wish and mnemic elements related to wish furnish the disguised representation of the wish.

Freud did not include the "psychic censor" in his sketch, but his text description of psychic topography placed its elements between the unconscious and preconscious; the psychic censor acts to screen and block wishes unacceptable to consciousness. The psychic censor is conceptually derived from the set of neurons in the "Project" that were responsible for repression. This subject will be discussed in more detail in a later section of this paper.

The outline of Freud's dream theory is straightforward. The ego wishes to sleep (the causal basis for this wish is not clear); it withdraws its cathexes from the motor system, resulting in sleep paralysis. The dream process begins when something in the day's ex-

perience stirs up a repressed wish in the unconscious. They pair forces and seek to move in the usual direction of flow in Freud's model (see arrow in figure 3) toward the preconscious system. Entry of the undisguised wish is blocked by the censor, and there is a regressive movement of the "currents of excitation" toward the mnemic elements of the psyche, which, as in the model of the "Project," are close to the perceptual side of the psychic apparatus. There the "dream's work" of condensation, displacement, and symbol formation takes place with a disguise of the wish by the imagery of those mnemic elements with the strongest associative links to the wish. The disguised wish thus becomes acceptable to the censor and is passed into consciousness. Freud believed that the dream functioned as a guardian of sleep by preventing the intrusion of undisguised and unacceptable wishes into the conscious system, with subsequent arousal.

As for dream phenomena, Freud concentrated on the symbolic grammar employed and focused his interpretive powers on unraveling the psychic meaning of symbols and the thematic material of dreams. He paid relatively little attention in "The Interpretation of Dreams" to certain formal sensory, temporal, and intensity attributes of the dream, but he did note that the psychic or subjective intensity of a dream image reflects the underlying intensity of the wish (its charge) and the number of excitations condensed into a single symbol or wish. He suggested that the frequent subjective sense in dreams of little or no movement was related to the sensing of sleep paralysis. Most importantly, Freud asserted that the forgetting of dreams is "inexplicable unless the power of the psychic censorship is taken into account," i.e., we forget our dreams because of psychic repression. Freud saw in the forgetting of dreams the prime support for his "wish fulfillment-disguise" theory of dream generation.

Thus Freud's psychic model was quite simply derived from the physiological constructs of the "Project." The psychic and neural agencies are virtually the same, their organizational sequence and function are the same, the direction of flow of energy is the same, and the reflex model is retained. The psychic "elements" share the properties of the "Project's" "neurons" in that "excitation" is "transmitted" according to "conductive resistance." "Association" results from "facilitating paths." Freud even talked about "currents of excitation." In short, virtually the entire neural theory of the "Project" was retained despite Freud's assertion of creating a psychological model.

MODERN PHYSIOLOGY AND REVISIONS IN FREUD'S THEORY

Implications for Psychoanalytic Dream Theory

We have discussed in detail how Freud's theory, his model of the psyche and of dream processes, is based on his neuronal model of the "Project." We will now briefly address the revisions in Freud's theory that are necessitated by the 80 years of neurophysiological research since the "Project." Our related article (3) will present the results of modern sleep physiology on which these conclusions rest.

Freud believed that the dreaming sleep state (D) and dreaming were initiated and powered by the combination of the day residue (certain memories from the day) with the energy contained in a repressed, unconscious wish (see table 4, item 3). It can now be categorically stated that there is *no* experimental support whatsoever for Freud's theory of D-state generation. Instead, modern investigations point to autochthonous, periodic, and motivationally neutral activation of pontine generator neurons as the cause of the D state (25–27).

The rhythmic state-generation property of these pontine brain stem cells points to other fundamental difficulties with Freud's CNS model, since he nowhere postulated autochthonously active and autochthonously regulated neural systems or their counterpart. The simple conclusion is that Freud's theory regarding the lack of autochthonous activity and the lack of regulation and endogenous energy for the brain must be abandoned. This is not to suggest that day residue material or motivationally important themes do not enter into dream content; they may do so, but neither is a causal factor in the dream process.

There is no need to postulate the repression of *Qn* (or of wishes, if one prefers this label for neural energy) to provide generative power for the dream state. The energy is already *in* the CNS. For D generation there is also no need to postulate the existence of the unconscious as the psychic subsystem or collection of neurons necessary for the storage of the energy of repressed wishes, and there is nothing to suggest that the concept of repression is in any way germane to controlling the activity of the pontine cells responsible for D generation. Intrapontine, autochthonously proceeding, and active cellular mechanisms are all that is required. Further, the concept of regression in the sense of a reversed flow of neural energy is not needed or even correct. The pontine executive cells conduct in the same direction that they do during waking; they just become some 40-fold more active in the D state.

There is also serious difficulty with Freud's theory that the primary motivating force for the dream language and dream plot is disguise of a repressed wish. The driving force for D sleep is a biologically determined and *motivationally neutral* activation of cells in the pons, not a repressed wish. There is no evidence whatsoever that these cellular mechanisms of generation are in any way driven by hunger, sex, or any other instinct or by repressed wishes for consummation of instinctual drives. The *primary motive* for the dream language and dream process cannot be disguise if the prime force of dreams is not an instinct or repressed "wish" in need of disguise. Our related article (3) will discuss alternative mechanisms for some aspects of dream phenomenology.

Implications for Psychoanalytic Theory in General

It is beyond the scope of this paper to present a detailed discussion of the implications of the inaccuracies in Freud's neural model for general psychoanalytic theory, as opposed to dream theory. It does appear worthwhile, however, to use what had been discussed as a basis for looking at responses to the problems in Freud's theory and the merits of various strategies for dealing with them.

There have been two major reactions to the inaccuracies in Freud's model. The first of these is seen largely in clinical settings. Those engaged in clinical work may acknowledge the theoretical difficulties with many of Freud's concepts but continue to use them in their conceptualizations of patients simply because there is no equally comprehensive alternative theory. Thus at the clinical level the use of concepts such as impulse breakthrough, symptom substitution, object cathexis, and instinctual energy remains common, even though the user may have some intellectual misgivings.

The second kind of response is to attempt to deal with the problems associated with a certain specified and apparently delimited portion of Freud's model, usually to what is termed "energetics" or the "energy economy." One then suggests the substitution of some more appropriate concept, i.e., one more in harmony with modern neurobiology. The often unappreciated difficulty is that what is labeled as a simple problem in "economics" is, in fact, a problem with fundamental postulates and thus the *entirety* of Freud's model. Freud's model, like complicated models in general, cannot be easily revised by the simple substitution of a single concept. The alteration of one of the fundamental premises has profound effects on the model as a whole, and it is further impossible to segregate aspects of Freud's model into neat, easily differentiable sections that can be dealt with in isolation.

An examination of the concept of repression illustrates some of the complexity of revision. This concept originated in the neuronal model of the "Project." Freud noted that hysterical repression is based on the mechanisms of diversion of nervous energy (*Qn*). "Thus there is a defensive process emanating from the cathected ego which results in hysterical repression . . ." (14, p. 351). The basic mechanisms and concepts are the same as those we have discussed for primary defense: the use of side-cathexes, release of unpleasure by key neurons, and quantitative aspects of the energy economy. "The stronger the release of unpleasure the harder was the task for the ego, which, with its side cathexes, can after all provide a counterweight to the *Qn* up to a certain limit . . ." (14, p. 358).

Can this concept of the mechanisms of repression originating in the mechanisms of the "Project" be easily revised by a simple updating of the "neuronal energetics"? One must conclude that it cannot, since it rests on several incorrect assumptions of Freud's model: not only on his erroneous theory of energetics, but also on the absence of inhibitory elements, the absence of the possibility of cancellation of excitation through inhibition, and a mistaken notion of synaptic transmission (side-cathexes do not "attract" and bind neuronal energy). It is apparent that to update the concept of repression one would have to alter several assumptions of the model. Thus this example of a clinically central concept supports the position that one cannot revise by, for example, simple substitution of "neuronal discharge" wherever Freud talks about *Qn* or nervous energy.

Still another reaction to the inaccuracies in Freud's model has been to ignore the erroneous neurobiology and attempt to create more abstract and purely psychological models, such as Breger and associates (28, 29) sought to do for dream theory. In their discussion of the "Project," Pribram and Gill treated Freud's discussion of neuronal physiology and mechanisms as a metaphor for psychological processes, "something that should . . . become part of the heritage of academic cognitive psychology" (20, p. 11). We believe that this approach runs into serious difficulty because such abstract models are even more difficult to specify in detail than the original and thus even more difficult to test (see a more complete discussion of this topic in reference 10).

In contrast to these reactions, we take a critical historical approach that attempts to acknowledge, identify, and correct the erroneous neurobiological assumptions. We submit that these assumptions cannot be denied, but they *do* critically determine psychoanalytic psychology, and that their adequate revision represents an important source of strength for dream psychology. The succeeding paper on the activation-synthesis model of dream phenomena will continue in the tradition of seeking mind-body isomorphisms but will recognize both the value and necessity of limited approaches consonant with the current level of development of neurobiology.

SUMMARY AND CONCLUSIONS

Freud built his model of the mind and his hypotheses about dreaming, which appear in "The Interpretation of Dreams," directly on the structure of his neurobiological model of the brain as developed in his "Project for a Scientific Psychology." Freud developed the theory of both models during the same year, and both are closely related to his work as a student and research worker in neurobiology and to the theories current in the neurobiology of the 1890s. The neurobiological foundations of "The Interpretation of Dreams" and the extent of the isomorphism between Freud's model of the brain and of the mind are generally unrecognized, as is the need to modify those portions of Freud's dream theory and general psychology that are based on inaccurate neurobiology.

Because he developed the theory for both models in 1895, Freud could not have known of modern evidence

that neurons are elements of a signaling network, that neurons have their own metabolic sources of energy and influence one another by the transmission of *small* amounts of energy. Freud conceived of neurons as if they were passive elements in a power transmission network; he believed that all neural energy was entirely derived from outside the brain, chiefly from somatic sources, from instincts. Neurons acted as passive conduits and storage vessels for this energy. Freud did not conceive of the possibility of inhibitory neurons that could *cancel* excitatory signals; in his model energy could only be *diverted* or repressed. Motoric discharge was the sole method of energy dissipation. This neural model was one that left the psychic system, in Freud's words, "at the mercy" of somatic drives. The "Project" and its neural model are of great importance because most of Freud's theoretical concepts were developed there, including those of the ego, nirvana principle, primary and secondary process, hallucinatory wish fulfillment, the inhibiting ego, repression, displacement, and regression.

Examination of Freud's theory of dreams and D-sleep generation (for which Freud assumed the same mechanisms) shows the problems associated with theory developed from the inaccurate neurobiological assumptions of the "Project." In accordance with the assumption that energy comes only from outside the brain, Freud postulated that the "power" for generation of D sleep arises from a repressed instinctual wish, from diverted instinctual energy that becomes linked to a recent memory (day residue). Freud's model assumed a nervous system that reacted to external and somatic stimuli, not one with independently active neural regulation.

Modern investigations have established that the generators for D sleep are motivationally neutral and consist of actively, autochthonously regulated oscillations of neural activity in the pontine brain stem, in contrast to Freud's theory of passive neural elements. Freud believed that the characteristic phenomenology of dreams was the product of an attempt to disguise the instinctually based wish that generated the dream. Since D-sleep generation results from the motivationally neutral activation of pontine executive cells, disguise cannot be the primary motive for the characteristic dream phenomena. Our related article (3) will build on modern knowledge of sleep physiology in proposing an activation-synthesis model of some elements of the dream experience.

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Cost, Utilization, and Utilization Review of Mental Health Services in a Prepaid Group Practice Plan

BY SIDNEY S. GOLDENSOHN, M.D.

The author describes the utilization review process, utilization patterns, and service cost of the Mental Health Service of the Health Insurance Plan of Greater New York (HIP). He finds that a mental health care delivery system within a health maintenance organization offers the advantages of sophisticated utilization review procedures, reduced cost per mental health incident, and a low utilization rate and low cost for psychiatric hospitalization. However, the HMO's return-to-function treatment goals may be too limited for the minority of patients who would benefit only from long-term intensive treatment. He recommends that cost accounting be based on cost per illness rather than cost per service.

IN RECENT YEARS the public, legislators, and mental health professionals have shown a growing concern that mental health care be of high quality and be provided at reasonable cost. A critical challenge is thus posed: how do we improve the effectiveness of mental health care and at the same time control or reduce its cost? The delivery of comprehensive mental health care as part of a health maintenance organization (HMO) has several attractive accountability features: 1) reasonable cost per illness for outpatient mental health care due to its "return-to-function" treatment goals (these goals are compatible with an enrolled population that is largely lower-middle to middle class or blue collar); 2) low cost for inpatient mental health care due to a low rate of hospital utilization; and 3) availability of utilization review mechanisms to help achieve quality of care. A regrettably limiting feature in the search for quality care in the HMO system is that its return-to-function therapeutic goals may not be optimal for the nearly 1 in 4 psychiatric patients who come from well-educated and middle- to upper-middle-class populations.

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In this paper I will present my experience with accountability based on my work with the Mental Health Service of the Health Insurance Plan of Greater New York (HIP). I will describe this program's utilization review process, utilization patterns, and cost of services.

Relevant aspects of HIP's Mental Health Service will be reviewed briefly; more complete descriptions have been reported elsewhere (1–4). Mental health services are available to HIP subscribers on the basis of a rider to the prepaid health insurance plan. Approximately 180,000 enrollees have purchased the psychiatric rider. The premium (for the time period covered in this paper, January–December 1975) was $10.80 per person per year, $21.60 per couple, or $32.40 for a family of 3 or more. When adjusted for large families, the cost came to $8.76 per person. Psychiatric coverage is provided without coinsurance or deductible features. Premiums are paid to the plan and are used entirely for prenegotiated mental health services. This financing mechanism contrasts with fee-for-service insurance, in which a subscriber is indemnified for expenses incurred in obtaining mental health services in the open market.

The Mental Health Service of HIP offers a broad spectrum of mental health services. The basic therapeutic orientation is psychodynamic; the goal of treatment is to return the patient to a functioning level as promptly as possible. The usual in-hospital treatment modalities are provided for the maximum period of time covered by the patient's Blue Cross contract. Psychotropic drugs are supplied without additional cost to the patient both in and out of the hospital. A ratio of 12 full-time professionals per 60,000 enrollees is necessary to provide mental health services in HIP's three regional centers.

UTILIZATION REVIEW

Utilization review is an important tool in achieving both quality assurance and cost containment. A procedure designed to assure that all services given are necessary, it is used to analyze and identify factors that may contribute to unnecessary or ineffective utilization; potentially it might lead to the development of more precise standards of diagnosis and treatment through statistical analyses (5). Utilization review includes review of individual cases as well as review of

program-wide patterns of utilization. Program-wide review is based on the assumption that an important dimension of quality care is the degree to which mental health services are reaching populations at risk.

Utilization Review of Individual Cases

All cases are reviewed after a 6-month period of relatively uninterrupted weekly therapy. This review can uncover stalemated, nonproductive patient-therapist relationships. When problem cases are routinely discussed, the continuation of treatment without evidence of progress is less likely to occur unchallenged (6). In keeping with our treatment goals, one question that is always asked in the case review is, Has the patient or family already returned to a functioning level of performance? Case review may help the therapist terminate treatment when these goals have been achieved; help the therapist terminate treatment because no further change (within our system) can be anticipated; help the therapist overcome a clinical impasse; and, finally, provide agreement with and support for the therapist's current management of treatment.

Program-Wide Utilization Review

In 1975 the Mental Health Service's overall annual utilization rate for new psychiatric consultations was 11.0 per 1,000 population; 18.3 for the Medicaid population; 11.7 for both state and federal employees; and 5.8 for Medicare enrollees. The number of therapeutic sessions provided to each subscriber group per 1,000 population followed a similar pattern (see table 1). Data are also available on utilization in each of the three regional centers.

Review of utilization data of the entire Mental Health Service has helped to improve care by indicating needed program modifications. Two examples will illustrate the usefulness of program-wide utilization review. Two years ago one of the three regional centers was found to be highly successful in reaching its underserved poor population. It was then relatively simple to examine and determine what this center was doing that the other two centers were not. Within an 18-month period, all three centers were reaching their Medicaid enrollees at about the same high utilization level. A second example was the discovery that one center was reaching less than half the number of elderly patients that the other two centers were reaching. Again, examination of the operation led to the discovery of what should prove to be corrective measures.

In both these instances the single most important factor necessary to adequately reach underserved populations proved to be a sufficient number of staff. Patients (particularly the poor and aged) who must wait for services tend to turn away, and so do the sources that referred them. Another factor of importance for serving minority groups is the presence of a multilingual and interracial staff. The aged and poor also need a staff that is able to move out into the medical group centers where these subscribers receive their medical care.

Program-wide utilization review can also predict future patterns of service with fairly good accuracy; some of these predictions may be helpful administratively. For example, Medicare utilization data reveal that only approximately 18% of aged patients attended more than 15 treatment sessions in a year, that only 3% attended more than 40 sessions, and that 50% were seen for 5 or fewer visits per year. The adult state employee group offers the greatest contrast to Medicare enrollees: 33% of these patients attended more than 15 sessions and another 33% were seen for only 5 or fewer visits per year. (Patterns of utilization by major subscriber groups are presented in table 2.) The stability of these patterns helps in planning staff size and staff deployment. The information is also useful in supervising clinical personnel.

Utilization by Medicare

Although the Social Security Administration's policy on mental health coverage is beyond the scope of this paper, I would like to make a few comments on the subject. The over-65 subscriber (the Medicare subscriber) has the lowest utilization rate of mental health services nationally (7). The consultation rate for HIP's Medicare population was 5.8 per 1,000 in 1975. This compares with a consultation rate of 14.3 per 1,000 for those below the age of 65. The rate of services that the Medicare population receives was also low: 83 per 1,000 population, compared with 221 services per 1,000 for the under-65 population (see table 1). The low utilization of mental health services by the aged contrasts strikingly with their high utilization of other medical services. These figures make it hard to understand why the federal government discriminates against psychiatric treatment benefits under Social Security. The present limit of coverage for outpatient treatment in the open market is a maximum payment of $250 or 50% of reasonable charges, whichever is less, after a $50 deductible. A case for coverage equal to that for medical illness is clearly indicated.

COST

Cost per Illness

The cost per session at this HMO is not significantly different from that in private practice. At present it is about $50 per session. A natural question follows: Is there any cost advantage to mental health care provided by an HMO delivery system? I believe that the answer is yes but that the costs must be computed on a cost per illness per year basis rather than on a cost per service basis. There is a growing tendency in the mental health field to report costs on a per illness basis (8–12).

Obviously, a reduction in the number or length of sessions required to help a patient will reduce the cost proportionately. Spaced monthly visits and half-hour

TABLE 1
Utilization Data for HIP's Mental Health Service (MHS) for 1975

Item	Medicare Subscribers (N=70,242)	Medicaid Subscribers (N=40,168)	Federal Employees (N=37,761)	State Employees (N=25,879)	Total* (N=177,096)
Number of treatment sessions to MHS rider patients in 1975	5,848	9,931	8,039	4,926	29,512
Treatment session utilization rate per 1,000 1975 rider population	83	247	213	190	167
Average number of treatment sessions per patient per year	14.4	13.5	18.1	16.3	15.2
Number of new patient consultations in 1975	407	735	443	303	1,941
New patient rate per 1,000 1975 rider population	5.8	18.3	11.7	11.7	11.0
Number of patients admitted to hospitals	—	—	—	—	122**
Hospital admission rate per 1,000 1975 rider population	—	—	—	—	0.7
Approximate average length of stay in hospital (days)	—	—	—	—	15.8***

*Totals are slightly higher than the indicated breakdown due to utilization by small subscriber groups (not shown).
**Represents 6.4% of patients seen in 1975.
***Represents 6.5% of patient visits provided by the MHS.

TABLE 2
Utilization Data for HIP's Mental Health Service for One Year During January 1972–December 1974

	Medicare Patients (N=905)		Medicaid Patients (N=1,730)		Federal Plan Patients (N=1,146)		State Plan Patients (N=631)		Total* (N=4,601)	
Number of Services	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number	Percent
1–2	242	26.7	553	31.9	284	24.8	117	18.5	1,259	27.4
3–5	223	24.6	369	21.3	210	18.3	105	16.6	950	20.6
6–10	173	19.1	289	16.7	213	18.6	117	18.5	832	18.1
11–15	104	11.5	161	9.3	129	11.3	92	14.6	506	11.0
16–20	51	5.6	126	7.3	97	8.5	62	9.8	346	7.5
21–25	39	4.3	88	5.1	60	5.2	43	6.8	235	5.1
26–30	22	2.4	50	2.9	43	3.8	34	5.4	154	3.3
31–35	17	1.9	27	1.6	49	4.3	27	4.3	124	2.7
36–40	7	0.8	20	1.2	16	1.4	14	2.2	60	1.3
More than 40**	27	3.0	47	2.7	45	3.9	20	3.2	135	2.9

*Totals are slightly higher than the indicated breakdown due to utilization by small subscriber groups (not shown).
**These figures indicate that 2.9% of all patients received 13.1% of all services (N=49,884); for the Medicare subscribers, 3% of the patients received 15.2% of all services (N=9,005); for the Medicaid subscribers, 2.7% received 11.3% of all services (N=16,335); for the federal employees, 3.9% received 15.6% of all services (N=14,200); and for the state employees, 3.2% received 11.7% of all services (N=8,558).

sessions have been found to be highly effective for selected patients. Even dynamic insight therapy can be productive with visits at monthly intervals.

Socioeconomic Class and Goals of Treatment

The middle-class and blue-collar subscribers who make up the majority of this HMO population find the return-to-function treatment goals of our plan basically compatible with their own treatment goals. The average number of services per patient per year in the Mental Health Service has ranged between a low of 14.5 in one year to a high of 16.3 in another (15.2 in 1975). Using $50 as the average cost per session, the average cost per illness or mental health incident at HIP comes to $760 per year. This includes outpatient and inpatient mental health services but not the cost of hospitalization.

In private psychiatric practice, which, as a rule, in-

volves middle- and upper-middle-class patients, treatment goals often involve characterological change, self-realization, or achievement of a patient's highest possible potential; these goals require lengthy treatment at high cost. A recent survey of private psychiatric office practice conducted by the Joint Information Service of APA and the National Association for Mental Health (13) reported an average of 38 sessions per patient per year when psychoanalytic patients were excluded and 46 visits per patient per year when they were included. For psychoanalytic patients only, the number of visits jumped to 138 per year. Using $40 as the average private fee, the cost per mental health incident for the average private patient, excluding psychoanalysis, came to $1,520 per year. When psychoanalysis was added, the cost per incident per year rose to $1,840; the cost per average patient in psychoanalysis was about $5,000 per year.

With return-to-function goals, the cost per illness will predictably be modest; with goals that include long-term therapy, the cost per illness must rise. Nevertheless, long-term therapy appears to be insurable (7, 14) because only a small number of recipients utilize these services.[1] In comparison with the cost for physical illnesses, the cost of mental health services is relatively stable, predictable, and reasonable. The late Dr. Louis Reed's report on insurance data (15) supports the belief that, providing the insured base is national, probably less than $15 per person per year could cover both inpatient and outpatient psychotherapy, including long-term psychoanalytic treatment. However, elective treatment, i.e., treatment for self-actualization or training purposes, would not be covered.

A HIP pilot study concluded that optimum coverage in an HMO would provide intensive-reintegrative therapy for the approximately 3% of the patient population who would not significantly improve otherwise, as well as for the estimated one in 10 who could electively benefit by it. For most of the patient population (approximately 87%), return-to-function goals are more appropriate, more practical, and more therapeutically valid. Theoretically, there is no reason why long-term therapy could not be included in an HMO plan if the premium were increased to cover the utilization of an upper-middle-class or mixed population.

Indirect Expenses

As would be expected in view of their basic philosophy, HMOs are concerned with preventive psychiatry. They operate on the premise that reduction of psychopathology through preventive measures and comprehensive mental health care reduces the requirement for other medical services and therefore may indirectly save the organization money (16, 17).

Approximately 30% of the therapist's time in the HMO is spent in personal and/or telephone contacts with outside agencies, school personnel, and relatives of patients; in consultation with referring physicians; in teaching seminars for HIP medical group physicians; in subscriber education programs; and in in-service education. In comparison, it is estimated that 5%–10% of the private practitioner's professional time is expended on preventive measures and what can be called "comprehensive aspects" of mental health care. Since these indirect services are absorbed in determining the cost per visit, HMO and private practice cost per visit data are not truly comparable.

Interdisciplinary Staff

Another consideration in cost containment for an HMO is its use of the lesser paid disciplines as providers of psychotherapy. Approximately 30% of the mental health services in our program are provided by psychiatrists, 30% by psychologists, and 40% by psychiatric social workers. A 1975 report from the Group for the Advancement of Psychiatry (4) pointed out that the HMO system tends to attract highly skilled nonpsychiatrist professionals because there is a greater supply of available manpower in nonphysician categories and because salaries for them are more competitive in HMOs.

Community Resources

Our Mental Health Service uses available community resources for services that are not covered, such as vocational rehabilitation services, day hospital centers, state hospitals for chronic hospitalization, and visiting nurse services for home care. This permits broad mental health coverage without increasing the premium to the subscriber; however, for national health insurance purposes, the cost for these services should be included in order to arrive at a total cost per illness.

HIP's Mental Health Service maintains most of its chronic psychotic patients within the community and outside the state hospitals. It is fiscally feasible to have these patients seen by a physician indefinitely as long as monthly visits, with or without medication, prove to be sufficiently supportive for their maintenance.

Hospital Utilization

Still another very important fiscal concern is the high cost of psychiatric hospitalization. The utilization of psychiatric hospitalization is characteristically low in prepaid group practice programs. During 1975, only .7 per 1,000 of the HIP mental health rider population were hospitalized for psychiatric care. This represented 6.4% of the year's Mental Health Service patient population (see table 1). Non-HMO hospitalization rates are more than 4 times higher for comparable socioeconomic groups (18). The patient's average length

[1]Blue Cross and Blue Shield data for Washington, D.C., showed that approximately 13% of the psychiatric patients used 64% of the dollar expenditures (7). Our data at HIP showed that 10% used 34% of the expenditures. However, the Joint Information Service survey data (13) are disquieting in that if they are projected into national health insurance coverage, the 86% of families in the United States whose earnings are under $20,000 a year would pay the greatest share of the cost for services largely rendered to individuals in families whose incomes are over $20,000 a year.

of stay, once he or she is hospitalized, is higher for the HMO than for comparable groups, 15.8 days compared with 12.1 days (18). The markedly lower number of patients hospitalized, however, makes for a critical cost saving.

The low rate of psychiatric hospitalization in the HMO is due to two factors: the early, continuing, and ever-ready (24-hour) availability of outpatient coverage; and the absence of the kind of financial incentive for hospitalization that exists in indemnity plans that offer payment for psychiatric patients only if they are hospitalized.

MEDICAL AUDIT

The best means of assuring quality control is through an assessment of the treatment process and outcome. At HIP we have started to collect data on outcome. We hope that this research will provide information on the interrelationship of the patient's chief dysfunctional behavior or symptoms and the following factors: the therapist's predicted treatment plan, subsequent changes in the treatment plan, the duration of treatment, the psychopharmacologicals prescribed, and treatment progress and outcome.

CONCLUSIONS

The inherent danger in concentrating on accountability is that we may lose sight of the patient. We must ask ourselves (19), Are the clinical needs of the patient, rather than the need to contain costs, the primary focus? The critical challenge remains one of maintaining or improving the quality of mental health care for the patient while at the same time controlling or reducing its cost. We expect that this problem will be met creatively by the development of new and more effective modalities and techniques of psychotherapy, by the discovery of illness-specific treatment regimens, and by improved and more economical systems of mental health delivery.

Cost accounting in mental health care should be based on cost per illness rather than cost per service in order to better evaluate the financial effectiveness of different delivery systems. At present, however, such comparisons are at best complex and of limited validity (12).

An HMO delivery system for mental health care offers certain advantages in the area of accountability: the use of sophisticated utilization review procedures, reduced cost per mental health incident, and a low utilization rate and low cost for psychiatric hospitalization. However, an important positive factor for cost containment, namely, the HMO's return-to-function treatment goals, also proves to be a potential handicap to providing quality care. These goals are perceived by some of the better educated and upper-middle-class patients as too limited. For some patients from all socioeconomic classes (estimated variously between 3% and 25% of psychiatric patients), these goals may prohibit the delivery of an essential treatment modality. However, with a modest increase in premium, the more costly long-term therapeutic modalities can be covered without losing many of the advantages of prepaid group practice.

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Cyclothymic Disorder: Validating Criteria for Inclusion in the Bipolar Affective Group

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The authors identified 46 cyclothymic probands from a random pool of 500 psychiatric outpatients and prospectively followed them over a 2–3-year period. They used 50 bipolar patients with a definite history of mania and 50 patients with personality disorders as control groups. Although 66% of the cyclothymic outpatients had previously received the diagnosis of hysteria or sociopathy, their pedigrees were similar to those seen in classical bipolar manic-depressive illness; furthermore, 44% of the cyclothymic group experienced brief hypomanic episodes while taking tricyclic drugs, and 35% developed full-blown hypomanic, manic, or depressive episodes during drug-free follow-up. The authors conclude that these findings provide evidence for a cyclothymic-bipolar spectrum.

> There are certain temperaments which may be regarded as *rudiments of manic-depressive insanity*. They may throughout the whole of life exist as peculiar forms of psychic personality without further development; but they may also become the point of departure for a morbid process which develops under peculiar conditions and runs its course in isolated attacks.
>
> —Kraepelin (1, p. 118)

THE TERM "CYCLOTHYMIA" was coined 100 years ago by Kahlbaum (2) as a unifying clinical concept for what was then known as "circular insanity." Although some European authors such as Kurt Schneider (3) have referred to the entire domain of manic-depressive disorders as cyclothymia, the Anglo-American literature (4–6), following Kraepelin (1) and Kreschmer (7), generally restricts cyclothymia or cycloid personality to the subaffective personality conditions seen in the biological relatives of manic-depressive patients. Furthermore, other workers have reported that cyclothymic and related temperaments occur in 30%–80% of various manic-depressive populations (6, 8, 9).

As quoted above, Kraepelin believed that cyclothymia also existed in attenuated forms, without ever progressing to the fully developed illness. Thus, as currently used, cyclothymia refers to three related conditions: 1) affective temperaments seen among the biological relatives of manic-depressive probands, 2) premorbid affective traits predisposing to manic-depressive illness, and 3) subaffective or "abortive" forms of manic-depressive illness. Although sanctioned by tradition, the postulated relationship between certain affective personality types and manic-depressive illness can be challenged on the grounds of retrospective bias. In more recent years, armed with the methodological rigor of blind family interviews, Gershon and associates (10) have demonstrated an excess of cyclothymic and related personality disorders in the biological relatives of affectively ill probands. Such data provide general support for the hypothesized relationship between affective personality and affective illness.

However, a stronger test for the cyclothymic-bipolar continuum hypothesis should come from the reverse strategy. In other words, given a cyclothymic population selected without regard for family history, one should demonstrate in a prospective design that such patients share some of the crucial validating characteristics of the more typical or fully expressed forms of the illness. For instance, the following should be shown: 1) that such a cohort of cyclothymic probands comes from families "loaded" with the same kind of affective illness as classical bipolar manic-depressive illness; 2) that the spontaneous risk for developing frank and full-blown episodes of depression, hypomania, or mania is higher than that expected by chance and exceeds that of other personality disorders; 3) that, as in other forms of bipolar illness, the risk of developing hypomania is increased by the administration of tricyclic antidepressants and other drugs that mobilize catecholamines (11); and, finally, 4) that their response profile to lithium carbonate is comparable to the classical forms of the illness.

We were unable to locate any publication solely devoted to subaffective cyclothymic disorder studied in a

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prospective design, yet we came across several reports advocating the use of lithium for such subaffective forms of the illness (12–14). Campbell's anecdotal case histories from the prelithium era remain the most detailed descriptions on the subject (15). Since the advent of lithium therapy for bipolar manic-depressive disorder, accurately describing and classifying these disorders has assumed major clinical and research significance. Dunner and associates (16) and Fieve and Dunner (17), for instance, proposed the following criteria for subtyping bipolar conditions: 1) bipolar I, hospitalized for mania; 2) bipolar II, hospitalized for depression with history of hypomania; and 3) other bipolar and cyclothymic personality, for those residual cases of bipolar illness hospitalized for neither depression nor mania. In order to subsume cyclothymic disorder under the general rubric of bipolar illness, one must first provide explicit phenomenological criteria to permit its reliable identification and then demonstrate that such a sample of cyclothymic probands drawn from a general psychiatric population shares other fundamental characteristics of the more classical forms of the illness.

In this article we will report our operational findings on a cohort of ambulatory cyclothymic probands studied in a prospective design and validate the manic-depressive nature of our case material on the basis of the first three principles outlined above. The pharmacological response profile of cyclothymic disorder to lithium carbonate will be presented in another communication.

PATIENTS AND METHOD

Screening of Patients

The study began 3 years ago when, over a period of 15 months, we screened about 500 ambulatory psychiatric patients who had never been hospitalized for depression, hypomania, or mania in order to identify cases of cyclothymic temperament. Forty-six patients, representing roughly one-tenth of the population under study, were selected.

Initially, we identified cyclothymic patients on the bases of our intuitive clinical impression that these patients represented incomplete forms of bipolar illness. They exhibited some features of both hypomania and depression as outlined in the criteria of Feighner and associates (18), but they failed to meet the sustained duration and/or the syndromal criteria for these disorders.

The cyclothymic patients came to psychiatric attention because of various personality maladjustments; the affective basis of their dysfunction became evident only after intensive clinical evaluation. Although the patients experienced considerable subjective distress, their primary reasons for seeking psychiatric help were in all cases related to serious problems in interpersonal, scholastic, or vocational areas. Therefore, it is not surprising that they had previously received the entire gamut of *DSM-II* personality labels, such as hysterical, emotionally unstable, explosive, passive-aggressive, sociopathic, inadequate, narcissistic, borderline, or obsessive-compulsive (19). Histrionic and sociopathic traits were noted in about two-thirds of the sample.

To avoid biasing our sample, we did not use family history of proven bipolar illness as a diagnostic aid.

Spitzer and associates (20, 21) stipulated that cyclothymic personality is an intermittent type of affective illness characterized by "recurrent periods of depression lasting at least a few days alternating with similar periods of clearly better than normal mood" (20, p. 25). We found that exclusive reliance on either depressed or elated mood provided many false positives. For instance, many patients who satisfied the Feighner criteria for Briquet's disorder (hysteria) and antisocial personality (18) frequently reported such mood swings. Therefore, we required the presence of unequivocal behavioral manifestations with the mood changes. (Patients who reported mood swings but did not exhibit behavioral signs were classified as "pseudocyclothymic.")

The population we selected and prospectively examined has given us the opportunity to identify the most striking clinical features derived from formal psychotherapeutic sessions and from interviews in which we collected systematic demographic, developmental, phenomenological, vocational, and interpersonal data. We undertook an extensive phenomenological and biographical analysis of each patient in long-term (6–12 months) weekly or biweekly psychotherapeutic sessions. The unpredictable mood swings, the dramatic shifts in behavior from day to day, and the tempestuous nature of object relations in these patients had produced multiple episodes of romantic breakups or marital failure. Therapy therefore focused on dyadic interactions.

Follow-Up

The patients were prospectively followed over a 2–3-year period. When clinically warranted, 54% received tricyclic antidepressants for brief periods of several weeks and/or lithium carbonate later during follow-up for periods ranging from 6–18 months.

Throughout the period of prospective observation, mania and depression were recorded on the basis of the Feighner criteria (18) by clinicians who were blind to the previous diagnosis of cyclothymia. Differentiating hypomania from mania is a matter of convention. Dunner and associates (16) distinguished hypomania (bipolar II) from mania (bipolar I) on the basis of hospitalization, but Spitzer and associates (20) required a shorter duration and one less symptom from the Feighner list for mania in the differential assignment of patients to one category or the other. Because mania is the more severe form of the two, in our view the presence of any of the following marked the transition from hypomania to mania: 1) general changes in demeanor from pleasant cheerfulness to sustained

querulous belligerence; 2) presence of affective delusions or hallucinations; and 3) serious social impairment that might require hospitalization.

Control Groups

We used two control groups. One was composed of 50 patients with bipolar I disease with a definite history of mania. By excluding bipolar II (hospitalized for depression) patients from this control group, we provided an unequivocal sample of classical bipolar I manic-depressive patients for comparison.

The other control group was composed of the 50 patients whom we had identified as pseudocyclothymic. These patients had various kinds of personality disorders and were superficially similar to the experimental group in that they reported frequent mood swings in the direction of both elation and dysphoria. However, they did not manifest the behavioral characteristics seen in the true cyclothymic probands.

Both control groups were randomly selected from consecutive admissions into our mood clinic during the same period when the cyclothymic probands were being screened. Because we were interested in a comparative analysis, we specifically did not control for age at first psychiatric presentation, sex, race, or social class. Actually, these demographic data turned out to be quite comparable and will not be further mentioned in this report. About 60% of both control groups received tricyclic antidepressants for several weeks or months; the bipolar I group was ultimately maintained on lithium carbonate. As with the experimental group, individual and/or couples therapy were provided for interpersonal conflicts.

Family History

Family history was obtained in a blind fashion according to the method of Winokur as incorporated into the family history-research diagnostic criteria (FH-RDC) (22). History of affective illness in first-degree biological relatives was obtained from at least two of the following sources: 1) the affected family member, when accessible; 2) records, facilitated by the fact that about 35% of the affected relatives were patients in our clinic; and 3) the spouse, sibling, parent, or children of the affected family member. Because cyclothymia represents the dependent variable in this study, family history was coded positive only for definite unipolar and definite bipolar biological relatives as well as for unequivocal suicides.

We used the chi-square test with Yates' correction for continuity when appropriate. Informed consent was obtained from all patients who participated in the study.

RESULTS

Demographic Features

Thirty-eight percent of the cyclothymic sample were men and 62% were women; 10% were black and 90% were white; there was an approximately equal distribution of Hollingshead's classes II–IV (23). The age range at first psychiatric presentation was 15–45, with a preponderance of patients between the ages of 15 and 25. The salient clinical features of the sample of cyclothymic patients are outlined below.

General Criteria for Cyclothymia

As seen in our 46 patients, the disorder usually had its onset in the early or mid teens but in no instance later than early adulthood. However, despite significant psychopathology, several patients in our series did not seek psychiatric help until much later in life.

Typically, patients manifested recurrent and short cycles of depression and hypomania that clearly fell short of the full syndromal criteria for these disorders. These episodes, according to selection criteria, generally did not crystallize into the full syndromes of depression or hypomania, although they did so in about 10% of the cases. Again according to selection criteria, the episodes typically lasted a few days, rarely up to a week. Failure to meet the sustained duration criterion was the major reason for the fact that these patients were not assigned the diagnosis of definite manic-depressive illness. The episodes alternated with each other in an irregular fashion. Intervening even periods were rare, occurring in less than 10% of the cases. As a consequence, partial mixtures of the subsyndromes often appeared as mixed states of a few days' duration, with irritable and explosive mood in about 50% of the patients. The onset of episodes was generally unrelated to external events or circumstances, although the episodes provoked life events of a tempestuous nature in well over 75% of the patients.

Operationally Derived Phenomenological and Behavioral Criteria

Although each of the items listed below might suggest a personality disorder when viewed individually, the clustering of these signs and symptoms should alert the clinician to the possibility of an underlying affective disorder. Because patients usually manifest subsyndromes and mixed states, ordinarily each patient will not manifest more than several of the characteristic types of behavior that follow:

1. About 50% displayed irritable periods lasting a few days that did not crystallize into an anxiety syndrome. For instance, the autonomic signs and symptoms of anxiety were rarely observed.
2. Fifty percent suffered from explosive aggressive outbursts that were ego-dystonic and often followed by periods of guilty rumination. These features differentiate cyclothymia from hysteria and sociopathy, and the absence of amnesia serves to distinguish it from epilepsy.
3. Repeated buying sprees, financial extravagance, or financial disasters were seen in about 75% of the patients.
4. Repeated shifts occurred in line of work, study, interest, or future plans in about 50% of the patients.

These shifts created the impression that their lives were chains of improvident activities, yet there were usually no other observational signs of flagrant sociopathy.

5. Episodic or unexplained promiscuous behavior or extramarital affairs occurred in an ego-dystonic fashion, appeared self-defeating, and often led to self-reproach when the depressive swing supervened. These features, found in about 40% of the patients, distinguish cyclothymia from sociopathy.

6. Finally, 25% of the patients exhibited a pattern of joining new movements with impulsive zeal that rapidly changed to utter disillusionment. Thus they displayed a lower threshold for seduction by all kinds of recondite movements and eschatological cults.

The presence of the following criteria should help to establish the biphasic nature of cyclothymia. Again, such behavior occurred in a dissociated form, and each patient characteristically exhibited just a few of the behavior patterns listed: hypersomnia alternating with decreased need for sleep (75% of the patients); shaky self-esteem alternating between lack of self-confidence and naive or grandiose overconfidence (75%); marked unevenness in quantity and quality of productivity whether at work or in school, often associated with unusual self-imposed working hours (75%); periods of mental confusion and apathy alternating with periods of sharpened creative thinking (50%); uninhibited people-seeking alternating with introverted self-absorption (50%); and an alternating pattern of drug abuse (stimulants and psychedelics alternating with sedative-hypnotic drugs) or dipsomania. Drug and alcohol abuse, which occurred in about 50% of the cases, represented either attempts at self-treatment or the self-indulgence that characterized the "up" periods.

Illustrative Case History

Case 1. A 29-year-old man was referred by his ex-wife for psychiatric evaluation. After 7 years of marriage they had just been divorced for the second time as a result of his many extramarital affairs. During their second marriage, he had undergone 2 years of outpatient analytical treatment to alter his "narcissistic personality makeup." Shortly after the second divorce, the wife went to graduate school to train as a social worker; there she became acquainted with descriptions of manic-depressive illness. She started experiencing guilt feelings because she felt that her husband might have been sick rather than "simply refusing to grow up." After many months of persistent effort, she finally succeeded in getting her ex-husband to go to the psychiatrist who had lectured to her class.

The patient had 3–4-day periods of "hypersexuality" during which he would sleep 4–5 hours a night and demand frequent intercourse. This was enjoyable to his wife during the first year of their marriage, but then she started experiencing discomfort, which was further compounded by her endometriosis. He then started frequenting bars in order to satisfy his "satyriacal needs." At other times he was intermittently lethargic or irritable, indulged in alcohol, slept 8–9 hours a day, and neglected his wife sexually. He was employed as an architect and during his "up" periods he would sometimes go to his office at 4:00 A.M. on weekends; during his "down" periods he would accomplish very little and resort to the use of amphetamines to keep going. He felt that what he termed his "naturally occurring up periods" were superior to "artificial speeding."

His employer valued him because he developed outstanding and creative projects at certain times of the year, even though his overall yearly output was considerably less than that of his colleagues. He was quite unpredictable in many areas; for instance, during a vacation, while his wife slept in a motel he took off from Miami to New York and returned the following afternoon. The patient denied subjectively experiencing mood swings; he was only aware of being "active and up" or "lazy and down." He stated that during his "up" periods he felt driven to do "crazy" things, such as reading *Time* magazine from cover to cover, including all the ads. He was known to be generous and showered his wife and lovers with expensive jewelry.

He had no biological relative who had received psychiatric treatment, but a maternal uncle had committed suicide in his mid-40s after having lived "a wild life of luxury and lechery." This patient's short cycles were evened out with a 3-month trial of lithium, and the couple married for the third time. For a while his wife complained that sex relations occurred far too infrequently, but 6 months of couples therapy restored conjugal homeostasis. The patient is still creative, but his productivity is evenly distributed throughout the year and he no longer uses amphetamines.

This case illustrates some of the cardinal features of cyclothymic disorder: presentation as a personality problem resulting in repeated conjugal failure; nonprominence of the affective symptoms; a clearly biphasic nature of the symptoms; short cycles; episodic and alternating pattern of drug abuse; and self-imposed hours of work and cyclicity of productivity.

We are grateful to this patient's wife for bringing him for psychiatric consultation. He was our first patient in this series on cyclothymia and alerted us to the possibility that manic-depressive illness may manifest itself in subtle and disguised forms.

Family History Characteristics

Table 1 summarizes the family history characteristics of the experimental group compared with the two control groups. The cyclothymic patients and bipolar I patients have very similar patterns of familial incidence of suicide, unipolar disorder, bipolar disorder, and total incidence of affective illness in two consecutive generations. In contrast, the incidence of these same disorders (with the exception of unipolar disorder) is negligible in the families of the pseudocyclothymic patients.

More importantly, 5 of our cyclothymic probands appeared to provide the link for the transmission of the illness in three consecutive generations, as shown in the sample pedigree in figure 1. In this pedigree a cyclothymic prepositus is shown in generation II. Her husband's side of the family is completely free of affective illness; therefore, the prepositus is the only possible intermediary for the transmission of manic-depressive illness from her mother in generation I to her children in generation III.

TABLE 1
History of Affective Illness in Biological Relatives of 46 Cyclothymic Patients, 50 Bipolar I Patients, and 50 Pseudocyclothymic Patients

Affective Illness in Relative	Cyclothymic Patients		Bipolar I Patients		Pseudocyclothymic Patients		Significance*
	Number	Percent	Number	Percent	Number	Percent	
Unipolar	7	15	11	22	5	10	n.s.
Bipolar	14	30	13	26	1	2	p<.01
Suicide	10	22	9	18	2	4	p<.03
Total**	30	65	28	56	7	14	p<.001

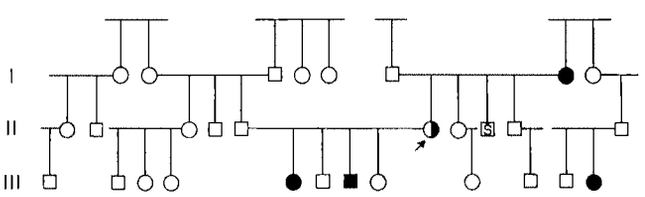
*The cyclothymic patients and the bipolar I patients were very similar in these comparisons. The p values represent the cyclothymic and bipolar groups individually compared with the pseudocyclothymic group. In all cases the p values were the same for both comparisons.
**The total number of patients with a positive family history of affective illness is less than the sum of the first three rows in each column because some cases of suicide were from the ranks of either unipolar or bipolar patients.

TABLE 2
Affective Episodes of 46 Cyclothymic Patients and 50 Pseudocyclothymic Patients During Drug-Free Periods of Follow-Up

Affective Episode	Cyclothymic Patients		Pseudocyclothymic Patients		Significance
	Number	Percent	Number	Percent	
Depression	11	24	2	4	p<.02
Hypomania and mania	10	22	0		p<.01
Total*	16	35	2	4	p<.001

*The total number of patients is less than the number of episodes shown in the first rows of both columns because several patients experienced both depression and hypomania.

FIGURE 1
Pedigree of 1 Cyclothymic Patient*



*As the figure shows, the cyclothymic patient served as the genetic link in this three-generational pedigree with manic-depressive illness. The Roman numerals to the left of the figure indicate first, second, and third generations; ■ and ● = manic-depressive male and female family members; □ and ○ = unaffected male and female family members; S = completed suicide in a male family member; and ◑ = cyclothymic prepositus.

Drug-Free Follow-Up

As shown in table 2, 16 of the 46 patients in the experimental group (35%) progressed to spontaneous affective episodes that met the full duration and syndromal criteria for hypomania or depression. Eleven developed depression, 7 developed hypomania, and 3 progressed to mania. These episodes were spontaneous in the sense that they occurred during medication-free periods. Therefore, 3 patients could be reclassified as bipolar I (frank episode of mania) and 13 patients as bipolar II (frank episode of depression, hypomania, or both). These episodes occurred, on the average, 1–2 years after the index diagnosis of cyclothymia and 2–4 years after patients first came for psychiatric help. By comparison, only 2 of the 50 patients classified as pseudocyclothymic (5%) developed frank depression, satisfying the duration and syndromal criteria, and none developed hypomania or mania. The two groups differed significantly in all of these respects, but the most important difference was seen in the cyclothymic probands' increased vulnerability to hypomania or mania (p<.01). It is difficult to determine how many additional patients from the cyclothymic group would have developed full affective episodes if lithium had been withheld.

"Pharmacological" Hypomania

Brief hypomanic episodes during the course of tricyclic therapy occurred at a similar rate in the cyclothymic and bipolar I patients. Thus 14 of 40 bipolar I patients (35%) and 11 of 25 cyclothymic patients (44%) experienced hypomania, but none of the pseudocyclothymic patients receiving tricyclics did. The differences between the pseudocyclothymic versus both the cyclothymic and bipolar I groups are highly significant (p<.001). Whether the episodes under consideration were pharmacologically induced or would have occurred anyway is difficult to determine; although brief in duration, they were phenomenologically identical to the naturally occurring episodes and tended to subside after the antidepressant was discontinued. The exclusive occurrence of this response to tricyclics in both cyclothymic and bipolar I groups in comparison with the pseudocyclothymic group suggests that the bipolar I and cyclothymic

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patients shared important phenomenological and pharmacological response profiles.

DISCUSSION

Kraepelin described four overlapping temperaments, identified either by premorbid traits in manic-depressive patients or by constitutional peculiarities in their biological relatives (1). These were the depressive, the hypomanic, and the irritable (a simultaneously occurring mixed form of the first two) temperaments, plus the cyclothymic temperament proper (an alternating pattern of the first two types). Kurt Schneider later substituted "dysthymic" for the depressive and "hyperthymic" for the hypomanic personalities, respectively (3).

Our selection criteria—requiring biphasic clinical features—excluded pure dysthymic subjects, who would currently be subsumed under the rubric of depressive characters. We are studying a population of dysthymic patients and will report our findings elsewhere. As to the other subtypes, we discovered that hyperthymic patients did not account for more than 2 patients, i.e., 5% of the total sample; moreover, these 2 patients occasionally experienced depression. Similarly, the irritable group seemed to represent just one phase of the overall cyclothymia rather than a separate category. These findings are analogous to the relative rarity of pure recurrent mania (6, 9) and the occurrence of mixed states as transitions from mania to depression (1, 24) in the full-blown forms of manic-depressive illness.

There are two major difficulties in identifying "true" cyclothymic patients. Many hysterical and sociopathic patients report mood swings in both elated and dysphoric directions. Also, characterological cases of unipolar depression sometimes masquerade as moody states because their occasional normal periods are reported as "highs" relative to their depressive states. To avoid such pitfalls one must document clear-cut behavioral changes consonant with the mood and insist on the presence of definite biphasic behavioral changes.

Klein and associates (25) described a type of personality that occurs predominantly in young women and is characterized by wide mood fluctuations on an hour-to-hour or day-to-day basis. They distinguished such "emotionally unstable personalities" from cyclothymic personalities on the grounds that the former are "irresponsible, hedonistic, extractive and exploitative," while the latter "pursue socially acceptable and valued goals." We cannot make such a dichotomy because our patients exhibited mixtures of both patterns; this finding is actually in line with Kraepelin's classical descriptions (1). Dunner (26) stated that the syndrome identified by Klein's group is the same as bipolar II disorder. Since Klein's group did not provide family history data, it is hard to resolve this difference of nosological assignment for the emotionally unstable group. We obtained family history data on our cohort of 46 cyclothymic patients; therefore, we can address this question with some degree of rigor. Our findings provide strong support for the existence of a group of attenuated, cyclothymic, or subaffective disorders that merge imperceptibly with the bipolar II (and sometimes bipolar I) forms of manic-depressive illness. The evidence is as follows:

1. Phenomenologically, cyclothymic disorder presented with characteristic signs and symptoms that clustered into incomplete and short cycles with definite evidence for a biphasic course.
2. In terms of family history, the distribution of affectively disordered biological relatives in the pedigrees of cyclothymic and bipolar I probands was almost identical. Moreover, in several pedigrees the cyclothymic proband provided the only possible link for transmitting the illness in three-generational perspective; this is perhaps the most compelling evidence for considering cyclothymic disorder as an attenuated form of the more classic manic-depressive illness. Comparison with the pseudocyclothymic group of personality disorders indicated that family history of bipolar illness is the most useful familial predictor of true cyclothymia.
3. In longitudinal perspective, almost one-third of the cyclothymic group progressed into the full-blown clinical picture of manic-depressive illness. However, ours was a psychiatric sample; conceivably such progression may occur less frequently in a general population sample.
4. Pharmacologically, the frequency of hypomanic episodes during tricyclic therapy was very similar in the cyclothymic and bipolar I probands.

Our selection criteria are further validated because control probands with personality disorder and mood swings but without behavioral manifestations of a biphasic nature displayed none of the characteristics noted above. Therefore, to diagnose cyclothymic disorder, major emphasis should be laid on the behavioral manifestations of the syndrome and not mood swings per se.

Of sociological relevance is the finding that cyclothymic patients, who comprised 10% of a nonhospitalized psychiatric population with mixed personality disorders, displayed a high incidence of promiscuous behavior and marital failure, financial disasters, heightened but uneven creativity and unstable employment records, an episodic pattern of drug and alcohol abuse, and a low threshold for seduction by various recondite cults and eschatological movements. Some psychiatrists have suggested that psychiatry should withdraw from the arena of problems of living represented by personality disorders (27). Our data, however, raise the possibility that some forms of minor social "deviance" may have their origin in attenuated, unrecognized, or masked forms of potentially treatable affective illness. The therapeutic fate of these patients with lithium carbonate will be presented elsewhere.

In closing, we wish to caution the reader that at the present time we do *not* advocate the routine diagnosis of cyclothymic disorder in everyday practice. The identification of this disorder should be contemplated only after in-depth phenomenological examination of individual patients seen in specialized affective disorder clinics where continuity of care and follow-up are provided by a team of experienced clinicians. The same caution applies even more stringently to lithium trials in such subaffective forms of manic-depressive illness.

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Clinical Pharmacology of Phencyclidine Toxicity

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Phencyclidine appears to be unique in action compared with other psychedelic drugs, and its effects are less dependent upon the individual's personality than are the effects of LSD or mescaline. The authors discuss the sensory, psychological, and behavioral symptoms of phencyclidine intoxication. Most cases are of short duration and the only treatment necessary may be observation together with minimal stimulation and diazepam. However, prolonged and severe behavioral disturbances, exaggeration of preexisting thought disorder, and serious medical complications commonly occur and must be considered in the treatment plan.

PRIMARY CARE physicians throughout the United States are being confronted by patients intoxicated by a relatively new psychedelic drug, phencyclidine, 1-(1-phenylcyclohexyl) piperidine (Sernyl, Sernylan). While phencyclidine is the parent compound of the intravenous anesthetic ketamine, it was discarded as an anesthetic agent for humans and is now legally sold only to licensed veterinarians for use as an immobilizing agent for subhuman primates. In its illegal forms it is known as PCP, "angel dust," and a variety of other street names. As with many other popular mind-altering drugs, phencyclidine appeared on the West Coast in the 1960s and from there spread throughout the country.

Although its many unpleasant side effects initially discouraged its widespread use, recently the manufacture and use of illicit phencyclidine have become extensive. Phencyclidine is frequently a drug of choice, especially when it is sprinkled over marijuana and smoked. It is frequently misrepresented by sellers as TIC or THC (tetrahydrocannabinol, which is the active ingredient in marijuana and is probably not found in illicit circles), mescaline, peyote, psilocybin, and a variety of synthetic psychoactive substances. Misrepresentation of phencyclidine is widespread because of its undesirable side effects, its ready availability compared with what it is represented to be, and the wishful belief of the drug user that he or she is experiencing a more exotic natural drug such as mescaline or psilocybin. These latter drugs are infrequently found in analyses of street drug samples. Such deception adds to the physician's difficulty in making an accurate diagnosis in the case of a suspected drug intoxication, even with a cooperative patient or his well-meaning friends.

When correctly synthesized, phencyclidine is a glistening white, crystalline powder, readily soluble in both water and alcohol. In veterinary medicine the liquid injectable form is used, although the powder is still legally manufactured. Phencyclidine produced by underground laboratories appears in liquid form, as an off-white powder, or in tablets of various sizes and colors and of variable purities. The powder may be found in capsules, foil, or any similar makeshift containers.

Since the ingredients for phencyclidine are readily available and synthesis is relatively simple, "basement" chemists have become involved in its production on a large scale and at great profit. The production of a pound of phencyclidine requires about a $75 investment in raw chemicals and can bring street sales in excess of $20,000. In 1974, seizure of an underground laboratory in Maryland yielded 90 pounds of phencyclidine, half in powder and half in liquid form, and a similar confiscation in Detroit shut down production of an estimated one million phencyclidine tablets a week. With production and profits of this magnitude it is not surprising that this drug has spread across the United States as well as Canada and Europe. A recent study conducted in Detroit (1) reported that phencyclidine was the most frequently used drug in 1,000 cases of substance intoxication. Several cases of accidental ingestion of phencyclidine by children have been reported in the literature (2, 3).

SENSORY AND PSYCHOLOGICAL MANIFESTATIONS

While phencyclidine is often labeled a hallucinogen, it is questionable whether true hallucinations occur with its use. In fact, some researchers (4, 5) have used the absence of hallucinations to distinguish the effects of this drug from those of LSD and mescaline. Phencyclidine appears to be unique in action compared with other psychedelic drugs, and its effects are less dependent upon the individual's personality than are the effects of either LSD or mescaline. Individuals who have taken phencyclidine do not describe the in-

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creased ability for self-observation or the kaleidoscopic visual hallucinations reported by users of the more classic hallucinogenic substances (6). On the contrary, the phencyclidine experience is more likely to be unpleasant, with impaired clarity of thought, marked anxiety, and psychosis.

A consistent sequence of psychological impairment has been found when the drug is given in an experimental situation (4). The first symptoms to appear are changes in body image related to parts of the body or to the total body image. Some subjects describe their limbs as elongated or floating away from their bodies, while others report alternate contraction and expansion of body size. It is likely that it is these body image distortions that are incorrectly interpreted as hallucinations by both patient and physician. Progressive feelings of depersonalization, isolation, and depression frequently follow and often persist for hours. Anxiety is one of the most common findings and is often the most severe presenting symptom. Disorganization of thought with concrete thinking, blocking, and neologisms may be present. With increased dosages the unpleasant psychological effects are likely to increase. Negativism, hostility, and apathy are prominent and sometimes progress to a cataleptic state—a state of "dissociative anesthesia" during which major and minor surgical procedures were performed during the drug's early clinical trials. Coma, convulsions, and death, usually attributed to respiratory failure, may follow. Additional clinical findings include vertigo, diaphoresis, flushing, and frequently nausea and vomiting. In spite of these seemingly unpleasant effects, phencyclidine is becoming increasingly popular among drug users, with parenteral administration becoming more frequent.

Morgenstern and associates (7) tested subjects given phencyclidine orally and found sensory impairment in all modalities tested over the first two hours. Some modalities may be affected more than others, with two-point discrimination and touch being most affected, followed by impairment in visual fields, auditory acuity, sense of position, taste, and visual acuity. Clinical observation reveals that a general sensory impairment precedes the drug's psychological effects. The disturbances resulting from phencyclidine intoxication apparently require exteroceptive input for their arousal, and the psychological effects of the drug may be attributed at least in part to disturbances in sensory input or impairment of the ability to integrate sensory input into meaningful behavior (7–9). This theory has been supported experimentally by some improvement in phencyclidine intoxication with isolation and greater improvement with sensory deprivation (10–12). Accordingly, "talking down" the phencyclidine-intoxicated patient has not had the success attributed to the use of this technique with the LSD-intoxicated patient.

A number of studies (4, 9, 13, 14) have compared the effects of phencyclidine to psychotic processes. Several of the investigators concluded that phencyclidine characteristically produces disturbances similar to the primary symptoms of schizophrenia (such as blocking, impaired motor performance, and disruption of abstract and sequential thinking), while substances such as mescaline and LSD mimic the more secondary symptoms of schizophrenia (such as hallucinations). Phencyclidine does produce severe impairment of symbolic cognition (proverb interpretation) and of sequential thinking (serial sevens). This level of impairment is quite similar to that of a group of patients diagnosed as schizophrenic (13). Schizophrenic patients given a single dose of phencyclidine have experienced an intensification of their thought disorder lasting from several days to several weeks (4). LSD does not generally produce these effects in schizophrenic patients. The prolonged effect of phencyclidine ingestion is not limited to patients with preexisting thought disorders. Furthermore, some researchers (15) have reported a delay of several days before the appearance of severe behavioral toxicity. Anterograde amnesia frequently occurs following administration of the drug.

PHYSIOLOGICAL MANIFESTATIONS

The psychological effects of phencyclidine intoxication are accompanied by many abnormal neurological findings (3, 15). With lower doses the user experiences a giddy, drunken feeling with euphoria resembling ethyl alcohol intoxication, although marked anxiety and emotional outbursts are frequently seen in the emergency treatment situation. Ataxia, slurred speech, and tremulousness with increased deep tendon reflexes and clonic movements are common. Paresthesias and analgesia, especially of the face and extremities, and muscle weakness may also occur. As with other responses to phencyclidine, neuromuscular effects are dose related. Inebriated behavior, paresthesias, and analgesia are lower-dose phenomena, and there is a progression to anesthesia and catatonic-like posturing with higher doses. When toxic levels are reached these effects may progress to myoclonic movements, convulsions, and coma.

In early clinical trials of phencyclidine, muscle rigidity was common and was a major obstacle to its acceptance as an anesthetic. Reflex activity is exaggerated with low doses, but with high doses there is an eventual depression of reflexes (polysynaptic reflexes are affected more than monosynaptic reflexes) (16). Pharyngeal and laryngeal reflexes may remain active, thus increasing the risk of laryngospasm, bronchospasm, and coughing (17, 18). This, together with hypertonicity of facial and neck muscles, will pose serious difficulties should tracheal intubation become necessary. Eye signs of phencyclidine intoxication are many and nonspecific. Pupils are usually either constricted or normal. Nystagmus is frequently found, and diplopia, decreased pupillary light reflexes, absent corneal reflexes, and bilateral ptosis are often seen (19, 20). EEG changes occur predominantly in

the occipital, temporal, and parietal regions and are characterized in man by slowing, particularly to theta activity, but becoming more diffuse with high doses (21). These changes are different from those that occur during sleep or use of barbiturates (16). It has been suggested that phencyclidine specifically alters synaptic transmission in the brain (22). A number of studies have demonstrated altered concentrations of central neurohumoral transmitters after administration of phencyclidine (23–26). Areas of the central nervous system where phencyclidine is believed to act include the sensory cortex, thalamus, midbrain, and spinal cord (16).

Several other physiological manifestations of phencyclidine intoxication accompany the neuropsychiatric disturbances. Systolic and diastolic blood pressures are frequently elevated. In one series of tests, mean systolic elevations of 25.7 mm Hg and diastolic elevations of 19 mm Hg were reported (19); in another study systolic pressure ranged from 120–200 mm Hg and diastolic pressure from 60–110 mm Hg in high-dose ingestions (27). The pressor effects are not influenced by ganglionic blocking agents, adrenalectomy, decentralization, or pretreatment with reserpine but are at least partly reduced by adrenergic blocking drugs (28, 29). Ilett and associates (28) have suggested that the site of action of phencyclidine is the alpha adrenergic receptor; and they have demonstrated experimentally that phenoxybenzamine (Dibenzyline) and phentolamine (Regitine), alpha adrenergic blocking agents, markedly reduce or completely block the pressor response to phencyclidine. Phencyclidine also potentiates the pressor response to epinephrine, norepinephrine, and serotonin (16, 30). Hitner and associates (29) believe that the phencyclidine potentiation of catecholamines may be cocaine-like in inhibiting the neuronal reuptake of these transmitters. While the direct pressor effects of phencyclidine may last only 15–60 minutes, the pressor effects of the neurohumoral transmitters are enhanced for as long as an hour after the baseline blood pressure returns to normal (16).

Elevated blood pressure does not appear to be the result of increased cardiac output, since phencyclidine has been found to be a direct myocardial depressant. Marked reduction in the force of contraction and decreased rate of conduction have been demonstrated in isolated cardiac muscle (28). Tachyphylaxis to the pressor effects of phencyclidine develops rapidly (29). Sinus tachycardia, occasional arrhythmias, bradycardia, and hypotension have also been reported (17, 26, 31). With low dosages of phencyclidine, respiration is either unaffected or stimulated; higher dosages may result in respiratory depression, Cheyne-Stokes respiration, apnea, and respiratory arrest (3, 19).

Contrary to early and persistent reports, phencyclidine has been shown experimentally to have anticholinergic actions (32–34). The anticholinergic effects of phencyclidine are of rapid onset, of short duration, dose related, and considerably less potent than those of atropine. It is unlikely that mydriasis would be seen clinically. The anticholinergic effects may be demonstrated in isolated gut and striated muscle. Changes in body temperature have not been determined with certainty. Phencyclidine's anticholinergic action is believed to be by competitive antagonism through specific binding at the receptor site (32–34). Phencyclidine has been found to be a potent competitive inhibitor of butyrylcholinesterase (pseudocholinesterase) (32). Should surgery be necessary for the patient with phencyclidine intoxication, decreased pseudocholinesterase activity may prolong the paralyzing action of the depolarizing muscle relaxant succinylcholine. Prolonged apnea has occurred when the phencyclidine derivative ketamine was used with succinylcholine (35). No such prolonged effect was seen when ketamine was used in combination with nondepolarizing muscle relaxants.

Phencyclidine is rapidly metabolized in the liver by hydroxylation and the conjugates are excreted into the urine. Man excretes mainly the mono-4-hydroxypiperidine derivative. These metabolites are relatively weak and apparently do not contribute significantly to the pharmacological activity of the parent compound (16). In cases of phencyclidine poisoning, high concentrations of the unmetabolized drug have also been found in the urine (3). Hepatic drug metabolism is enhanced in animals after four days of treatment with phencyclidine (36), and tolerance to the effects of phencyclidine may develop with regular usage (37). Cross-tolerance of phencyclidine with other drugs has not been demonstrated. Acute hepatic cellular toxicity following phencyclidine use has not been reported at this time.

TREATMENT

Treatment of acute phencyclidine intoxication is often a psychiatric emergency, and there may be other serious medical complications as well. In one hospital study in a large metropolitan area (38), phencyclidine psychoses exceeded both schizophrenia and alcoholism as the cause for inpatient psychiatric admission. Evaluation of suspected cases of phencyclidine intoxication should rule out the possibility of other organic central nervous system pathology, a primary psychosis, or ingestion of another drug. Elevated blood pressure, frequently normal or stimulated respirations, and bizarre neurological and behavioral signs and symptoms help to distinguish phencyclidine intoxication from most narcotic and sedative overdoses. Methaqualone intoxication perhaps most closely resembles acute phencyclidine poisoning, especially in coma. A prolonged recovery phase and occasional delayed onset of behavioral disturbances may also lead one to suspect phencyclidine over many other psychedelic drugs.

The basis for treatment lies in a knowledge of the pharmacology of phencyclidine and its unique, dose-

related clinical manifestations. For the majority of the cases of low-dose intoxication, successful treatment may require only minimal stimulation for an hour or two, with observation to detect any changes requiring support. While talking down the anxious patient may be attempted, it often results in intensification of anxiety and agitation. Placing the patient in a quiet, dimly lit room has been our most successful approach. Parenteral diazepam is often useful in calming the patient and in treating myoclonus and seizures. It is doubtful whether there is any need for antipsychotic agents such as chlorpromazine, except perhaps in situations of severe behavioral disturbances or aggravation of preexisting thought disorders. Patients with such disorders will probably require hospitalization in a psychiatric unit. Therapeutic doses of phenothiazines have resulted in significant hypotension when used in the treatment of phencyclidine intoxication (27). In general, in cases of ingestion of unidentified drugs it is best to avoid phenothiazines and other drugs with anticholinergic properties. Illicit drugs are often adulterated with belladonna alkaloids, and phencyclidine itself has anticholinergic effects. A butyrophenone such as haloperidol is preferred when treatment with an antipsychotic drug is believed necessary.

The behavioral aspects of phencyclidine intoxication are most often the reason a patient is brought for treatment, but serious medical complications should be anticipated. To minimize serious and prolonged complications, gastric lavage is indicated in suspected cases of high-dose ingestion. Respiratory irregularities or arrest will require ventilatory support. Intubation may be difficult because of intact pharyngeal and laryngeal reflexes as well as muscle hypertonicity. Prolongation of the action of depolarizing muscle relaxants by phencyclidine should be a consideration in anesthetic management. The use of CNS stimulants in the management of respiratory depression or coma is controversial, but in our opinion the risk of increased cerebral hypoxia and resultant hypoxic seizures is too great with a drug that may inhibit respiratory response to the increased oxygen demands of the stimulant action.

Hypertension frequently occurs after phencyclidine ingestion, and treatment may sometimes be required. In view of experimental data that phencyclidine has direct pressor activity, an alpha adrenergic blocking agent such as phenoxybenzamine or phentolamine may be useful. In a case of severe hypertension after phencyclidine ingestion, the patient's blood pressure was successfully lowered with diazoxide (Hyperstat) (2). Renal function should be monitored since it has been reported that high doses of phencyclidine impede urinary output (30); at the same time hydration should be adequate to promote renal excretion of unmetabolized phencyclidine and its metabolites. Because high concentrations of phencyclidine are often found in the urine, a specimen should be sent to the laboratory for drug identification. Since many laboratories are not prepared to do analysis for phencyclidine and because of the delay in receiving a report when the test is done, treatment must begin with the working diagnosis.

CONCLUSIONS

In conclusion, the increasing availability and abuse of phencyclidine make consideration of this unique drug necessary in suspected cases of drug intoxication or in acute delirium states. Most such cases are of short duration and the only treatment necessary may be minimal stimulation, observation, and the use of diazepam. However, prolonged and severe behavioral disturbances, exaggeration of preexisting thought disorders, and serious medical complications are commonly found and must be considered in the treatment plan.

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Rape: Power, Anger, and Sexuality

BY A. NICHOLAS GROTH, PH.D., ANN WOLBERT BURGESS, R.N., D.N.SC., AND LYNDA LYTLE HOLMSTROM, PH.D.

Accounts from both offenders and victims of what occurs during a rape suggest that issues of power, anger, and sexuality are important in understanding the rapist's behavior. All three issues seem to operate in every rape, but the proportion varies and one issue seems to dominate in each instance. The authors ranked accounts from 133 offenders and 92 victims for the dominant issue and found that the offenses could be categorized as power rape (sexuality used primarily to express power) or anger rape (use of sexuality to express anger). There were no rapes in which sex was the dominant issue; sexuality was always in the service of other, nonsexual needs.

WITHIN the last few years, increasing awareness of and concern for the rape victim has become evident in this country. However, relatively little attention has been directed toward understanding the rapist and the motivations underlying his offense. A major handicap in this regard has been access to offender populations; rapists rarely report voluntarily to mental health clinics, and the conviction rate for rape is extremely low. In commenting on the professional literature on the rapist, Abel and colleagues (1) pointed to the lack of accessibility of offenders for evaluation and treatment and the failure of the professional community to develop effective and efficient models of treatment. Pacht (2) believes that until a sufficient number of controlled research studies are completed, ineffective approaches may be used in efforts to provide appropriate treatment for the rapist.

Research in the field of sexual assault is multidisciplinary (3). Amir (4), in his survey of several hundred rape cases handled by the Philadelphia Police Department, discussed the role relationship between victim and offender. MacDonald (5) evaluated rape cases in the Denver area on such epidemiological factors as time of day, season, and the relationship of victim and offender. The operational behavior approach to studying deviant sexual behavior has been undertaken by Abel and Blanchard (6), Barlow (7), Evans (8), and Marshall (9). Rada (10) devised a combined social and psychiatric diagnostic classification for use with rapists in a clinical setting. In the psychological literature, the motivational aims and needs of the rapist have been described by Guttmacher and Weihofen (11), Gebhardt and associates (12), and Cohen and colleagues (13). Sociologists and criminologists have reported on the issue of power and dominance as factors in rape (14) and on rape as social control (15–18)[1]; Gelles examined power, sex, and violence in marital rape (19).

To date, few reported comparative data have been available on the phenomenon of rape from the experiential standpoint of both the offender and the victim. This paper examines forcible rape through an analysis of separate offender and victim accounts and suggests a clinical typology of rape derived from these data.

METHOD

Written into the General Laws of Massachusetts is a statute (Chapter 123A) that provides for diagnostic evaluation of anyone convicted of sexual assault in regard to the probability of repetition and dangerousness. If it is adjudicated likely that the offender will repeat his offense and in so doing jeopardize the physical safety of his victim, he may be civilly committed for an indefinite period of from one day to life (in lieu of or in addition to a prison sentence) to a special maximum security treatment center operated by the state Department of Mental Health. The clinical and statistical data reported in this paper in regard to the offender are derived from a random sample collected by Groth of 133 convicted rapists committed for clinical assessment to the Massachusetts Center for the Diagnosis and Treatment of Sexually Dangerous Persons.

The victim sample population is derived from a one-year counseling and research study conducted at the Boston City Hospital that included all persons admitted to the emergency service with the chief complaint, "I've been raped." Burgess and Holmstrom clinically assessed the 146 victims of sexual assault according to three categories: rape trauma, accessory to sex, and

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The authors wish to acknowledge the clinical work of Murray L. Cohen, Ph.D., Boston University, on the psychology of rape, which has contributed to their conceptualization of sexual assault.

[1]An abridged version of Bart's paper (18) was printed in the June 1975 issue of *Viva* magazine.

sex-stress situation (20). The 92 adult rape victims from that study comprise the sample for this paper.

The offenses committed by the offender population and those experienced by the victim population were congruent in time, but they were unrelated in that none of the women in the sample were victims of the offenders studied.

The descriptions of the rape assaults given by offenders and victims were analyzed in order to develop a typology of rape. A major intent was to see if the accounts provided by the two groups could be encompassed within one conceptual framework.

RESULTS

Typology of Rape

One of the most basic observations one can make about rapists is that they are not all alike. Similar acts are performed for different reasons or different acts serve similar purposes. Our clinical experience with convicted offenders and with victims of reported sexual assault has shown that in *all* cases of forcible rape three components are present: power, anger, and sexuality. The hierarchy and interrelationships among these three factors, together with the relative intensity with which each is experienced and the variety of ways in which each is expressed, may vary, but there is sufficient clustering to indicate distinguishable patterns of rape. We have found that either power or anger dominates and that rape, rather than being primarily an expression of sexual desire, is, in fact, the use of sexuality to express issues of power and anger. Rape, then, is a pseudo-sexual act, a pattern of sexual behavior that is concerned much more with status, aggression, control, and dominance than with sensual pleasure or sexual satisfaction. It is sexual behavior in the service of nonsexual needs and, in this sense, is clearly a sexual deviation (21).

Power rape. In this type of assault, the offender seeks power and control over his victim through intimidation by means of a weapon, physical force, or threat of bodily harm. Physical aggression is used to overpower and subdue the victim and is directed toward achieving submission. The aim of the assault usually is to effect sexual intercourse as evidence of conquest; to accomplish this, often the victim is kidnapped, tied up, or otherwise rendered helpless.

This type of offender often shows little skill in negotiating interpersonal relationships and feels inadequate in both sexual and nonsexual areas of his life. Because he has few other avenues of personal expression, sexuality becomes the core of his self-image and self-esteem. Rape is the means by which he reassures himself of his sexual adequacy and identity, of his strength and potency.

Since it is a test of his competency, the rape experience for this offender is one of anxiety, excitement, and anticipated pleasure. The assault is premeditated and preceded by an obsessional fantasy in which his victim may initially resist him but, once overpowered, will submit gratefully to his sexual embrace. She then will be so impressed with his sexual abilities that she will respond with wild abandon. In reality, this offender may be handicapped by impotency or premature ejaculation (22). Even if he is not, he still tends to find little sexual satisfaction in the rape. The assault is disappointing because it never lives up to his fantasy.

Often he must convince himself that his victim became "turned on" to him, really wanted sex but could not admit it, and clearly consented nonverbally and enjoyed the sexual contact. However, at some level he realizes that he has not found what he is looking for: something he cannot clearly define is lacking. He does not feel reassured by either his own performance or his victim's response and therefore must go out and find another victim—this time the "right one."

The offenses become repetitive and compulsive. The amount of force used in the assault may vary and there may be an increase in aggression as the offender becomes more desperate to achieve that indefinable experience that continues to elude him. Usually there is no conscious intent on the part of this offender to hurt or degrade his victim. The aim is to have complete control over her so that she has no say in the matter. She will be submissive and gratify his sexual demands.

This rape may be understood as the result of an endogenous developmental psychological crisis precipitated by some perceived challenge from a female or threat from a male that activates the offender's feelings of inadequacy and insecurity (23). Rape is the way in which this type of person asserts his identity, potency, mastery, strength, and dominance and denies his feelings of worthlessness, rejection, helplessness, inadequacy, and vulnerability.

The victim of this assault is typically within the same age group as the offender or younger. Hospital examination generally shows minimum or inconclusive evidence of physical or sexual injury, and clinical evidence of intercourse—presence of motile sperm—may be absent. The victim often reports being questioned by the offender regarding her sexaul life, her reaction to his sexual performance, and her identity, with the implication of expected further contact. The victim is fearful that the offender will attempt to return and is upset about the forced conversation and sexual assault. Her coping behavior is often in the service of trying to talk her way out of the rape or resisting intimidation initially by being assertive and confrontational.

Rapists who fall into the power assault category can be subdivided on the basis of whether the major goal in the offense is assertion or reassurance.

The *power-assertive rapist* regards rape as an expression of his virility and mastery and dominance. He feels entitled to "take it" or sees sexual domination as a way of keeping "his" women in line. The rape is a reflection of the inadequacy he experiences in terms of

his sense of identity and effectiveness. Case vignettes below illustrate this subtype.

Mr. A, a 20-year-old single man on leave from the Coast Guard, picked up an 18-year-old hitchhiker and drove her to a secluded beach. She begged to be let go, but he grabbed her, saying, "You don't want to get hurt, baby—you want to get laid. You want it as much as I do." He forced her to submit to intercourse and then offered to buy her dinner. While out on bail he committed an identical offense. As an adolescent, he had been involved in a number of sexual incidents involving exhibitionism and sexual play with children and was treated at a local mental health clinic. As an adult he had no steady girlfriends and while in the service was living with and being supported by a 30-year-old man in exchange for sexual favors. However, he does not regard himself as a homosexual. He committed two previous rapes for which he was never apprehended; his only other arrests were for motor vehicle violations. Although he is of above-average intelligence, his academic and vocational accomplishments were mediocre, and the only activity he had pursued with any diligence was body-building.

Ms. B is a 45-year-old divorced woman. A former boyfriend came by her home with a bottle of gin and asked her to share a drink with him. She agreed. Later that evening she refused to have sex with him and her account of what happened follows: "He messed me over the street way. I'm not supposed to tell. He said he'd beat me if I tell and he gave me a sample tonight to show me. They work you over to control you so they can have you sexually any time they want. He hit me on the ear, pulled my hair, hit me in the back by my kidneys—very strategic. But it's not the physical part that's the thing. It's mental—to control you."

The *power-reassurance rapist* commits the offense in an effort to resolve disturbing doubts about his sexual adequacy and masculinity. He wants to place a woman in a helpless, controlled position in which she cannot refuse or reject him, thereby shoring up his failing sense of worth and adequacy.

Mr. C is a 24-year-old married man who pleaded guilty to six charges of rape. In every case he approached the victim with a gun in a shopping mall. His fantasy was that the woman would say, "You don't need a gun. You're just what I've been waiting for," and then "rape" him. He kidnapped the women, tied them up, forced them to submit to intercourse, and then questioned them as to whether he was as good as other sex partners they had had. Mr. C had no criminal history other than the sex offenses. His background was unremarkable, but he had never felt that he did particularly well in any area of his life (academic, social, vocational, marital) with the exception of his military service. He dated actively as a teenager and indulged in pregenital sexual play, stopping short of intercourse. He found intercourse unsatisfying, partly because of premature ejaculation and partly because it never lived up to his expectations, although he could not define what was missing. He saw marriage as a solution, but since he could not be sensitive or attentive to anyone else's needs but his own, his marriage floundered. Mr. C discovered he was sterile and began having disturbing homosexual thoughts before his offense. He described the offense as a compulsion—something he felt he must do. Although he realized the consequences of what he was doing, he could not control himself. The six assaults occurred within a 2-month period.

Ms. D, a 21-year-old single woman, had just stepped off a bus on her way home from work and was walking in a well-lighted area of the city. A man approached her from behind, put a knife to her back, pushed her into some bushes, and pulled her sweater over her head so that she would not see him. He raped her vaginally and kept asking her questions such as: "How does it feel? Am I as good as your boy friend?" She said, "He seemed as if he needed to be reassured and so I tried to reassure him. I was lying on my back in the gravel . . . he was odd saying those things . . . then I had to bargain with him to get my clothes back. He also wanted my address and to know if he could come and do it again. I felt if I could convince him I was being honest, he would let me go. He had my pocketbook and would know who I was so I told him where I lived but not the right apartment." The medical record indicated abrasions on lower and upper back, with no other signs of bleeding or trauma.

Anger rape. In this type of sexual assault, the offender expresses anger, rage, contempt, and hatred for his victim by beating her, sexually assaulting her, and forcing her to perform or submit to additional degrading acts. He uses more force than would be necessary simply to subdue his victim. The assault is one of physical violence to all parts of the body: the rapist often approaches his victim by striking and beating her, tears her clothing, and uses profane and abusive language.

The aim of this type of rapist is to vent his rage on his victim and to retaliate for perceived wrongs or rejections he has suffered at the hands of women. Sex becomes a weapon, and rape is the means by which he can use this weapon to hurt and degrade his victim.

This offender displays a great deal of anger and contempt toward women. He sees them as unloving, ungiving "whores and bitches." Sex itself is regarded at some level as base and degrading, and the anger rapist typically finds little or no sexual satisfaction in the rape. He tends to react subjectively to the sexual act with revulsion and disgust, and he may experience difficulty achieving erection or ejaculation during the assault (22).

His relationships to important women in his life are fraught with conflict, irritation, and irrational jealousy, and he is often physically assaultive toward them. His sexual offenses tend to be episodic and sporadic, triggered by conflicts in his relationships to these women (mother, girlfriend, wife), but the anger is often displaced onto other individuals.

The rape experience for this offender is one of conscious anger or sadistic excitement. His intent is to hurt his victim, and his assault is brutal and violent. The motive is revenge and punishment; in extreme cases, homicide may result.

He finds it difficult to explain his assault when he cannot deny it except to rationalize that he was drunk or on drugs. Often the details are lost to his memory because he becomes "blind with rage" during the assault. Satisfaction and relief result from the discharge

of anger rather than from sexual gratification. He derives his pleasure from degrading and humiliating his victim.

Older or elderly women are a particular target victim for this type of rapist, although victims may be of any age. Medical examination generally reveals considerable physical trauma to all areas of the victim's body, often requiring X rays and consultation from other medical specialties. Victims report experiencing the rape as life-threatening, and their symptoms after the rape are disruptive to physical, behavioral, social, and sexual lifestyles.

Victims describe a blitz style of attack (20) or a sudden and dramatic switch in the offender's behavior. Having used a confidence ploy to entrap his victim, the offender then exhibits a behavior change in which he assaults her in a fury of anger. Victims may be physically immobilized and unable to use any coping strategies during the rape (24). Abusive and humiliating language may be used, and "perverted" acts are often a major aspect of the assault.

Rapists who fall into the anger-assault category can be subdivided into two groups on the basis of whether or not the degradation of the victim represents conscious rage or pleasure on the part of the offender.

The *anger-retaliation rapist* commits rape as an expression of his hostility and rage towards women. His motive is revenge and his aim is degradation and humiliation.

Mr. E is a 25-year-old married man and father of four. His mother abandoned the family shortly after his birth, and throughout his life his father reminded him that his mother was a "whore and never trust any woman; they are no good." During his adolescence he became acquainted with his mother and once when she was drunk she exposed herself to him and asked him to fondle her. He fled, terrified. In vain efforts to win his father's recognition and approval, he put a premium on physical toughness. In high school he played sports "like a savage." He then entered the Marine Corps and had an outstanding service record. After his discharge he married (against his father's wishes) and attended college. One day he got into a dispute with his female history teacher over the merits of the Viet Nam war and felt she was ridiculing and humiliating him in front of the class. He stormed out of the room, very angry, thinking "women are dirty, rotten bastards" and went to a bar for a few drinks. On his way to his car he spotted a 40-year-old woman (whom he described as looking older) in the parking lot. He grabbed her by the throat and hit her in the mouth; then he ripped off her clothes and raped her. Before this offense, Mr. E's only criminal record consisted of arrests for gambling, loitering, and drunkenness.

Shortly after midnight, Ms. F, a 33-year-old mother of two, was in bed reading and waiting for her husband to return from work. A man known to the family for 10 years knocked at the door and the 14-year-old son let him in. He said he was leaving town the next day and wanted to say goodbye. Ms. F talked to him from the bedroom and wished him well on his trip. Suddenly the man ordered the boy to his room, grabbed Ms. F, and forced her to go outside to another building. He beat her far more than was necessary to subdue her, choked her, and sexually assaulted her. On follow-up, Ms. F reported considerable physical pain from the beatings. She had pain and swelling of the face, neck, arms, and leg, and she reported loss of appetite, insomnia, nightmares, crying spells, restlessness, and a fear of being followed as she walked home from work. She was unable to carry out usual parenting and household tasks for the first week following the assault. There was tension within the marital relationship, delay in resuming sexual relations, and flashbacks to the assault.

The *anger-excitation rapist* finds pleasure, thrills, and excitation in the suffering of his victim. He is sadistic and his aim is to punish, hurt, and torture his victim. His aggression is eroticized.

Mr. G is a 30-year-old divorced man charged with first-degree murder. His victim, a 20-year-old woman he picked up at a singles bar, was tied to a tree, whipped, raped, sodomized, and slashed to death. Although found to be sane, he claimed he was high on drugs and could not remember what had happened. He had a criminal record that included assault and battery, breaking and entering, nonsupport, and motor vehicle violations. At the age of 17 he had tied a 13-year-old neighbor girl to a bed and assaulted her. He beat his children and burned his wife with cigarettes during intercourse. Shortly after his conviction, Mr. G committed suicide.

Ms. H, a 33-year-old single woman, was returning to her apartment at about 9:00 p.m. when she was suddenly jumped from behind and knocked to the ground. She remembered nothing until she awoke in the assailant's apartment. She was held captive there for 2 days, during which time she reported being continually beaten, raped, and forced to perform fellatio. She said, "He bit me and burned me. He had pictures of rattlesnakes on his wall." The medical exam revealed multiple injuries to her face and eye, multiple bruises and lacerations to her body, and human bite marks and cigarette burns on her chest. Ms. H was hospitalized for several days.

Rape, then, rather than being an expression of sexual desire, in fact is the use of sexuality to express issues of power and anger. In every case of rape all three factors coexist but their relative intensities and interrelationships vary. The incidence of each type of rape assault in our respective samples is presented in table 1.

Overall, power rapes outnumber anger rapes: of the 225 offenses studied, 146 (64.9%) were classified as power rapes and 79 (35.1%) as anger rapes. The anger-retaliation assault was the most frequent type of offense in the offender sample, but it ranked only third in the victim sample. One possible explanation is that the incidence of anger-retaliation rape is in fact lower than the incidence of power offenses, but since the physical evidence of anger assault is more apparent, the likelihood of conviction is greater. Consequently the anger-retaliation rapist may be overrepresented in the convicted-offender sample. The anger-excitation offender seems to be the least prevalent type among both samples.

TABLE 1
Incidence of Sexual Assaults Classified According to Typology

Type of Assault	Sample					
	Offender (N=133)*		Victim (N=92)**		Total (N=225)	
	Number	Percent	Number	Percent	Number	Percent
Power						
Assertive	50	37.59	48	52.17	98	43.55
Reassurance	22	16.54	26	28.26	48	21.33
Anger						
Retaliation	53	39.84	14	15.21	67	29.77
Excitement	8	6.01	4	4.34	12	5.33

*For those convicted offenders who also had a codefendant (N=21) each rapist is categorized separately.
**For those victims confronted with more than one assailant (N=33), the rape is categorized for the initial assailant.

DISCUSSION

The proposed conceptualization of the issues of power, anger, and sexuality in rape has several implications. Clinical work with offenders and victims indicates that the initial impact of rape is not sexual for either group. Although the act is sexual, what is traumatizing to the victim is the life-threatening nature of the assault, her helplessness and loss of control in the situation, and her experience of herself as the object of her assailant's rage. This is important because the etiology of the victim's trauma is the offender's pathology. Rape is more than an illegal act and more than an extreme of cultural role behavior. From a clinical point of view, it is important that rape be defined as a sexual deviation and that the pathology of the offender be recognized.

The clinical typology of offenders we have presented offers one approach to differentiating offenders with regard to identification, disposition, treatment planning, and prognosis. Further research is warranted to determine whether the trauma reaction varies according to the motivation of the rapist and to ascertain whether conviction rates differ for the four types of offenders.

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Problems for Women in Psychiatric Residency

BY ELISSA P. BENEDEK, M.D., GAIL BARTON, M.D., AND CHRISTINE BIENIEK, M.D.

Some of the basic problems confronting women in psychiatric training are reflected in four major areas: supervision, administration, research, and peer relationships. The authors recommend a residency seminar on female psychology, flexible-schedule residencies, inclusion of more women in all phases of the profession, reeducation in female psychology for supervisors, more research opportunities, the availability of male and female mentors, and training program encouragement of peer support; these suggestions would help overcome the problems and improve training programs for women and men.

WOMEN PSYCHIATRIC residents encounter special problems in the areas of supervision, administration, research, and peer relationships. The similarity and intensity of the problems have stimulated this commentary, which highlights and describes common difficulties of women supervisors and trainees in psychiatry. We hope this paper will foster greater understanding and remedial action toward constructive change.

The literature, like the social setting in the United States today, emphasizes the differences between men and women entering medicine. For example, Cartwright (1) says that women are motivated toward medicine because of a desire for individuation; yet they share with men the additional motivations of encouragement by others and personal life-long interest in science. Nadelson and Notman (2) note that this motivation for individuation becomes blunted at the point of specialty choice, since women weigh this motivation against a "desire to feel accepted and needed rather than to continue to fight for recognition in a specialty such as surgery, where hostility to women is considered great."

Macoby, Jacklin, Hoffman, and others (3–5) report that women are more affiliative by nature than men. We concur and suggest that because of this characteristic, women tend to gravitate toward psychiatry. It is seen as a people-oriented rather than a technique-oriented specialty, and trainees expect affiliative needs to be met by peers, teachers, and supervisors, as well as patients. Unfortunately, this supposed people-oriented specialty seems unable to recognize that flexibility in role definement, didactic course content, supervision, and assignment would allow women to function simultaneously as psychiatric trainees, women, wives, and mothers (6). Therefore, the need for affiliation that initially drew women into psychiatry is largely unmet during training.

We suggest that although these problems exist, they are not inherent in residency training and are not insoluble. Awareness of such difficulties and concrete attempts to solve them would enrich the professional and personal lives of male and female psychiatric trainees, supervisors, and patients.

SUPERVISION

Supervision forms the core of psychiatric training and provides the trainee the opportunity to learn by identification, modeling, and education. The paucity of female supervisors available to trainees is undisputed (7). Moreover, both male and female supervisors tend to neglect important issues related to femaleness that arise between resident and patient and resident and supervisor. This discussion will be limited to those issues which arise between female residents and supervisors of both sexes. It serves only to illustrate a few problem areas and does not purport to be an extensive discussion of supervisory issues.

Unrecognized and unresolved conflicts between female resident and female supervisor may include problems about the handling of gender identity, as well as aggression and sexuality, between therapist and patient and therapist and supervisor. Preoccupation with the patients' dress, hairstyle, and other externals may overshadow concern about the true problems and roles of a particular patient, especially when these problems deal with the changing roles of women. For example, some senior female supervisors still encourage trainees to regard as normal in female patients the absence of a desire for home, husband, and children and the presence of an interest in career, education, and profession which may reflect their own orientation being imposed on the patient and the supervisor. Issues involving homoerotic transference and countertransference are often unrecognized or recognized and ignored because neither partner in the supervisory

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dyad wishes to explore these issues; neither of them may have completely resolved these issues as yet. This is in marked contrast to the ease with which supervisors explore heterosexual transference and countertransference (8). In addition to disagreements about patient care, the relationship between a female supervisor and trainee may involve competition; the supervisor may see the trainee as a daughter who will achieve educationally and professionally what the supervisor is perhaps too old to do.

Problems with male supervisors may be similar or different. Male supervisors tend to view the woman trainee as a stereotypic female, that is, either warmly nurturant and passive or cold and castrating. For example, one young female trainee was rebuked by her young male supervisor for not being as warmly outgoing as his mother was. Another rather assertive woman resident was confronted by a generally passive and insecure male supervisor about her "castrating behavior" toward him. When such a stereotype is projected upon her, a woman's individual personality characteristics can be under-recognized, making a real and unique relationship difficult. Simultaneously and paradoxically, supervisors often seem bent on creating another stereotypic resident, one who is aggressive, strong, and dedicated to psychiatry to the exclusion of other important interests.

The male supervisor may often feel and act either overtly or covertly seductive with the female trainee. It is the rare supervisor who can reflect and handle his feelings appropriately if they emerge in supervision. Trainees comment on supervisors' patting them, holding their hand, asking them out, and behaving in even more flagrantly seductive ways. When confronted with problems of this sort, trainees are often reluctant to turn to other supervisors or colleagues for help, perhaps partly out of fear that they will be accused of somehow "inviting" such behavior. This situation is somewhat analogous to the manner in which rape victims in our society have been held responsible for encouraging their attackers.

Male and female supervisors frequently block the assignment of adolescent male patients to female trainees. Although no such restrictions are imposed on male residents treating female adolescents, some supervisors reason that the female trainee will be unable to deal with the sexual fantasies aroused in her patients and that acting out will be stimulated by the transference. One young female trainee was told by her experienced supervisor, an older woman, that she would not be able to treat adolescent males "until your hair is as white as mine." Female trainees can thus be denied the experience of treating male adolescents in a supervised situation. It is unusual for a supervisor to perceive the relationship of a female resident with a male patient as potentially constructive and therapeutic. One trainee reported that although she had announced her intentions of training in child and adolescent psychiatry early in her residency, it was impossible for her to have an experience on the adolescent ward. There were other female trainees in the department at that time, and the supervisor in charge of the adolescent ward flatly refused to allow more than one female trainee on the ward at a time. He felt that more than one female resident would "excite the boys."

Supervisors often are extremely solicitous and overprotective of women trainees, thereby excluding them from consideration for committee work, team leadership, or teaching opportunities with students from different health disciplines. One trainee reported perplexedly that although the department was short of resident staff to teach medical students, no one asked her to assume such a role. When she volunteered, she was confronted with "Are you sure you can do it?"

Typecasting of women residents as housewives and mothers is so strong that frequently when a woman resident is pregnant she is urged to discontinue her residency to care for her unborn child. One supervisor took a pregnant trainee to view a movie concerned with the effects of maternal deprivation on children. The trainee's own ideas about child rearing and career planning were not solicited, and her expressed wishes to discuss the issues openly were ignored. Ironically, when another woman trainee asked for a leave of absence to be with her children during their summer break, her request was denied. Individuation, both in completing a residency and supervising one, seems far in the future.

It is the exceptional supervisor who can discuss femaleness in supervision. When he/she can focus on such topics as pregnancy, menstruation, women in careers, divorce, courtship, and parenting, it adds a rewarding dimension (and from the woman's vantage point an *essential* one) that enables the resident to learn adequately about relationships with patients, families, staff, and colleagues.

ADMINISTRATION

Sexual role stereotyping also confronts women residents who desire training in administration. They are often invited to do the committee work associated with social occasions, decorating, or libraries but seldom are they considered appropriate candidates for chief resident, membership on a policy committee or on a committee to study program curriculum changes, or specialized course work in management.

The lack of a female role model among psychiatric administrators is very disheartening to the would-be administrative trainee. The female administrator needs female role models to most appropriately learn how to lead or to be heard in a meeting; how to create a leader-follower relationship, female to male and female to female; and how to handle the nuances of collegial relationships, female to male and female to female.

The feeling that one must learn these techniques and be, in a sense, an activist minority spokesperson may be eased somewhat when admission policies to medical school become more equalized and when con-

sciousness-raising experiences enable more men to accept women in leadership roles outside the home. Because of challenges to authority in general, female administrators have to struggle all the harder to survive. Role models of women in administrative posts exist if women are willing to look at the other health professions, especially nursing and social work. However, even in these predominantly female fields, the top posts quite often have been given to men. In addition to the heavy responsibilities of the administrative position, a female administrator-doctor faces a constant barrage of unacceptance or disbelief when she contacts others professionally for the first time (i.e., secretaries, ticket agents, lawyers, and other physicians).

Certain biases against a female administrator-psychiatric trainee will not likely pass without a period of consciousness raising. For example, supervisors assign women trainees to the less desirable posts, where prestige will be less likely to evolve. Women trainees are almost always questioned about the quality of their work and whether the responsibility being assigned is too much, whereas the competency of men in comparable posts is rarely doubted. The years of experience a candidate brings to an administrative position are usually taken into account when a man is assigned a task, but the woman administrator-trainee soon learns she must actively remind people that she is responsible, well trained, competent, interested, and available.

If women are going to project an image of competency, they will have to learn early in their training as administrators to speak up concisely (not to fumble with microphones but to handle them with aplomb), as well as to manage the financial and business aspects of administration.

These techniques of the administrator are usually conveyed in an informal, father-son relationship from one generation of administrators to the next. Until there are enough women in administration to make mother-daughter dyads of role modeling possible, the nourishing of a female administrative-trainee will have to be done by concerned, dedicated male administrators. These men will have to be willing to take the jibes of their fellow administrators, who will frequently allude to the sexual implications of the relationship. The administrator-mentors must become more aware that most male administrators are quite often quite intolerant of "this *woman* who thinks she's the boss." Both male and female trainees need to be aware that this may happen so that the male trainee will be less likely to feel so put out if his superior is a woman and so that the female trainee can be forewarned that she may be challenged. Frequently, disagreements develop when the female administrator-trainee tries to assert her role in the hierarchy only to be confronted with extreme hostility, resentment, and cries of unfair treatment by male peers. Too often the resulting clashes are passed over by the mentors as a woman's "bad days," "hysteria," or "sibling rivalry." They do not give opportunity for discussion, consciousness raising, or resolution of the problem because they lack understanding of the real issue—the conflict between a woman in an administrative role and a male peer with no experience or training in this type of relationship.

RESEARCH

The woman trainee in psychiatric research also finds that sex role stereotyping puts her at odds. She is perceived as much more capable of the clinical role, which is seen as nurturing, than the research role, which is more scientific, logical, competitive, and aggressive. The relative paucity of distinguished women researchers as role models in this field is alarming until one looks at the adverse conditions for training a woman in the field. Because there are relatively few research-oriented residency programs, geographic incompatibility may arise for women wishing to pursue this type of career.

Women may also have particular difficulties in finding a research mentor. Most psychiatrists (and other scientists) who go on to make significant research contributions have had, early in their research development, a close relationship with a senior research mentor who encourages, stimulates, and constructively criticizes and who may suggest research areas or offer collaboration in his or her own work. If both are male, this relationship often takes on a close father-son quality. There is considerable intellectual and personal excitement in developing and discussing ideas—and later, often some ambivalence in the growth phase of "challenging the old man" (7). These relationships also have rich and interesting transference-countertransference aspects (whether they are ever explicitly discussed or not).

However, when the younger researcher is female and the older one is male, there may be special complications. If a father-daughter relationship exists between a male senior researcher and a female resident, the intimacy of sharing gestating ideas can frequently have erotic overtones for either or both of them. Moreover, if the mentor is middle-aged, he may be involved in a mid-life crisis of his own, which, in our society, is frequently handled by seeking overt sexual relationships with a younger woman. This can increase the tension related to the resident-mentor relationship.

If the female resident finds a woman mentor, this relationship, too, has its problems. Traditionally, mothers in our society have taught daughters to limit their aspirations for individual accomplishments, warning them of the pitfalls and hardships ahead and teaching them how to find trustworthy men on whom to lean. Although these may be adaptive strategies in social life, they are not research attitudes. A woman mentor may not be able to adequately prepare the female resident for special difficulties in getting faculty positions and research grants. More dynamically, she may have her own difficulties in these areas (e.g., the

use of denial) so that she either cannot help the younger woman with the same problems or finds her own denial threatened painfully. A special kind of envy may develop if a woman resident can resolve conflicts with male colleagues without passively submitting. Occasionally, one may find an older female mentor encouraging her student to act out her conflicts with male colleagues—conflicts that the mentor was forced to deny. Despite these difficulties, however, many young women have been very effectively encouraged by mentors of both sexes.

The woman resident, as a consumer of research, finds that many of the authorities cited in literature seriously question whether she has the ability to understand them and join them as colleagues. They describe women as narcissistic, having a weaker superego, more passive, or more masochistic. These negative descriptions are not likely to stimulate her to delve more deeply into psychiatric literature or to pursue role modeling toward that type of research.

PEER RELATIONSHIPS

The female resident frequently finds herself alone in her peer group, facing all the very real problems mentioned in the literature by Wolman (9). In a training center athletics is a traditional outlet for social interaction among resident-resident, resident-senior staff, and senior staff-senior staff. However, the woman resident is usually given the clear message that it is for men only—women are not sports participants anyway. The recent upsurge in interest in tennis has changed this attitude slightly, but it is still strong in regard to paddleball, softball, and skiing. Study groups among peers tend to be segregated sexwise also, excluding the solitary woman resident. Should the woman trainee try to participate in the sessions, the kidding and innuendos that go on highlight the awkwardness and cancel the advantages of group study. The tendency to nominate the other "guys" for committee and prestige positions and to ask women to plan social events in the residency program further isolates the women trainees; recognition of the woman trainee's individual skills is still buried in sex stereotyping.

This solitariness and lack of peer support poses a more critical problem for women than for men when one considers the importance of affiliative need in female development. The professional woman alone in a peer group has no one with whom to build affiliative bonds. If she is, as Hoffman (4) postulates, developmentally more affiliative than competitive, she finds some of her most basic psychological needs thwarted and unmet. Additionally, because sexual feelings are part of our daily lives and because residents are learning to deal with sexual transference, these feelings occur between male and female peers in training. Unfortunately, this can sometimes lead to acting out by both male and female residents. However, if residents avail themselves of the unique opportunities to more closely explore these feelings, it can lead to strong bonds of friendship.

Psychiatric residency training can be a lonely exploration of one's self if one is without a cohort or peers. The female resident who is alone in a group complains of feeling isolated, lonely, and depressed; while such feelings may be due to the stresses of the training program and/or interpersonal dynamics, often the training situation itself is such that the resident is alone or isolated.

RECOMMENDATIONS

We hope that highlighting the problems of women in psychiatric residency will stimulate improvement of training programs. Men in psychiatry will benefit from the improvements as well: suggestions for changing the residencies should create an atmosphere conducive to learning about both women and men.

We suggest the following actions to overcome the problems:

1. A seminar on female psychology should be part of the standard curriculum. It should involve surveying the existing literature, noting the lack of a researched data base and the obvious inconsistencies, so that some residents will be stimulated to enrich the field by researching female psychology.
2. Flexible-schedule residencies should be created, not just in a few geographic areas but nationwide. Then women could choose programs that are more realistic for them in regard to where they wish to locate permanently and to the amount of time they feel is important for child rearing. This flexibility should also allow men in medicine the opportunity to be more involved in child rearing, in contrast to earlier attitudes, where the father was expected to work long hours and sacrifice his and his family's emotional needs for those of his patients during his training or career.
3. More women should be admitted to medical schools, psychiatric residencies, supervisory positions, administrative posts, and research consortiums. Having women in such posts and training them for these positions will create an environment conducive to accepting women in these roles, providing role models for other women, and creating a cadre of women experts from which to learn.
4. In-service education is necessary for supervisors, administrators, and teachers in psychiatry to help them see that they do tend to view women stereotypically rather than individually. Most need to explore women's roles beyond those of wife and mother. They also should explore what roles men might play beyond that of provider, such as participating more in child rearing.
5. More specific data collection, research, and training around the whole aspect of supervision of psychiatric trainees is needed. All too often a person becomes a supervisor simply because he/she is in the care-delivery system and not because he/she is partic-

ularly skilled at teaching or has specific skills or knowledge. Supervisors should have as part of their usual armamentarium a thorough grounding in female psychology. They should be willing to discuss this with their male and female trainees and to work through any male and female transference and countertransference issues that might develop in the course of supervision.

6. More specific attention should be given to training women psychiatric administrators so that they learn requisite business, financial, and technological skills as well as clinical skills.

7. All training centers for psychiatry should have research available as part of the training program so that geography is not a barrier to a potential researcher. Otherwise, women, who tend not to move themselves and/or their families to the few areas where research opportunities are currently available, become excluded from contributing to research.

8. Research trainees should have both male and female mentors available. Affirmative action may be necessary to implement this. Since there is an underrepresentation of women in this area and too few role models, research centers will have to be more willing to encourage female trainees into more research-oriented roles.

9. Training programs may well want to pay more attention to providing peer support. Residency-staff social, athletic, and study get-togethers should not be left to chance. Peer support means the difference in camaraderie, a sense of belonging for all trainees, an esprit de corps for the institution, and the building of lasting friendships and professional relationships.

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Phenytoin Treatment of the Binge-Eating Syndrome

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Nineteen subjects completed a 12-week double-blind crossover study comparing phenytoin with placebo in the treatment of severe binge eating. In the placebo-phenytoin sequence the number of binges decreased during phenytoin treatment from both placebo and pretreatment levels. However, in the phenytoin-placebo sequence, not only did phenytoin reduce the number of binges from pretreatment levels, but there was no reversal of this effect during the placebo period. This sequential effect was unexpected and is still unexplained. The mechanism of phenytoin action and the significance of EEG abnormalities found in 7 of the subjects remain uncertain.

THIS STUDY was stimulated by a report on the effectiveness of phenytoin (diphenylhydantoin, Dilantin) in treating 9 of 10 persons with "compulsive eating" problems (1). The purposes of this study were to gain a clearer understanding of the clinical syndrome of binge eating and to test the efficacy of phenytoin treatment in a controlled clinical trial.

METHOD

Subjects

The criteria for the diagnosis of binge eating were as follows:

1. Impulsive, unpredictable, episodic, uncontrolled, and rapid ingestion of large quantities of food over a relatively short period of time.
2. Termination of the episode only when a point of physical discomfort has been reached (e.g., abdominal pain, and feelings of nausea or distention). Self-induced vomiting supported but was not required for the diagnosis.
3. Subsequent feelings of guilt, remorse, or self-contempt.

A frequency of at least one binge per week with no binge-free intervals of longer than three weeks during the previous year was also required for inclusion of subjects in the study.

Twenty subjects were recruited from the following three sources: 1 from the Stanford Student Health Service, 4 from the Stanford Eating Disorders Clinic, and 15 from newspaper advertisements. Since binge eating has been described only occasionally and the frequency of its occurrence has never been estimated, we will briefly describe our selection procedure to provide a first such estimate. The subject from the student health service was the only person known to have complained of binge eating out of approximately 600 men and women of college age seen during a 5-month recruiting period. The four binge eaters from the eating disorders clinic were drawn from the files of approximately 200 persons, primarily obese, who had been seen over the preceding 2 years. The newspaper advertisement ran for 11 consecutive days in the San Jose *Mercury* (circulation 207,000) and the Palo Alto *Times* (circulation 44,000). There were 242 telephone inquiries from this ad, of which 201 were screened out; 152 subjects did not meet the diagnostic criteria and 49 had only rare episodes of binge eating. Of the 41 persons who were personally interviewed, 26 were screened out. Fifteen were eliminated because the frequency of binge eating was less than demanded by our criteria and 11 for other reasons, primarily insufficient interest. Fifteen of the subjects answering the advertisement met all of the criteria for inclusion in the study and entered treatment.

Careful medical and psychiatric evaluations were carried out by two psychiatrists who ascertained the suitability of subjects for the study. The nature of the study was fully explained to the subjects prior to obtaining their informed consent. Because of earlier reports of EEG abnormalities in binge eaters, each subject received a waking and sleep EEG with nasopharyngeal leads (1–3). Initial laboratory tests included a complete blood count and a chemical screening battery (SMA-12).

All of the subjects were women, and they had a median age of 26 years (range, 17–42), as shown in table 1. Using the Metropolitan Life Insurance Company's

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TABLE 1
Description of Sample of Binge Eaters

Sequence and Subject	Age (years)	Age of Onset (years)	Initial Weight (kg)	EEG Findings
Phenytoin-placebo				
1	23	21	50	Normal
2	36	19	72	Small sharp spikes
3	28	17	57	Normal
4	26	16	65	Normal
5	25	13	75	Normal
6	17	12	64	Normal
7	21	11	65	Small sharp spikes
8	37	26	73	Bitemporal slowing, L>R
9	33	13	112	Normal
10	40	25	75	Small sharp spikes
Placebo-phenytoin				
11	29	24	40	Normal
12	42	18	89	Diffuse polyphasic spike and slow wave discharge
13	18	15	52	Normal
14	32	20	63	Normal
15	31	13	58	Normal
16	37	24	52	Normal
17	25	12	83	Normal
18	26	9	66	Small sharp spikes
19	19	15	57	Normal
20	20	18	54	Diffuse spike and slow wave discharge

height-weight tables, we found that 10 subjects were of normal weight, 4 were overweight (10–20% overweight), 5 were obese (more than 20% overweight), and 1 had active anorexia nervosa (20% underweight). Two subjects had a past history of anorexia nervosa, and 2 (including the active anorexic subject) reported problems with alcohol. Other than these difficulties, the subjects were in good physical health, worked or attended school, and showed no evidence of emotional disturbances except that associated with their eating binges.

The onset of the eating binges occurred at a median age of 16 years (range, 9–26), with a median duration of 11 years (range, 2–24) and infrequent binge-free intervals. The median frequency of the binges at the onset of treatment was 3–4 per week (range, 1–7). Most subjects had undergone many treatments for their binge eating, including diets, medications, hypnosis, and commercial or self-help weight control programs. Seven had been in psychotherapy. No treatment had been successful. Descriptive data about the subjects are presented in table 1.

Procedure

Active medication and placebo were administered for 2 6-week periods, each according to a double-blind crossover design. One 6-week period involved treatment with a placebo, the other with active medication. Subjects were randomly assigned to each protocol. Thus there were 2 treatment groups: a phenytoin-placebo sequence and a placebo-phenytoin sequence. Medications were prepared in sets of 6 bottles for each subject, each bottle covering a 2-week treatment interval. All bottles and capsules looked alike. The initial dosage was 1 capsule of 100 mg of phenytoin or placebo 3 times a day.

The subjects were seen weekly by a psychiatrist who did not know which medication they were taking. Each week the psychiatrist reviewed with the subjects the individual forms that they had completed at the time of each binge. The forms included a rating of the intensity of the binge as recorded on a 10-cm visual analogue scale. The psychiatrist weighed each subject, noted any changes in mood, behavior, and eating patterns, and checked for any sign of adverse drug reaction. At no time were instructions in dieting or weight control given, nor was there any psychotherapy.

Serum phenytoin levels and routine hematological studies (complete blood count and liver function tests) were obtained every 2 weeks and the results monitored by another member of the research team. This medical monitor attempted to keep the phenytoin levels in the range of 10–20 μg/ml by requesting appropriate changes in dose. Similar dosage changes were ordered during the placebo period to keep the treating psychiatrist as uninformed as possible.

RESULTS

Nineteen of the 20 subjects completed the study. One subject (number 20) developed a generalized urticarial reaction to phenytoin and had to be withdrawn from the study. Twelve subjects achieved final serum phenytoin levels of 10–20 μg/ml and 5, levels of 5–10 μg/ml. Two subjects (numbers 9 and 19) had phenytoin levels consistently below 3 μg/ml, which raised the possibility of noncompliance. Their data were included in the analysis since they completed the study before the situation was corrected.

The data were first analyzed by comparing the mean number of binges per week estimated by subjects (historical control) and those during both experimental periods (see table 2). In the phenytoin-placebo sequence, the mean number of binges per week was significantly decreased during phenytoin treatment to 2.4 ± 1.9, compared with the historical control of 3.8 ± 1.4 ($t=3.66$, $p<.01$). However, when these subjects then changed to placebo, their improvement continued so that no significant difference was found between the phenytoin and placebo treatment periods. In the placebo-phenytoin sequence, the mean number of binges

TABLE 2
Results of Treatment with Phenytoin and Placebo

Sequence and Subject	Estimated Binges per Week	Binges per Week	Binges per Week	Clinical Degree of Change
Phenytoin-placebo		Phenytoin	Placebo	Phenytoin versus pretreatment estimated binges
1	3.5	0.8	2.0	Marked
2	4.0	0.8	3.7	Marked
3	2.5	2.5	3.0	None
4	4.0	3.2	2.5	Slight
5	3.0	2.0	1.2	Slight
6	7.0	7.0	5.7	None
7	5.0	2.8	4.5	Slight
8	3.5	1.5	0.3	Moderate
9	2.5	0.8	0.5	Moderate
10	2.5	2.5	0.5	None
Placebo-phenytoin		Placebo	Phenytoin	Phenytoin versus placebo
11	3.0	5.2	5.5	None
12	2.0	2.3	0.0	Marked
13	3.5	3.3	1.0	Marked
14	5.0	1.3	1.3	None
15	3.0	3.0	0.5	Marked
16	1.0	1.3	0.5	Marked
17	7.0	7.0	4.7	Slight
18	3.5	5.3	4.2	Slight
19	5.5	3.7	2.0	Slight

per week during the placebo period (3.6±1.9) was not significantly different from the historical control (3.7±1.8); however, there was a significant decrease during the phenytoin treatment to 2.2±2.0 ($t=3.98$, $p<.01$). Differences between the phenytoin-placebo sequence were compared with the placebo-phenytoin sequence. The former sequence showed significantly fewer binges ($t=2.64$, $p<.02$) than the latter, indicating that an unexpected carryover effect was present in that sequence.

Individual data were assessed by comparing the number of binges during phenytoin treatment with that of the control period. For the placebo-phenytoin subjects the placebo period served as the control, while for the phenytoin-placebo subjects the pretreatment estimate of binge-eating frequency was used because of the carry-over contamination effect of the placebo period. If the number of binges was reduced by 75% or more during phenytoin treatment, the subject was considered to be markedly improved; reduction of 50% to 74% was considered moderate improvement; reduction of 20 to 49%, slight improvement; reduction of less than 20%, no change; and an increase of 20% or more was considered to represent worsening.

As can be seen in table 2, 2 subjects in the phenytoin-placebo group were markedly improved on phenytoin, 2 were moderately improved, 3 were slightly improved, and 3 showed no improvement. In the placebo-phenytoin group 4 subjects showed marked improvement, 3 were slightly improved, and 2 showed no improvement.

Subjects whose binge eating was markedly reduced during treatment with phenytoin reported more self-control, a marked improvement in self-esteem, less preoccupation with eating, and movement toward more normal eating habits.

There were no consistent changes in weight or intensity of binges during the study.

Definitely abnormal EEGs were seen in only 3 subjects (table 1). In 4 others "small sharp spikes" were noted; this is a controversial pattern of questionable significance (4, 5). Excluding one patient who had a definite EEG abnormality and was withdrawn from the study, the remaining 6 subjects with abnormal EEGs had the following clinical responses: 1 showed no improvement, 2 showed slight improvement, 1 showed moderate improvement, and 2 showed marked improvement. These improvements were similar to those experienced by subjects without EEG abnormalities, and thus EEG abnormalities were not correlated with treatment response. Since the EEGs were not obtained close to the time of an actual binge episode, these abnormalities may be unrelated, or we may have found normal EEGs in subjects who might have shown abnormalities on other occasions.

We examined the clinical and experimental data for variables that would predict favorable or unfavorable treatment response. Only two items seemed to predict outcome: length of binges and history or presence of anorexia nervosa. Short duration of binges (one hour or less) was uniformly associated with a good response. A history of anorexia nervosa was associated with no improvement in both cases, as was similarly found in a recent single case report (6). The following items did not predict treatment outcome: EEG findings, phenytoin blood levels, weight, frequency of binges, night eating, frequent snacking, eating continuously during the day, eating at regular mealtimes, and postbinge emesis.

DISCUSSION

The report that stimulated this study was an uncontrolled trial of phenytoin with what was called "compulsive eating" (1, 3). Our study was a controlled clinical trial of the effects of phenytoin on what we prefer to call "binge eating," a more descriptive term for what we believe to be the same syndrome. The reasons for this preference have been described elsewhere (7). A brief description of the syndrome follows.

The binge-eating syndrome consists of episodic, uncontrollable, rapid ingestion of large quantities of food over relatively short periods of time. It occurs while the person is alone and has an air of secretiveness. The food is usually consumed rapidly with little or no chewing. The foods eaten tend to be sweet and starchy

items that lend themselves to rapid consumption. Often there is little preparation of the foods and they are eaten directly from their containers, often without the usual preparation such as thawing or heating. As the episode of binge eating progresses, there is decreased selectivity of food items, and unusual food combinations or foods not generally enjoyed are then consumed. If food runs out before the binge terminates, the person will seek out additional food. The binge usually lasts 1–2 hours but can be as brief as 20 minutes or as long as an entire day. The binge is terminated by severe abdominal discomfort, vomiting, or sleep. Social interruption usually temporizes a binge, which then continues when the intruder leaves.

Immediately after a binge, the person experiences strong self-contempt and guilt. There is a resolve not to binge eat again. A strict diet is begun. Normal eating patterns are rarely achieved and the person becomes preoccupied with food, occurrence of binges, weight control, and dieting. The onset is in adolescence or early adulthood, and the syndrome is more prevalent among females than males. There may or may not be an associated weight problem, depending upon the degree of regurgitation immediately after a binge and the success of dieting between episodes. With many individuals the course appears unrelenting and unresponsive to a wide variety of therapies. These findings support an earlier contention that binge eating is a distinctive type of eating pattern abnormality (7, 8).

Our study intentionally selected persons with frequent binges, omitting a larger number with less frequent binges; thus milder forms of the syndrome were excluded. Judging from the large number of these milder cases and the relative ease with which we located our subjects, the syndrome is probably not rare. The very secretiveness and guilt surrounding it may prevent more binge eaters from identifying themselves.

The efficacy of phenytoin as a treatment was considerably less in this study than in an earlier one in which 90% of the subjects returned to normal eating (1). This discrepancy could have resulted from the inclusion of more seriously afflicted subjects, the elimination of placebo response, and the shorter duration of treatment employed in our study.

Our results were further confounded by the effect of treatment sequence, in which there was an unexpected persistence of treatment effect in the placebo period for the subjects in the phenytoin-placebo sequence. The action of phenytoin terminates soon after it is discontinued, and during periods of placebo treatment, virtually all subjects had no detectable plasma concentrations of phenytoin. Thus a prolonged drug effect is most unlikely. When the data from the placebo phase of the phenytoin-placebo sequence were examined at successive 2-week intervals, we detected no progressive increase in frequency of binges over time, which sustains the impression that a residual, but slowly disappearing drug effect was not present. One may speculate that a learning effect is created once the pattern of binge eating is interrupted. In any case, this sequence effect will have to be considered in planning any future controlled trials of drugs in this disorder.

Two recent reports are relevant to the present findings. One report (9) described the response of transvestite behavior of 20 years' duration in a 38-year-old man to small doses of phenytoin. As in the current study, there was no recurrence of problematic behavior after discontinuation of medication. (The patient was followed for a 20-month period.) The second report (10) described the successful treatment of binge eating by purely behavioral measures. These two studies suggest that, whatever the initial mechanism of action of phenytoin in controlling binges, learning may play an important part in their continuing control.

Treatment did not result in consistent weight changes, which may be explained by the short duration of treatment, our lack of emphasis on weight loss, and the majority of normal-weight subjects. Our experience suggests that in the overweight and obese population, binge eating is an infrequent problem.

The etiology of this syndrome remains speculative. Its episodic, uncontrollable nature and occasional favorable response to phenytoin suggests a possible variant of psychomotor epilepsy. The few EEG abnormalities, while epileptiform in morphology, do not correlate with treatment response and are of unknown significance. A hypothalamic etiology has been proposed and is based largely on the interpretation of 14- and 6-per-second positive spikes as abnormal (1, 3). Most electroencephalographers, however, consider these spikes to be normal variants (11–13). The existing data are too inconclusive to support a claim of hypothalamic etiology, and other alternatives must be considered.

The mechanism of action of phenytoin in binge eating is unclear, but it resembles its anticonvulsant action, i.e., the frequency of binges but not their intensity is diminished. However, three other features suggest that at least conventional anticonvulsant activity did not account for the binge-reducing activity of the drug: the effectiveness of phenytoin in preventing binges at serum levels as low as 5 μg/ml, the small minority of persons with abnormal EEGs, and the lack of correlation between EEG abnormality and treatment outcome. This medication is reported to have a wide variety of properties, other than anticonvulsant action, that may play an important role, thus suggesting the usefulness of future comparisons between phenytoin and drugs with nonspecific sedative action (14, 15).

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ADDENDUM

Since completion of the original study, all but 3 subjects who could not be located (patients 9, 11, and 19) were followed over the subsequent 18-month period. Their data provide the first longitudinal study of a group of binge eaters.

Only the 6 subjects who showed marked improvement during phenytoin treatment were given the option of continuing treatment on a supervised, open-trial basis. Four subjects elected to continue treatment, while 2 decided not to for personal reasons. Of the 4 subjects who continued treatment, 2 (patients 1 and 12) had no further episodes of binge eating. Two (patients 2 and 15) noted the return of their binge-eating pattern during the first 2 months and discontinued treatment. At the present time they continue to have binges at their estimated pretreatment frequency. The 2 subjects who did not elect to continue treatment (patients 13 and 16) also continue to have binges at their estimated pretreatment frequency.

Of the 14 subjects who were not treated during the follow-up period or who discontinued treatment during the first 2 months, there was a small tendency ($p<.05$, Mann-Whitney U Test) for binges to be less frequent than during the pretreatment period, but the meaning of this difference is not clear. Eleven subjects reported a decreased frequency of binges, 1 reported a slight increase, and 2 subjects showed no change. Much of this effect was contributed by 3 subjects who showed marked decreases in binge frequency. The number of binges per week for patient 14 decreased from 5.0 to 0 after a divorce, for patient 5 from 3.0 to 0.5 after a particularly meaningful encounter group experience, and for patient 2 from 4.0 to 1.0 for reasons that are not clear.

Did the carry-over effect persist after the end of the active study period, and did it contribute to the decreased binge frequency during the follow-up period? The answer to both questions is probably no. Consider first the 5 subjects in the phenytoin-placebo sequence who apparently showed the carry-over effect (patients 1, 4, 5, 8, and 9). The data on 2 subjects are noncontributory; patient 1 continued on phenytoin, and patient 9 was lost to follow-up. Two (patients 4 and 8) are binge eating at pretreatment levels, and the decreased binge frequency of patient 5 seems adequately explained by her encounter group experience. It is difficult to interpret the results from the placebo-phenytoin sequence. Three phenytoin responders (patients 13, 15, and 17) have returned to pretreatment rates of binge eating, while 2 responders (patients 16 and 18) have maintained lower rates.

From these follow-up data one might conclude that phenytoin continues to be highly efficacious in the treatment of a few selected binge eaters. For those subjects who did not initially respond to phenytoin or those who did not elect to continue phenytoin treatment, binge eating persisted; the few exceptions are perhaps attributable to psychological changes in the subjects' lives.

Psychiatric Disturbances Associated with Erhard Seminars Training: II. Additional Cases and Theoretical Considerations

BY MICHAEL A. KIRSCH, M.D., AND LEONARD L. GLASS, M.D.

In a previous article, the authors reported on 5 individuals who developed psychoses after participation in Erhard Seminars Training (est). Two additional cases are reported, and the combined case material is discussed in terms of group and psychodynamic theories. The authoritarian est leadership style may mobilize in trainees an overdetermined and pathological reliance on identification with the aggressor. Such a mechanism may be central to the production of psychiatric casualties, particularly in individuals with defective ego boundaries. Future controlled research is necessary to ascertain the rate of occurrence of psychiatric disturbances associated with est and to test the authors' hypotheses.

IN THE FIRST part of this report on psychological complications associated with Erhard Seminars Training (est) (1), we described 5 cases of psychosis temporally related to the training. A large, quasi-therapeutic group experience, est is becoming widely available in this country. The training occurs over two weekends, usually in hotel convention halls, in groups of 250 trainees who now pay $300 for the 60–70 hour experience. The trainer employs an authoritarian model, and trainees are not permitted to leave, talk, eat, or go to the bathroom during the marathon sessions except as dictated by the leader. The training consists of Gestalt-like exercises, didactic material, and scheduled self-disclosure, carried out in a forceful, confrontational style. A fuller description of the est training program appears in our earlier report (1).

The est organization does not advocate the training for people who have a history of psychiatric hospitalization. Applicants who are in psychotherapy but are dissatisfied or believe they have fared poorly in therapy in the 6 months before the course are also reportedly discouraged. If the prospective trainee chooses to disregard these recommendations, clearance from the therapist and the therapist's pledge of availability to the enrollee during and after the training is said to be required (2).

There is no formal follow-up procedure. Post-training contact by the est organization consists of telephone calls and mailings urging the graduate to return for continuing "postgraduate" seminars.

Our experience with the est program includes 7 cases of psychological distress temporally related to the training, 5 of which were included in our first report. This group of est trainees came to our attention through our work in a variety of emergency psychiatric settings. In addition, one of the authors of the first article (F.P.) personally completed the basic est training.

CASE REPORTS

Case 6. Ms. F, a 26-year-old single high school teacher, developed hyperphagia and an acute depression after the est training. The oldest of two children born to parents of Asian extraction, she has a master's degree in education and her brother has a bachelor's degree. Her father had a successful health food business and her mother increased the family fortune by investing wisely in the stock market. When Ms. F was 21, her mother died of gastrointestinal cancer. Ms. F had many friends while growing up and energetically involved herself in church activities during adolescence. She excelled in school. Neither she nor any member of her family has a prior psychiatric history.

When she was 24, a long-term involvement with a man floundered, and Ms. F enrolled in est in an effort to salvage the relationship. During the training, she recognized her pattern of being the "victim" in her relationships, a painful insight that shook her self-esteem. She could not "share" her discomfort with the group because she felt "the leader did not want to hear negative experiences," and "the group would not be supportive."

During the first weekend of the training, Ms. F began gorging and in the next month gained 30 pounds. After completing the training, she became "apathetic" and "lost interest" in life. She broke off contact with friends and moved back into her father's house. Her life became a drab routine of attending school and returning home to eat and sleep. She contemplated suicide intermittently (but made no attempts) and describes this period as "the worst time in my whole life." In an attempt to ameliorate her psychic distress, she took a postgraduate est course entitled "Be Here Now." This course intensified her depression and made her feel like

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a "misfit" because she could not "get it."

Two months after the initial training and a total of 40 pounds heavier, she sought professional help and initiated twice-weekly psychotherapy. Five months into therapy, her depression began to clear, she began to lose weight, and she recontacted friends. Ms. F finished her master's degree and obtained work as a teacher. She is currently in uncovering psychotherapy twice weekly and continues her teaching career.

Case 7. Ms. G, a 24-year-old unemployed single woman, became confused, disoriented, and frightened immediately after the est training. This reaction improved spontaneously in a few days. Six months later during an est postgraduate seminar the patient developed delusional thinking.

Ms. G is the youngest daughter of an abusive alcoholic father; her mother, described as warm and loving, died when she was 11 years old and she was cared for by a series of foster parents until she was 18. Her two older siblings are both professionals. Although she was a poor student and a disciplinary problem, she graduated from high school. After graduation she worked intermittently as a cashier in a grocery store. Ms. G sporadically abused amphetamines while "dieting." She has no formal psychiatric history; her father was hospitalized several times for alcohol-related problems.

In the fall of 1975, after separating from a boyfriend, Ms. G enrolled in est to help her "communicate with people," "lose weight," and "pull out of a depression." On the last day of the second weekend of training, she became "confused" and was unable to locate her car without police assistance. Her confusion subsided in the next few days, but she felt inadequate and isolated herself from her friends. She did, however, reconcile with her boyfriend.

Five months later, Ms. G took the "Be Here Now" course. During the program, she felt "propagandized" and was bewildered by such statements by the trainer as "Let it be okay not to be something before you can be it." She stated that the concept, "talking in the back of my head," "blew my mind." During the training, her "thinking went wild" and she began "thinking about thinking." In the midst of the course she became delusional, thinking that she was the Virgin Mary, that the trainer loved her, and that Werner Erhard was inside her. Following the postgraduate est course, Ms. G became more isolated, her personal hygiene deteriorated, and she stopped doing housework.

Three months later she became preoccupied with est, isolated herself from her family, began staring into space, and heard voices. She had become convinced that Werner Erhard was inside her and that she was in communication with him. She was hospitalized at her community mental health center and treated with antipsychotic medication. During her 6-day stay, her behavior became more appropriate, but at the time of discharge, she continued to report occasional auditory hallucinations. Her discharge diagnosis was acute schizophrenic episode. She was referred for outpatient treatment with a recommendation that she continue taking haloperidol, 15 mg/day. Five months later she is able to work part-time, supported by antipsychotic medication and weekly psychotherapy.

RESULTS

Our population consists of the 5 cases reported in our previous article and the 2 presented above. Demographic characteristics and psychopathology are summarized in table 1. The average age is 30, and the group is nearly equally divided between men and women. Five individuals are Caucasian and two are of Oriental extraction, which is probably a reflection of the ethnic makeup of the San Francisco Bay area. Educational attainment of the group is above average, which is consistent with reports that est appeals to a more established, middle-class population than other popular psychological movements, which draw participants from the alienated subcultures.

Of the 6 patients who had clear psychotic episodes, only one had a history of psychosis. It is striking that the modal individual in our study was well educated and productive, with no history of treated psychiatric illness or hospitalization.

Five patients experienced schizophrenic episodes, 3 of the paranoid type and 2 of acute schizophrenia. The remaining cases were 1 first-break manic-depressive illness and 1 depressive neurosis. The severity of these difficulties is suggested by the diagnoses. The participants' subsequent courses (over a period of several months to 3 years) are consistent with those expectations. Only 1 patient has had no recurrence or evident impairment. The others have had marked constriction in life activities and/or relapses into psychotic states.

DISCUSSION

As we have stated previously, no determination can be made on the basis of our findings alone regarding the causation or frequency of associations between est and psychiatric morbidity. Nonetheless, we believe a discussion of possible etiologic mechanisms is warranted to aid clinical management and future systematic study. In this effort, consideration will be given to group and psychodynamic explanations.

Group Dynamics

The paucity of literature on complications of participation in large quasi-therapeutic groups necessitates a review of the relevant experience with encounter groups. Recognition of psychological injury to some participants of encounter groups led to the American Psychiatric Association task force report, *Encounter Groups and Psychiatry*. After outlining the shortcomings of research in this area, the task force concluded that

> no generalization may be made save that, in the hands of some leaders, the group experience can be dangerous for some participants. The more powerful the emotions evoked, the less clinically perspicacious and responsible the leader, the more psychologically troubled the group member, then the greater risk of adverse outcome. (3, p 17)

More recently, Lieberman and associates (4) wrote a book-length prospective study of encounter groups, including a case-by-case analysis of the 16 individuals

TABLE 1
Characteristics of Seven Patients Who Developed Psychoses After est Training

Case Number	Age	Marital Status	Education	Psychopathology	Course
1	39	Married	College	Acute schizophrenic episode	No recurrence
2	26	Single	College	Schizophrenia, paranoid type	Constriction
3	28	Married	Postgraduate	Acute schizophrenic episode	Constriction
4	34	Married	College	Manic-depressive illness	Recurrences
5	30	Single	College	Schizophrenia, paranoid type	Constriction
6	26	Single	College	Depressive neurosis	Constriction
7	24	Single	High school	Schizophrenia, paranoid type	Constriction

in a sample of 206 who suffered significant psychological injury. While conceding that their methods clearly underestimated the number of casualties, the authors stated that "a casualty rate approaching 10% is alarming and unacceptable in an endeavor calculated to foster positive growth."

It is reasonable to assume that some awareness of the possibility of similar injury to participants in est's large-group experience led the organization to adopt the screening procedures described earlier. However, in our small sample, 2 individuals failed to meet the stated est screening criteria (case 5, Mr. E; and after the first episode, case 4, Ms. D) and either were not detected or not acted upon. No contact was made with either treating psychiatrist and no authorization from the physician was required for further participation in the est program. Thus profoundly disturbed individuals may not be excluded by the est screening procedure.

The lack of emphasis on screening is consistent with est practice, which provides essentially the same training experience for each new group without consideration of the specific psychological needs of the individuals enrolled. Fenwick (5) has pointed out that sophisticated assessment of individual psychopathology is beyond the competence and training of the est personnel; it is also outside the est value system, since the training is held to be almost universally beneficial. Fenwick also believes that "est uses techniques indiscriminately which, in a certain proportion of the population, are known to be harmful and potentially quite dangerous."

The est organization does not follow its clients in a manner that would mitigate the deficiencies of the screening process or attempt to rectify whatever disruption might result from the training itself. Several of our patients commented that the only further attention they received after the course was pressure and inducements to enroll in additional est activities.

Clearly, much of the toxicity of any injurious group experience is determined by the approach and conduct of the leader.[1] Lieberman and associates (4) examined the contribution of the style of encounter group leaders to subsequent psychological injury of participants in the group they led and found that one leadership style was associated with a disproportionate number of the casualties and tended to produce the most severe injuries. These "Type A—Energizers" are intense emotional stimulators, most committed to an articulated belief-system and to the founder of their school of thought; they are highly charismatic, proselytizing, "religiostic," and intent on firmly directing participants toward "the road of salvation." Elsewhere, Yalom and Lieberman (6) have described these leaders as "aggressive stimulators—intrusive, confronting, challenging and authoritarian."

Although we did not observe the est trainers in the sessions our patients attended, information from our patients and other sources (5, 7) leaves little doubt that est leaders could be classed as Type A. This style so uniformly typifies the est trainers as to defy coincidence. Apparently this authoritarian, confrontational style is sought and developed to transmit the est message in an unmistakable and characteristic mode. However, this approach systematically replicates the leadership style with the greatest known capacity for

[1]Reference to the encounter group literature may be helpful in understanding specific mechanisms of psychological injury to group members. Lieberman and associates (4) have identified five factors that resulted in casualties to encounter group participants: attack by the leader, rejection by the leader, failure to attain unrealistic goals, coercive group expectations, and input overload. Descriptions of their experience of the est process make it clear that all of our patients believed they were harmed in one or several of these ways. Indeed, attack and rejection by the est trainer occurs universally in the training process. While expectations (by the leader and the mobilized group of trainees) have been experienced as coercive, they are also contradictory and bewildering. The possibility of input overload is structurally enhanced by the marathon format, aimed at overwhelming defenses by sustained pressure. Finally, the trainee's unrealistic goals are experienced as being simultaneously encouraged and disavowed by the leader.

psychological injury and exposes a virtually unscreened and highly stressed population to it.

Psychodynamics

Speculation about the psychodynamic mechanisms that might explain the major regressions seen in our cases can only be tentative, since our data are not based on long-term controlled study.

The est training is structured to promote regression. Oral intake, urination, and defecation are regulated by the trainers, as are motility and communication. This infantalizes the participants while elevating the leader to a position of omnipotence. Trainees deal with this regressive pull in a variety of ways. Some become observers and distance themselves from the process. Some submit passively. Others may question the process aggressively; the leader's response to their challenge is often delivered in an authoritarian, confrontational, and ridiculing manner that may produce anxiety, humiliation, and fear of future retaliation. Fear of the powerful leader is contagious and is enhanced by the agreed-to proscription against leaving the training room and by a mass psychology of ambivalent submission.

One mechanism of defense that may be used to handle anxiety aroused by forced submission to this powerful leader is identification with the aggressor (8), i.e., attempting to deal with the frightening dominance of the leader by becoming like him or her. However, the defensive identification with the aggressor, invoked to ameliorate the participant's anxiety, may also set up conditions that increase the likelihood of psychiatric disturbances. Shafer, in his definition of identification, says,

> In its fullest, the process of identifying with an object is unconscious, though it may also have prominent and significant preconscious and conscious components; in this process, the subject modifies his motives and behavior patterns, and the self-representations corresponding to them in such a way as to experience being like, the same as, and merged with one or more representations of that object. (9, p. 140)

The experiences of merging with and being the same as an object are fundamentally primary process phenomena, while the experience of being like an object is essentially a secondary process phenomenon. Identifications are composites of both of these.

The conduct of the est training elicits conflicts over dominance and submission that often generate signal anxiety. In response to this signal, an identification with the aggressor is mobilized in addition to other characteristic defenses. Difficulties may occur when a participant whose ego boundaries are defective because of early problems with self-object differentiation identifies with and unconsciously experiences merging with the feared leader. This unconscious experience of merging may precipitate self-object confusion, fear of loss of the self, dissolution of the self, and concomitant instinctual anxiety. In the face of the intensely and unselectively stressful est experience, the identification with the aggressor fails to contain the signal anxiety and paradoxically leads to what may be termed a pathological identification. Psychotic symptoms (alterations in ego functions, delusions, and hallucinations) are then mobilized to prevent the emergence of overwhelming id anxiety (10).

Evidence for a universal (and not necessarily pathogenic) identification with the trainer may be inferred by observation. During the two-weekend course, the trainees' style of dress often begins to mirror the characteristic open-collar style of the leader (2). The est terminology enters the graduate's vocabulary, and graduates often are identifiable outside of the training by virtue of their aggressive, stiffly self-confident, and proselytizing manner.

Examples of a more pathological identification with the aggressor include Mr. B (case 2 in our first paper), who, after taking the trainer's seat on the stage, experienced dissolution of the self, overwhelming anxiety, and subsequent delusions and hallucinations. Ms. D (case 4) felt she was the unacknowledged "other half of Werner Erhard." In case 7, Ms. G's pathological identification was probably associated with fantasies of incorporating the leader.

We have postulated that an overdetermined and pathological reliance on identification with the aggressor is central to the production of psychiatric casualties among est participants. How then would one account for positive outcomes, i.e., those people who report major improvements in emotional well-being and adjustment after est? (We assume that such improvements may occur, even after placebo-effects and other artifacts are considered. A determination of the frequency, extent, and duration of such changes transcends our data.)

We believe the same elements to be at work, but in a more favorable climate. An individual may have contact early in life with a threatening and ambivalently held object, without being as intensely traumatized as we speculate our est casualties were. Their earlier relationships may have been less noxious because the object was not as intrusive or menacing, or the contact may have been moderated by circumstances, e.g., availability of other need-satisfying objects or later or briefer exposure to the ambivalently valued and powerful object. In such a manner, the individual may retain some identification with the powerful object without being as inclined to fuse with him. In the setting of the est experience, a regression may occur that does not threaten ego-integrity but does provide the opportunity for an integration of the old introjects and a mastery of this early trauma.

CONCLUSIONS

Seven cases of psychological complications associated with exposure to est have been presented and

discussed. The nature of our material does not permit generalization with regard to causality and rate of occurrence. However, the group and psychodynamic explanations we have presented may, if substantiated by future systematic research, explain these findings. Specific consideration should be given to the potential significance of the leadership style of est trainers and the defensive maneuvers mobilized in trainees.

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Success Rates in Psychiatry and Neurology Certification Examinations

BY MILTON GREENBLATT, M.D., JEAN CAREW, PH.D., AND CHESTER M. PIERCE, M.D.

The authors traced the progress of a sample of 200 psychiatrists and 150 neurologists on the ABPN examination. Overall success rates were high—64% of psychiatrists and 75% of neurologists became Board-certified. Performance on the written exam was a significant predictor of outcome for both groups—88% of psychiatrists and 94% of neurologists who passed Part I on their first try eventually obtained certification. Given the criticism that has been directed toward the oral exam, it was interesting that only 10% of psychiatrists and 5% of neurologists failed to become diplomates because of difficulties with Part II. Future research will deal with the validity and reliability of the oral exam.

IN THE PAST five years the American Board of Psychiatry and Neurology (ABPN) has undertaken research efforts designed to guide policy decisions in its mission of certifying specialists. We hope that this article—the first in a series—will inform relevant constituencies as well as stimulate constructive criticism.

We followed a cohort of applicants in their pursuit of certification. All members of this cohort were licensed physicians who had finished residency training in psychiatry or neurology and had completed at least two years of experience in the field, making them eligible to take Part I, the written portion of the ABPN examinations. Upon successful completion of Part I, these candidates took oral examinations (Part II), during which they interviewed patients and were examined by diplomates of the Board. A candidate could pass, condition one specialty, or fail the oral examination on his/her first attempt, but on second attempts candidates either passed or failed. During the time this cohort was studied, a candidate who failed the oral examination a second time was required to wait two years before trying again. A candidate became a diplomate of the ABPN after he/she passed both Part I and Part II. This study reports on the successes and failures experienced by a sample of candidates over a four-year period in their quest for certification by the Board.

METHOD

The names of 214 psychiatrists and 152 neurologists were randomly selected from applicants who were accepted for the ABPN Part I examination in 1972. Of these applicants, 200 psychiatrists and 150 neurologists actually took the examination; the remainder simply did not show up to take Part I. The success of candidates on the Part I (written) and Part II (oral) examinations was then tracked over a four-year period from 1972 to 1976. The 1972 Part I examination was chosen as the starting point because various changes in the examination rules and content made in previous years had stabilized by that time. The January 1976 oral examination was chosen as the endpoint because it was

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The papers in this section are grouped around a specific topic. Publication here does not, however, imply that the Editor considers this material to constitute a comprehensive analysis of the topic.

TABLE 1
Success on Part I and Subsequent Performance

Candidate Group	Psychiatry (N=200) Number	Psychiatry (N=200) Percent	Neurology (N=150) Number	Neurology (N=150) Percent
Passed Part I on first try	136	68.0	108	72.0
Part II, try 1				
Pass	96	70.6	82	76.0
Condition/fail	38	28.0	24	22.2
Decline	2	1.4	2	1.8
Part II, try 2				
Pass	24	17.6	18	16.6
Fail	14	10.4	6	5.5
Final status				
Diplomate	120	88.2	100	94.4
Not certified	16	11.8	8	5.6
Failed Part I on first try	64	32.0	42	28.0
Part I, try 2				
Pass	14	21.9	14	33.3
Fail	18	28.1	11	26.2
Decline	32	50.0	17	40.4
Part II, try 1				
Pass	5	7.8	8	19.0
Condition/fail	9	14.1	6	14.3
Part II, try 2				
Pass	3	4.7	4	9.5
Fail	6	9.4	2	4.7
Final status				
Diplomate	8	12.5	12	28.5
Not certified	56	87.5	30	71.5

the last examination for which data were available for use in this study.

RESULTS

A total of 64% of the psychiatrists in our sample obtained ABPN certification in the four-year period; 48% passed both exams on their first attempts. Other psychiatrists passed Part I on the second try and were then successful with Part II (2.5%) or passed Part I on the first try but had to take Part II twice (12.5%). A small percentage of candidates in psychiatry (1.5%) had to repeat both the written and the oral exams before becoming diplomates.

Findings for candidates in neurology were similar, although their rates of success were generally higher than those for psychiatrists. Of the 150 neurologists in the sample, 74.7% obtained ABPN certification in the four-year period. Approximately 55% were successful after taking the written and oral exams only once; 5.5% had to repeat the written exam, 12% had to repeat part or all of the oral exam, and 2.7% had to take both exams a second time.

For both psychiatrists and neurologists, initial success or failure on the written exam seemed particularly significant. The data in table 1 indicate that of the 136 psychiatrists who passed Part I on their first try, 88.2% eventually obtained certification, although 17.6% had to repeat Part II. Similarly, of the 108 neurologists who passed the written exam on their first attempt, 94.4% became diplomates in the four-year period. However, of the 64 psychiatrists who failed Part I on their first try, only 8 (12.5%) were ultimately successful, and of the 42 neurologists who failed Part I initially, only 12 (28.5%) were eventually certified.

There are several reasons why candidates who initially failed the written exam did not achieve certification. Many of these candidates simply declined to repeat Part I (50% of psychiatrists and 40.4% of neurologists). Some retook Part I but failed again (28.1% of psychiatrists and 26.2% of neurologists). A few passed Part I on their second attempt (21.9% of psychiatrists and 33.3% of neurologists), but several individuals in this group subsequently failed to pass Part II after two attempts (9.4% of psychiatrists and 4.7% of neurologists).

Thus, although the oral exam has been severely criticized as being too stringent, relatively few candidates (10% of psychiatrists and 5.3% of the neurologists) failed to become diplomates because of difficulties with Part II. For most, the major stumbling block was the written exam: they either failed Part I on their first try and declined to pursue certification further or failed a second time and were compelled by ABPN regulations to wait two years before reapplying.

DISCUSSION

In the past, there has been much discussion by colleagues at meetings and in the literature regarding the pass/fail rates of the ABPN examinations. To our knowledge this study is the first effort to track the fate of a cohort of candidates to determine their actual success rates in the ABPN certification process.

A major implication of the data we have reported seems to be that Part I does serve its essential function as a "screening" device and thus seems to be the major barrier to certification. A candidate who passes the written exam is much more likely to persist in the examination process and become a diplomate of the Board than one who initially fails Part I. It is impressive that 88.2% of psychiatrists and 94.4% of neurologists who passed Part I on their first try were certified within four years, but it is disquieting that only 12.5% of psychiatrists and 28.5% of neurologists who initially failed this exam eventually became diplomates.

No one would deny that candidates who fail an exam are burdened by inconvenience, expense, anxiety, and embarrassment. It is of concern to the Board and to the profession in general that only 48% of psychiatrists and 54.7% of neurologists were able to pass both exams on their first attempts. This may mean that the exams are deficient, that candidates are insufficiently prepared, or that the exams, despite an admitted need for further improvement, do serve to maintain essential professional standards by not permitting overly easy access to specialty status. One may argue that the

ABPN standards implied by these success rates are too stringent—or too lenient. Our findings demonstrate that certification is within the reach of most candidates on the present exams: 64% of psychiatrists and almost 75% of neurologists did become diplomates, a success rate that is comparable to that of other medical specialty branches.

Of specialists who passed Part I without difficulty, eventually 88.2% of psychiatrists and 94.4% of neurologists received their diplomate. Of specialists who failed Part I on their first try but passed subsequently, 12.5% of psychiatrists and 28.5% of neurologists became Board-certified. Of all those who started the exam process 60% of psychiatrists and 66% of neurologists were certified with at most one repeat. Although the Board receives many comments from colleagues who are critical to varying degrees of the exam process, almost all are persuaded that oral examination, especially in psychiatry, is essential. The obvious problem is to ensure that the oral examination is valid and reliable. Future reports will deal with efforts to achieve this goal.

An Examinee's Perspective on Board Certification

BY BEN MAURICE BROWN, M.D.

The author reviews the objectives, sources, preparation, and test-taking strategies commonly used by successful candidates for the certification examination of the American Board of Psychiatry and Neurology, Inc. He suggests that examinees can benefit from the preparation process as well as increase the probability of passing by adopting these strategies.

THE PROCESS of certification in psychiatry by the American Board of Psychiatry and Neurology, Inc. (ABPN), has acquired increasing importance in the past five years for individual psychiatrists and for the institutions with which they are affiliated. During this period the certification process has been subjected to critical scrutiny within the psychiatric profession in the literature (1, 2) and in professional meetings (3). Furthermore, in the past five years the ABPN has promulgated a number of substantial refinements in its certification procedure to improve both the logistics and the reliability of the examination process. These events have focused attention on the status of Board certification as the prevailing standard for professional competence in psychiatry in the United States. In this regard psychiatry is moving toward an alliance with other medical specialties in viewing Board certification as a significant criterion of specialty status. Certification may also come to play a central role in recertification and third-party payment schemes in coming years.

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CRITICISM AND REFORM

Since 1975 professional criticism of Board examinations in the literature (1) and in professional meetings (3) has tended to focus on several allegations, including ambiguity in defining criteria for passing, logistical complexity, lack of assessment reliability, anxiety-provoking setting, and absence of a spontaneous feedback mechanism for candidates and for training program directors. However, it must be evident that the assessment tools used by the ABPN, whatever their shortcomings, bear far greater similarities to than differences from standard measures for assessing competence as a physician (e.g., the National Board or Flex examinations) and as a specialist in other areas of medicine (e.g., specialty board examinations in orthopedic surgery or dermatology).

Because ABPN examinations have evolved through a selective process like other medical certifying examinations, criticism of the Boards might best be taken as a source of input for refining a tool rather than an excuse for abandoning an encumbrance (2). For example, although efforts to increase the reliability of the oral part of the Boards (Part II) are certainly worth-

while, one could hardly expect to make a valid assessment of clinical competence without observing a candidate's immediate evaluation of clinical data. In any event, Board examinations continue to provide the best standard measure of theoretical mastery and clinical competence developed to date in psychiatry.

In the past three years the ABPN has taken a number of steps to reform the certification process and the assessment tools involved (4). Some of the changes promulgated include elimination of the two-year experience requirement between completion of psychiatric residency and eligibility for Part II, use of videotaped interviews for one hour of clinical psychiatry and one hour of clinical neurology for Part II, selection of multiple sites closer to the candidates' area of residence for Part II, provision for selected feedback by request on examination performance for candidates who condition or fail, provision for feedback to training program directors on the performance of their trainees, and expansion of detailed guidelines for the examiner's evaluation of Part II candidates. Some of these changes would appear to substantially alleviate the logistical difficulties faced by examinees; others aim at improving the reliability of examination procedures.

In view of the increasing importance of Board certification, psychiatrists planning to prepare for the examination would do well to maximize the benefits of the preparation process itself as well as try to increase the probability of successfully completing the examination. From the examinee's standpoint a number of general principles might bear reviewing in this regard. These principles can be categorized under the four general headings of objectives, sources, preparation, and test taking.

OBJECTIVES

At least four major objectives may be defined for the preparation process for Part I and Part II of the Board examination: 1) train to basic competence in all branches of psychiatry and to familiarity with all branches of neurology (Parts I and II), 2) develop an organized core framework for understanding new data from any branch of psychiatry and neurology (Part I), 3) review subspecialties of interest in detail (Part I), and 4) refine clinical diagnostic and treatment planning skills (Part II).

The first of these objectives would promote breadth of understanding for the subspecialist, e.g., the psychopharmacologist training to basic competence in dynamic psychiatry. The second would provide a source of reference for integrating new data from other subspecialties of interest, e.g., the psychoanalyst acquiring a core knowledge of forensic psychiatry, permitting integration of new data on peer review legislation affecting confidentiality. The third objective would permit a thorough review of a subspecialty interest area, e.g., the forensic psychiatrist reviewing in detail state legislation governing expert psychiatric testimony. The fourth would provide the opportunity to hone clinical evaluative skills to a fine edge, e.g., the dynamic psychiatrist refining his or her ability to do a rapid neuropsychiatric diagnostic workup and treatment plan formulation.

SOURCES

The second group of principles for consideration concerns the sources for literature review. Given a limited amount of preparation time, Board candidates must select the resources on which they will primarily rely from a wide variety of excellent literature. Since it is not practical to read in detail all of the available reference material in psychiatry, many successful Board candidates have begun their preparation by selecting one concise but thorough text providing a synopsis of psychiatry and one equivalent text in neurology. Successful candidates often study such texts intensively, reviewing and perhaps outlining each major subject area. They might then consult more detailed reference texts for a thorough review of particularly important areas.

Important areas for review for Part I include detailed factual data on subjects ranging from neuroanatomy to forensics. Basic science areas such as neuroanatomy and neurophysiology often require separate review sources in addition to standard psychiatry and neurology texts. There are several excellent but concise neuroanatomy and neurophysiology review texts. Some candidates also use review question books to survey their detailed factual knowledge in different areas of psychiatry and neurology.

Particularly important areas for the more clinically oriented Part II examination include diagnostic and treatment planning as well as clinical procedures applicable to the major clinical entities in psychiatry and neurology. Thus, clinical chapters in major texts are particularly helpful in clarifying important differential diagnostic distinctions and treatment procedures. An important resource area for both Part I and Part II is the wealth of recent reports of major findings in the journal literature of the past four years. Major theoretical papers and reports of carefully controlled clinical studies involving large numbers of patients provide invaluable source material for Board review.

In summary, there are at least five areas of the literature from which resources may be selected for review in preparation for Boards: psychiatry texts (5–8); neurology texts (9–12); recent journals, such as the *American Journal of Psychiatry, Archives of General Psychiatry, Archives of Neurology,* and *Brain*; neuroanatomy and neurophysiology texts (13–16), and review question books (17, 18). It should be noted that these references are not intended to summarize all the excellent available resources but to provide several examples in each of the five areas of resources that successful Board candidates generally review.

PREPARATION

The third set of principles for consideration concerns preparation. By virtue of their lengthy training and extensive test-taking experience, psychiatrists tend to develop a variety of successful examination preparation strategies that might serve as guidelines for Board preparation. At the same time, a number of general preparation guidelines are commonly cited as helpful by successful Board candidates. These general guidelines aim at providing candidates with adequate preparation inputs, time, practice in clinical skills, and familiarization with examination format.

One frequently recommended guideline involves systematically reviewing all major branches of psychiatry on the basis of literature resources. This review can be enhanced by preparing written outlines for each major topic in psychiatry. Some candidates also find it helpful to attend selected seminars and meetings in psychiatry to complement their reading.

A widely accepted guideline in preparing for Part II involves taking opportunities to personally interview, examine, and discuss patients with psychiatric and neurological disorders. This is especially true for candidates who arrange to repeatedly perform brief history, mental status, and neurological examinations followed by examination by preceptors who use a format similar to that of Part II. Making rounds to see selected psychiatry and neurology patients with a Board-certified specialist is also often cited as helpful.

Another guideline concerns the amount of time required for preparation. Naturally, this factor is highly variable among different candidates. Most successful candidates would probably agree that 4–5 hours a week for the 4–6 months before the examination is ample for review purposes. However, it is common for the preparation time to increase to 10 hours a week in the fortnight before the exam in order to capitalize on short-term retention of details.

A final guideline that might prove helpful is to decide on a clearcut contingency plan in the event of conditioning or failing. Barring major logistical obstacles to retaking the examination, conditioning or failing may provide helpful clues for a subsequent attempt. Conditioning may imply a weakness in one particular area of preparation, while failing may imply broader gaps of knowledge and test-taking skills. A disappointing initial outcome may be turned into an opportunity to identify specific deficiencies in the candidate's initial preparation. Subsequent preparation might then correct such deficiencies by focusing on a thorough review of underprepared topics, an increased effort to practice clinical examination skills, or the development of techniques to allay stress-induced anxiety.

TEST TAKING

The fourth and final category of principles involves the test-taking process. One major aim in this area is to reduce the candidate's anxiety to a level that will not markedly impair his or her recall of important facts or performance of clinical skills. This is vital because many candidates who fail or condition the Board examination appear to show not just a mild but a substantial decrement in performance secondary to excessive anxiety. Competent performance under stress is certainly a necessary aspect of the physician's role and therefore needs to be assessed in the Board certification process. The Board candidate, in turn, needs to minimize adverse stress effects and optimize the probability of responding in a basically sound and adaptive fashion in the face of examination stress. With respect to the test-taking procedures, a number of guidelines may prove useful.

Applying early is likely to facilitate obtaining one's choice of a favorable examination site. Arriving early at the site may permit a relatively leisurely day before the examination as well as allowing ample leeway for dealing with possible complications in transportation and lodging. Reviewing notes and selected text material for up to two hours on the night before the test should probably prove sufficient; 99% of the reviewing should be completed by that time. It may also be helpful to plan to use the examination as a self-measure of theoretical and clinical skills already mastered as well as special areas meriting further study. After all, most candidates who pass as well as those who condition or fail could profitably learn more about certain specialty areas. Last, but not least, it may prove helpful during the examination for candidates to be supportive of patients' roles as volunteers as well as examiners' efforts to be fair but thorough evaluators. Such an attitude might well enhance the potential benefits of the complex and stressful Board examination process for all participants—candidates, patients, and examiners.

CONCLUSIONS

Candidates preparing for the ABPN certification examination undertake a process aimed at affirming their role as medical specialists. This is a role that is becoming increasingly appropriate for psychiatrists. In fact, ABPN examinations have come to assume increasing importance in psychiatry as the specialty has shown educational and clinical developments increasingly parallel to those in other medical specialties.

Contemporary medicine is becoming increasingly concerned with the development and maintenance of professional competence among providers of health care, a concern that has become influential in psychiatry in several areas. In psychiatric training programs, conpetency-based residency education (19, 20) is becoming more prominent. In continuing education, a broad voluntary self-evaluation program has recently been developed (21). In certain clinical service agencies, peer review of clinical treatment procedures has been instituted (22, 23). In specialty certification, ABPN examinations are increasingly assuming the

role of a major index of professional competence in psychiatry. Thus, in summary, the psychiatrist undertaking the process of preparing for and taking Boards encounters a unique opportunity both to train to broad competence in his or her chosen specialty and to receive professional and public accreditation for success in this venture.

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From Board Candidate to Rookie Examiner

BY MICHAEL J. NAPOLIELLO, M.D.

The author describes his experience as a candidate for the American Board of Psychiatry and Neurology, Inc., examinations one year and as an oral examiner the next. He discusses the oral examination process, listing the errors most frequently made by candidates.

THE EXPERIENCE of taking the oral examination of the American Board of Psychiatry and Neurology, Inc. (ABPN), has been vividly if not accurately described in the literature (1–3). The predominant tone of these reports is less than positive. In attempting to assess these anecdotes, one has to question the degree of self-selectivity involved. Does the silent majority of candidates who pass the oral examination remain so because the ordeal is over with and they would just as soon forget it, or is the relief of passing a weaker motivator to verbalize than the anger and frustration of failing? In this article I will not attempt a scientific resolution of the matter; rather, I will describe the experience of looking at the oral portion of the Board examination from a unique perspective—as a candidate one year and an examiner the next.

At the orientation session for candidates in St. Louis, Mo., in October 1975, the Executive Secretary of the Board expressed the hope that after certification some of the candidates present would join the ranks of oral examiners. On the basis of a modest degree of previous experience and an interest in oral and written examinations in medical school settings, I decided to accept the invitation.

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I have described my own experience with the ABPN examination elsewhere (4). This experience had been positive but exacting. On the oral examination, I was treated fairly and courteously by all 6 of my examiners. Both psychiatry hours went smoothly; nearly all of the questions focused on the patient and the physician-patient interaction. My neurology hour, however, produced greater discomfort. The patient had complex and confusing extracranial findings. Despite a reasonable degree of neurological sophistication for a psychiatrist, I felt like a fish out of water. Fortunately, the examiners were understanding; by skillful inquiry they were able to prevent a few awkward moments from becoming a catastrophic half-hour.

The experience was both dissociative and draining. I found myself getting off the hotel elevator on the wrong floor, forgetting names of other candidates I had met a day or two previously, and feeling too fatigued to leave my room. This "pseudodelirium" disappeared by the following day, but the fatigue lasted several more days, along with gnawing anxiety as I waited for the good word from Chicago. I felt almost ashamed to have gotten so "psyched-up," but I found this to be well within the norm for Board candidates.

THE EXAMINATION PROCESS

One year later, I was a rookie examiner on a plane to Chicago, leafing through the same books and notes I had studied as a candidate. A few weeks earlier I had received a detailed orientation in the mail describing the nature and format of the examination, including an explicit caveat against inappropriate behavior toward candidates. This was followed up by a 3-hour orientation session the day before the examination. This session included more detailed instructions on format and a showing of sample videotapes, although my examining team was one of several that were not involved with the videotape portion of the examination.

The team to which I was assigned consisted of 14 examiners (12 men and 2 women), plus an additional psychiatrist who served as section chief. There was 1 other novice examiner in the group; he had become certified 6 months previously. Each candidate in our section was examined by 2 of us, 1 of whom served as primary examiner and the other as "floating" examiner. The primary examiner spent the entire hour with the candidate (30 minutes of patient interview followed by 30 minutes of questioning). The "floater" was expected to cover two candidate examinations within that hour. The section chief also participated as a floater. Immediately before each examining hour, the section chief introduced the candidate to his or her primary examiner. Examiners were expected to disqualify themselves if they happened to have a personal or professional relationship with the candidate.

The candidate was given 30 minutes to interview the patient. The examiner usually informed the candidate when 2–5 minutes remained. A question-and-answer session followed the interview and focused primarily on the clinical presentation of the patient: history, mental status findings, differential diagnosis, treatment, and prognosis. Questions unrelated to the patient interview were less frequently asked. The entire gamut of psychiatry was considered fair game for these questions. Minutiae and trivia were generally avoided, with the intent of highlighting sound clinical skills rather than pedantry. Morgenstern has reviewed the problems of patient interviewing and candidate questioning (5).

Following the examination hour, the primary examiner and the floater met for about 15 minutes to discuss the candidate's performance and to decide on a grade. The primary measures on which the rating was based included the ability to establish rapport with the patient, obtain information in a structured manner, and use the information to make a thoughtful *initial* approach to differential diagnosis and management plus the demonstration of an adequate amount of general psychiatric knowledge.

In my experience, no candidate was treated unceremoniously, nor was there any display of academic bravado on an examiner's part. Every effort was made to allow the candidate to demonstrate his or her fund of psychiatric knowledge in a problem-solving context within the allotted time.

THE CANDIDATES

I examined a total of 16 candidates, either as primary examiner or floater, within the 2-day period. The quality of interviews varied. A few were excellent, a few were poor, and the majority were marginal to satisfactory.

The most common interview errors included the following:

1. Failure to allow the patient to "tell the story." Too often the interview approach was mechanical, as though the candidate were going through a symptom checklist. This tended to impede the pursuit of emotional "leads" presented by the patient.
2. Too abrupt closure of the interview. The candidates often failed to decelerate as the end of the interview approached.
3. Inability of the candidate to control his or her own anxiety level. At times, this caused the patient's anxiety to increase; on one occasion it resulted in a candidate's being insulted by a delusional schizophrenic patient. Another candidate kept looking back and forth several times a minute from the patient to me; this made the patient uncomfortable.
4. Awkwardness and inconsistency of the interview structure, manifested by a great deal of jumping around and resulting in a further increase in candidate anxiety.

The errors most commonly observed during the question-and-answer period included the following:

1. Inability to summarize. This was noticeable in

the majority of the candidates I observed. Unfortunately, psychiatry has become so enamored of dealing with various levels of communication that we sometimes fail to teach our trainees to deal skillfully with the reality level. The ability to distill a mass of information and outline a problem and an initial working solution is essential to any profession. It is a sine qua non of being able to communicate effectively with others. Psychiatrists with experience in liaison psychiatry can well appreciate the value of this approach in their daily efforts to deal with physicians in other specialties.

2. Difficulty in coming to the point. This is an extension of the previous problem. I observed candidates who would digress in the middle of their answer and not succeed in finding their way back to the original question. One candidate mentioned Prolixin while discussing a patient's proposed management. When I asked him to tell me something about Prolixin, he proceeded to discuss Mellaril. The ability to be succinct is a function of self-discipline reinforced by good training.

3. Weakness in differential diagnosis. This was apparent in the majority of candidates. At times the candidate was not able to come up with more than one or two diagnostic possibilities.

4. Use of technical terms without being able to define them. Given the plethora of psychiatric textbooks and glossaries existing today, not to mention *DSM-II* (6), there is no excuse for this.

SYNTHESIS: THE VIEW FROM BOTH SIDES

My experience as a first-time examiner served to confirm the following major impressions made on me as a candidate: candidates are treated courteously and professionally; examiners attempt to elicit the strengths of candidates rather than exploiting their weaknesses; and the content of the examination is fair and relevant. The experience of being an examiner was valuable not only because of the opportunity it afforded to interact with a highly competent group of fellow examiners but also from the standpoint of insight into the oral examination process itself. In spite of the inherent limitations of oral examinations, I was impressed with the objectivity of the entire process and its many checks and balances.

Whether the oral examination in its present form is the most accurate method of evaluating clinical competence is beyond the scope of this article. So too is the problem of how to minimize the candidate's anxiety level to ensure an optimal display of competence. The ability to perform under fire may well reflect the expectations of real-life clinical crises, but the usefulness of the rite of passage mystique is equally debatable. Even if the oral examination were radically modified or eliminated entirely, no examination will be able to fully measure what a psychiatrist is expected to do or guarantee an anxiety-free state to every candidate. On the other hand, in an era when accountability and quality control are accepted professional mandates, it would be tragic, as Lipp suggested (3), to place the burden of proof on the certifying board of a medical specialty that is too often caricatured as unscientific, unstandardized, and undisciplined.

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Neurology in the Psychiatry Boards

BY BRUCE D. SNYDER, M.D.

On the basis of his experience as an examiner, the author discusses the problems encountered in assessing psychiatrists' abilities in clinical neurology for certification by the American Board of Psychiatry and Neurology. Hoping to stimulate discussion among trainees, training programs, and the Board, he outlines five paradigms designed to survey neurological knowledge and skill potentially needed by psychiatrists in various situations; these are reduced to specific topics on which Board candidates should be questioned. Relationships between training and examination expectations are discussed.

BOARD examinations for certification in psychiatry and neurology are an attempt to assure some basic uniformity of competence among practitioners. An operational consensus exists among examiners regarding what is to be required of the candidate, but it is not well defined, particularly in evaluations of colleagues in the affiliated specialty. As an examiner, I have had difficulty attempting to fairly assess the psychiatrist's abilities in clinical neurology. This paper presents my approach to this problem; I hope it will stimulate open discussion of the issues raised.

The American Board of Psychiatry and Neurology certifies clinical psychiatrists who practice in a variety of settings with varying patient groups that may be preselected in many different ways. Since an examination cannot be tailored to the predicted practice limitations of each candidate, it must be designed to cover knowledge and skill potentially needed in various situations. In terms of neurology, such situations may be summarized as follows: 1) evaluation of the demented or retarded patient for treatable organic disease; 2) evaluation of the demented or retarded patient who requires behavioral management; 3) evaluation of the delirious or confused patient with or without an altered level of consciousness; 4) assessment of the patient presenting with subjective symptoms that may represent neurologic disease; and 5) possession of sufficient background in neuroscience to interpret pertinent new developments in the psychiatric or neurologic literature.

I am fully aware that these paradigms may not represent actual practice situations for some psychiatrists; but they do represent potential types of referrals a practitioner may have to deal with. Further, psychiatrists functioning in areas where neurologic consultation is unavailable may be asked to help evaluate purely neurologic disease. If the ability to do so is implied by the joint title of the Board, perhaps testing for knowledge up to and beyond these limits is also reasonable. The validity of these paradigms could possibly be established by a survey of psychiatric practices for the frequency of their occurrence. Once established, these data could also provide a baseline for quality-of-care audits prospectively designed to evaluate the Board examination.

I have personally applied these five potential situations to the assessment of psychiatric candidates in the oral examination, and many of the candidates have had a great deal of difficulty with one or more of the areas. Given that the intelligence and motivation of the vast majority of candidates are beyond question, one must conclude that my expectations as an examiner are not congruent with the goals of psychiatric training programs and/or that candidates simply do not expect to be examined in these areas.

The awareness by candidates of the standards for success on the exams can only serve to help them seek and gain further expertise in these particular areas. I think, therefore, that open discussion of the criteria that I use may prompt further comment by others and perhaps aid in stimulating a dialogue among trainees, training programs, and the Board. I should emphasize that my criteria are not in any way officially stated by the Board or its officers and that my views do not reflect any official position of the ABPN.

STANDARDS OF PERFORMANCE

What specifically would I ask of the psychiatric candidate taking the oral examinations? Certainly the recognition of dementia (organic brain syndrome) by mental status examination is well within the area of expertise of the psychiatrist. Knowledge of treatable causes of dementia and the evaluation for these conditions is important, particularly in regard to those few readily treatable, practically emergent situations such as Wernicke's encephalopathy, the precursor to an irreversible amnestic syndrome.

Awareness of the management of the demented or retarded individual should include knowledge of the

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effect of psychotropic medications and their interactions with themselves as well as with other classes of medications. The practitioner should be aware of coexistent and coincidental conditions that could worsen the behavioral performance of such a patient (e.g., sensory deprivation). In view of the fact that the psychiatrist is often called to see the nursing home resident for deteriorating behavior, awareness of such conditions seems like a realistic standard of performance.

Recognition of impairment of consciousness is straightforward if looked for. A psychiatric candidate should be aware that the patient who has taken to bed and is sleeping all day may be either withdrawn on a psychogenic basis or lethargic due to organic disease. The clinician should have a clear idea of the urgency of such a situation and of the need for close monitoring of the patient, in addition to some idea of what must be monitored and when referral is indicated. In this regard, the candidate may be asked about certain basic applicable laboratory studies such as skull X rays, isotope scans, computerized tomography, or the EEG. Knowledge of diagnostic tests need not be asked for in detail but, rather, in terms of which test should be ordered at what point and what it might be expected to reveal.

Psychiatric consultants are not infrequently asked to see confused patients when the differential diagnosis lies between acute or chronic organic brain syndrome (delirium or dementia) and functional disease. This recognition of "organicity," as mentioned above, should be handled well. The psychiatrist should also be able to elicit and recognize such "primitive reflexes" as grasp responses, sucking, and rooting. He should probably be able to recognize asterixis and metabolic tremor as well as choreoathetosis. Awareness of the patient with partial complex seizure disorder (psychomotor seizures, automatisms) is important as these patients can appear to be globally confused or delirious. The candidate should be somewhat aware of the value of the EEG in assessing the confused patient.

The classic problem confronting the psychiatrist and neurologist is the patient with subjective symptoms that may be due to underlying disease of the nervous system. The candidate should be aware of the syndrome of atypical facial pain (these patients almost inevitably receive psychiatric referrals) and of paresthesias and the sometimes bizarre patterns of sensory abnormalities seen with early demyelinating disease. Awareness of the "danger signals" in the presentation of the headache patient is obviously important. The ability to differentiate between organic and functional bases of impotence and a familiarity with the relationship between behavior disorders and seizure activity should reasonably be expected of the psychiatric candidate. The ability to analyze and recognize classic conversion symptoms such as tunnel vision or hysterical blindness, feigned unresponsiveness, or convergence spasm is a reasonable expectation. Of course, one would hope that the candidate would be familiar with neurologic disease that mimics psychiatric disorders: this might include the behavioral abnormalities associated with collagen vascular disease, acute intermittent porphyria, and, for example, Huntington's chorea.

Finally, the candidate should have enough background in neuroscience to interpret new developments in the neurologic literature that may be relevant to the practice of psychiatry: this might include delineation of behavioral patterns related to seizure activity, developments in EEG, etc. It would seem reasonable that the candidate use abstracting services to keep somewhat abreast of neurologic literature.

REALISTIC EXPECTATIONS

It must be pointed out that although I am fairly comfortable with my choice of areas in which to question the candidate, I am unable to precisely define the level of expertise I am seeking. The candidate is asked to conform to my perception of the performance of medical nonneurologists with the provision that the psychiatrist should be quite expert with the mental status examination.

The actual oral examination provides an opportunity to watch the candidate elicit a neurologic history and perform a neurologic examination. Although a good physician can certainly elicit a clinical history, there is no question that one's fund of knowledge of the suspected pathology will guide that history and make it a more effective and efficient tool. Beyond a certain point, is it realistic to ask the psychiatrist to properly steer a history and look for subtler manifestations of neurologic disease? It is not unusual to see a candidate make serious errors of technique or misdirect his attention in the neurologic examination. Assessment is lenient, although candidates may fail on this basis. Learning the technique of examination requires a significant investment of time on the part of the neurology resident before he/she can not only go through the motions but elicit a sensitive, relevant, and reproducible performance.

Psychiatric training programs may provide the psychiatrist with a month of neurology elective; in many cases, there is none whatsoever. With the elimination of the internship, the candidate's total exposure to clinical neurology may be extremely limited. Nevertheless, candidates are asked to perform a neurologic examination and be familiar with such basic tools as the ophthalmoscope, reflex hammer, etc. Whether this is a realistic test criterion is open to question. Whether the psychiatrist should be sufficiently trained to make it a reasonable test criterion could also be a subject for some debate. Should the training program provide a minimum number of hours in supervised clinical neurology, perhaps on an outpatient basis? It may be useful to keep in mind that the availability of neurologic consultation is limited in this country. Psychiatrists

vastly outnumber neurologists and are available in communities of virtually any size. In my own experience, psychiatric colleagues in isolated areas have been asked to see and consult on instances of purely neurologic disease, and certification by a joint Board implied that they knew something about the situation at hand.

I would like to see more discussion about the possible modification of the current test format. Practically speaking, it seems to me that, given his training, it is not currently reasonable to expect the psychiatrist to perform a neurologic examination—but it is reasonable to ask him to possess a fairly broad fund of knowledge concerning the interfaces of psychiatry and neurology. I believe that these areas could be assessed adequately by an expanded written examination that might include test questions modeled after those used by the National Board of Medical Examiners. Specifically, I am referring to sequential questions designed to elicit problem-solving responses in relation to specific clinical situations. This could be done in conjunction with audiovisual aids such as films of patients and, I think, would provide a much more even and objective assessment.

Finally, one must consider certain implications of the examination for the candidate and his training program. Overall figures available from the ABPN suggest a fairly constant Board failure rate for the oral examination of psychiatrists of approximately 40%. This is not broken down in terms of how many failures are due to the neurology examinations. If one accepts that at least a significant number of candidates are failing their neurology examinations (personal observation) and if my view of the examining process and its expected levels of performance are roughly accurate (based on examining with examiners more experienced than I at two sessions of the Boards), then one is tempted to conclude that a significant portion of the psychiatric candidates are not receiving neurologic training congruent with the goals set forth in the examinations. Should the performance of candidates be analyzed according to training programs? Should such information be exchanged between the Board and training programs? Currently, such interchange is limited by legal strictures protecting the privacy of the candidate. Some feedback seems to occur in neurologic circles when performance data from self-assessment examinations are passed back to training program directors

COMMENT

The functions of the ABPN are of significance for psychiatrists, neurologists, and patients. Practitioners may soon rely upon certifying and licensing boards to protect their right to practice in a federally regulated health care system. Board activities involve a good deal of time and expense on the part of a large number of persons. The June 1976 session held in Minneapolis cost $58,000 in fees and $97,000 in expenses ($13,315 for patient and hospital expenses) for the Part II examination alone; 285 psychiatrists and 127 neurologists underwent the process at a cost of $205 per psychiatric candidate and $305 per neurologic candidate. These figures represent a relatively large undertaking for this small community of professionals and a significant and commendable effort at self-regulation. I feel that we owe it to ourselves to make the process as rational, effective, and instructive as possible.

ADDENDUM

Since the acceptance of this manuscript for publication, the American Board of Psychiatry and Neurology has announced (Archives of Neurology, 34:655, 1977) its intention to eliminate the bedside neurological examination for psychiatric candidates in the Part II examination. In addition, the Board has affirmed the desirability of providing feedback regarding candidates' examination performance to training directors if confidentiality can be preserved.

Thyroid Function and Growth Hormone Secretion in Amitriptyline-Treated Depression

BY STEVEN B. LEICHTER, M.D., LARRY KIRSTEIN, M.D., AND NEIL D. MARTIN, M.D.

The authors studied changes in indices of thyroid function prospectively in a group of 11 patients given amitriptyline to treat depression. The drug caused no significant alteration in these indices, but scores on the Hamilton Depression Rating Scale improved significantly. In another group of subjects with depression, the stimulation of growth hormone secretion by L-dopa was unaffected by amitriptyline therapy.

RECENT STUDIES have raised questions about possible interactions between tricyclic antidepressants and endocrine function. It has been suggested that thyroid hormones may alter the action of these agents (1), although their effect on endogenous thyroid function is unclear. Imipramine has been noted to have no effect on chemical indices of thyroid function (2). However, it has been reported to diminish the intestinal absorption of L-dopa (3), which is an accepted stimulant of growth hormone secretion. It is not known whether this would contraindicate the use of L-dopa in evaluating growth hormone secretion in patients who are taking tricyclic antidepressant agents. Since mood disturbance can be a symptom of thyroid disease or pituitary insufficiency, the effects of these drugs on thyroid function and on L-dopa stimulation of growth hormone secretion should be established.

When this work was done, Dr. Leichter was Chief, Endocrine Service, Department of the Air Force, David Grant USAF Medical Center, Travis AFB, Calif. Dr. Kirstein is Assistant Professor, Mt. Sinai School of Medicine, New York, N.Y., and Dr. Martin is Chief, Department of Nuclear Medicine, Kettering Hospital, Kettering, Ohio. Dr. Leichter is now Assistant Professor, Division of Endocrinology, Department of Medicine, University of Kentucky College of Medicine, Lexington, Ky. 40506.

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We prospectively followed indices of thyroid function in 11 female patients before and after the initiation of amitriptyline therapy. In addition, the effectiveness of L-dopa as a stimulus of growth hormone secretion was studied in depressed patients before and after the inception of amitriptyline therapy and in normal nondepressed subjects.

METHOD

Patients referred to the psychiatry, thyroid, or endocrine clinics of David Grant U.S.A.F. Medical Center were invited to participate in the study. All participants gave informed consent. All patients were seen by a staff endocrinologist (S.B.L. or N.D.M.), and those patients thought to have a depressive illness were evaluated by a staff psychiatrist (L.K.).

A depressive disorder was diagnosed in 11 female patients (23–59 years old), using the criteria of Feighner and associates (4). The initial score on the Hamilton Depression Rating Scale (5) in every patient was 20 or higher. All patients agreed to a trial of therapy with amitriptyline HCl and were seen by the psychiatrist before and 4 weeks after the start of amitriptyline therapy. They were also seen in the thyroid clinic at weekly intervals; physical examinations were performed, patients completed the Zung Self-Rating Depression Scale (SDS) questionnaire (6), and samples of serum were obtained for radioimmunoassay measurement of the serum concentrations of thyroxine (T_4), triiodothyronine (T_3RIA), and thyroid stimulating hormone (TSH), as well as measurement of triiodothyronine resin uptake (T_3U). After the first evaluation by the psychiatrist and endocrinologist, the patients were started on 50–75 mg/day of amitriptyline HCl. At each subsequent visit, the dosage of amitriptyline was adjusted depending on the clinical response and the occurrence of untoward side effects.

A group of 9 depressed subjects (8 women, 26–53

years old, and 1 man, 28 years old) including 3 women from the first group, were given stimulation tests of growth hormone secretion using 500 mg of L-dopa as described previously (7). These patients also fulfilled the Feighner and associates criteria and had Hamilton scale scores above 20. Patients fasted overnight before each test. Samples of serum for measurement of growth hormone concentrations were obtained before and at 30-minute intervals after the administration of oral L-dopa. Each test was carried out for 2 hours after L-dopa administration. Growth hormone secretion in response to the stimulus of L-dopa was judged to be adequate if the serum concentration of growth hormone increased 5 ng/ml or more over the fasting (baseline) value (7). L-Dopa stimulation tests of growth hormone secretion were carried out before and 1 week after the start of therapy with 50–75 mg/day of amitriptyline HCl. Similar tests were also carried out in 4 patients (3 women and 1 man, 24–53 years old) already taking 50–150 mg/day of amitriptyline, who met the criteria applied to the other patients with depression. Each of these 4 patients was taking the medication for periods exceeding 1 week. This test of growth hormone secretion was also given to 8 normal subjects (2 women and 6 men, 25–44 years old) who had no history of depression and were taking no medication.

Assays of serum T_4, T_3RIA, human growth hormone (hGH), and TSH were performed using double-antibody radioimmunoassays from commercially available kits and materials supplied by the National Institute of Arthritis, Metabolism, and Digestive Diseases. Estimation of serum T_3U was also carried out using a commercially available kit.

Statistical analyses were carried out on a Wang 2200B calculator using programs supplied by the manufacturer. Alterations in thyroid function tests were analyzed by using paired t tests, coefficient of correlation, and analysis of variance. Evaluation of growth hormone secretion was analyzed by paired and nonpaired t tests and chi-square analysis.

RESULTS

Of the 11 patients in the thyroid function phase of the study, 10 were still menstruating and 1 was receiving replacement estrogens. No patient was taking oral contraceptives at the time of the study. A subjective decrease in symptoms occurred in 10 of the 11 patients while they were taking amitriptyline HCl. In addition, their SDS scores fell from 71±1.8 to 43±2.9 ($p<.00005$), and their Hamilton scores decreased from 27.7±1.6 to 9.4±1.9 ($p<.0001$).

Physical examination for signs of thyroid dysfunction was unremarkable in all 11 patients before the start of the amitriptyline therapy, and the pretreatment serum levels of T_4, T_3RIA, TSH, T_3U were normal. The institution of therapy with 50–75 mg/day of amitriptyline did not cause a significant or consistent change in these indices of thyroid function in any patient. Similarly, further increases in the daily dose of amitriptyline failed to induce a significant alteration in these thyroid function tests in contrast to the significant decrease in the Hamilton and Zung SDS scores, which occurred in 10 of the 11 subjects.

The effectiveness of L-dopa as a stimulus of growth hormone release was not significantly impaired by amitriptyline. Only 1 patient was taking any other medication, and she was the only menopausal patient. Before amitriptyline therapy, 5 of these 9 patients had an adequate response to L-dopa; 1 additional patient responded adequately to L-dopa after the start of amitriptyline. The sums of the observed growth hormone levels in serum during the tests given before and after amitriptyline therapy were similar (31.8±24.4 and 22.3±12.8 ng/ml, respectively) as were the maximum increments noted in serum growth hormone concentrations over the fasting values (10.6±9.6 and 9.2±6.0 ng/ml, respectively).

Depression alone did not appear to alter the responsiveness of growth hormone secretion to L-dopa. Of the 13 patients and 8 normal subjects who had an L-dopa test before treatment, only 1 was taking medication. An adequate increase in serum concentrations of growth hormone was noted in 8 of the 13 depressed patients (61.5%) and 5 of the 8 normal subjects (71.5%). This difference was nonsignificant when tested by chi-square analysis ($\chi^2=.249$).

The results of L-dopa testing on all drug-free subjects was compared with all amitriptyline-treated subjects. Adequate responses to L-dopa were noted in 11 of 18 drug-free subjects (both control and depressed subjects) and in 5 of 12 amitriptyline-treated patients. The difference was nonsignificant ($\chi^2=.49$). In addition, the sum of observed serum growth hormone levels and the maximum increments over the baseline (fasting) values were similar in both groups.

DISCUSSION

Interactions between tricyclic antidepressant drugs and endogenous endocrine systems have been postulated. A number of groups, particularly Prange and associates, have shown that exogenous thyroid hormone will potentiate the antidepressant effects of imipramine (1) or amitriptyline (8), although this has been disputed recently (9). There is no available evidence to show that untreated patients with depression have any detectable abnormality of thyroid function or that these drugs alter endogenous thyroid function. Prange and associates (1) also reported that imipramine alone failed to alter plasma levels of T_4 or T_3U; they also noted similar increases in plasma T_4 concentrations following the administration of TSH given alone or with imipramine.

Messiha and Morgan (3) have presented data which suggest that imipramine may interfere with intestinal absorption of L-dopa. L-Dopa is an agent frequently used to test the integrity of endogenous growth hor-

mone secretion (7). It has been found to stimulate growth hormone secretion in a high percentage of normal subjects.

This report substantiates the data of Prange and associates (1) that imipramine does not produce clinically significant changes in plasma levels of T_4 and T_3U. In our study, amitriptyline given to euthyroid subjects failed to cause any consistent or significant alteration in plasma concentrations of T_4, T_3RIA, T_3U, or TSH; however, it did produce significant clinical improvement in depression ratings, which suggests that the effect of amitriptyline on depression is not related to detectable changes in endogenous thyroid function.

The efficacy of L-dopa in stimulating hGH secretion was not altered by amitriptyline therapy. No statistical difference was noted in observed hGH concentrations in depressed subjects tested before or after the start of amitriptyline or in the number of subjects who had adequate responses. We noted incidentally that depression did not affect the stimulation of hGH secretion by oral L-dopa, a finding that agrees with previous observations (10, 11).

CONCLUSIONS

This study suggests that thyroid function tests in patients taking amitriptyline are not altered by the drug itself and that these indices of thyroid function are apparently reliable. In addition, L-dopa can still be used as part of an evaluation of hGH secretion in patients on amitriptyline. The statistically nonsignificant changes in hGH secretion noted in those patients taking amitriptyline further underscores an accepted caveat that one should not rely on only one test of the integrity of hGH secretion (12).

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Anniversary Reactions Masquerading as Manic-Depressive Illness

BY JESSE O. CAVENAR, JR., M.D., JAMES L. NASH, M.D., AND ALLAN A. MALTBIE, M.D.

The authors suggest that many patients diagnosed as having manic-depressive illness, depressed type, may in fact be experiencing anniversary reactions—time-specific psychological and physiological reactions to a past trauma. They report three cases of cyclic depressive disorder, two of which were first diagnosed as manic-depressive illness, depressed type. Careful, complete interviewing and attention to temporal sequence led to the correct diagnosis of anniversary reaction.

MANIC-DEPRESSIVE illness has been recognized as a clinical entity since the age of Hippocrates. Through the years more attention has been focused on the description and causes of the disorder.

The incidence of the diagnosis of manic-depressive illness has varied considerably throughout the years. According to Cohen (1, p. 677), the incidence of the diagnosis has decreased over the past 50 years. He reported a decline of 17% to 8% in it at Boston State Hospital and a decline of 37% to 16% at McLean Hospital for the period 1900–1950. However, Cohen reported an increase in the diagnosis in general hospitals from 4,962 cases in 1951 to 6,335 in 1963.

Our general impression is that the incidence of the diagnosis is increasing. At our hospital the figures show that in 1954, no cases were recorded. However, there were 36 cases in 1964, 49 in 1974, and 57 by 1975. We believe there are several reasons why the incidence of the diagnosis is increasing.

Most authors describe three types of manic-depressive illness: manic, depressed, and cyclic. The manic patient, with pressure of speech, a disturbed sleep pattern, flight of ideas, and euphoric mood, poses little diagnostic problem. The patient who experiences severe mood swings from true mania to severe depression also poses little diagnostic difficulty. However, the patient who experiences recurrent depressions without any evidence of mania is much more of a diagnostic problem. We believe that most of these patients are increasingly diagnosed as having manic-depressive illness, depressed type, and that this fact accounts for the increased incidence of the diagnosis of manic-depressive illness. We suggest that diagnosing most recurrent depressive illness as manic-depressive illness is fraught with hazards; we believe that many of these disorders may be unrecognized anniversary reactions.

Anniversary reactions are time-specific psychological and, at times, physiological reactions that occur or recur at specific anniversaries in response to a trauma that the individual has experienced in the past and has not effectively mastered. The person attempts to master or control the trauma by reliving the experience—through symptom formation, dreams, or overt behavior—without necessarily being consciously aware that an anniversary reaction is occurring.

THE LITERATURE

The anniversary reaction was first described by Freud (2), who wrote in the case of Elizabeth von R., "This lady celebrated annual festivals of remembrance at the point of her various catastrophies, and on these occasions her vivid visual reproduction and expressions of feeling kept to the date precisely" (p. 163). Although Freud did not use the term "anniversary reaction," this is clearly what he described.

In 1920, in "Beyond the Pleasure Principle" (3), Freud attempted to explain masochism and other forms of pleasure that were not in keeping with the pleasure principle. He advanced the idea of the repetition compulsion; he felt that an individual made repeated attempts to master a traumatic situation that was so intense that it could not be mastered at the time it occurred. The clinical concept of the repetition compulsion is generally accepted today in psychiatry, and one of the more striking examples is the anniversary reaction.

Pollock (4), who has done extensive investigations on anniversary phenomena, has suggested that anniversary reactions result from incomplete or pathological mourning. He has described cases in which anniversary reactions were specific to a particular day of the week, hour of the day, or day of the year. His reported cases seem to validate his hypothesis of pathological mourning.

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Various diseases have been described as biological or somatic expressions of anniversary reactions. Bressler (5) has described the onset of ulcerative colitis as an anniversary response in which the loss of a significant external figure served to activate unconscious conflicts. Weiss and associates (6) have described hypertensive crises, occlusion of the coronary arteries, and irritable bowel syndromes as anniversary reactions. Other organic illnesses such as migraine headache (7), urticaria and dermatological conditions (8), and rheumatoid arthritis (9) have been described as anniversary reactions.

Hilgard has written extensively on anniversary phenomena and has described cases in which a parent experienced an anniversary reaction when his or her child reached the age at which the parent had experienced a trauma (10). She has also reported that depressive or psychotic symptoms may arise as anniversary reactions to the childhood death of siblings (11). In a third report (12) Hilgard presented material that described the appearance of an anniversary reaction when an adult patient reached the age at which a parent had died.

Engel (13), a psychiatrist and psychoanalyst, has recently written concerning his personal experiences with anniversary responses. He kept a detailed diary of his experiences and dreams and noted multiple examples of anniversary phenomena. These reactions were physical as well as psychological, and they seemed to be in reaction to the death of his identical twin, as well as to living through the age at which his father had died.

Cavenar and Nash (14) recently described patients with anniversary reactions who presented with physical complaints. The physical problems included psychogenic headache; back pain, depressive reaction; peptic ulcer; back pain, conversion reaction; and psychophysiological cardiac reaction. The dynamics in each case appeared to be an unresolved pathological grief reaction to a loss. The patients all responded to antidepressant medication and psychotherapy.

Cavenar and associates (15) also described a psychosis occurring in a young man when he reached the age at which his older sister had experienced an acute psychosis, and anniversary responses in two siblings after the father's death. These cases again seemed to be related to a pathological grief reaction.

The concept of anniversary phenomena was recently discussed in the lay press by Fishbien (16), who interviewed Pollock concerning his contributions to the field. In that report heart attacks, pleurisy and pneumonia, phobic fears, and successful suicides were identified as anniversary responses.

The purpose of this paper is to report several cases of cyclic depressive disorder, two of which were diagnosed by the referring physician as being manic-depressive illness, depressed type. It was only by careful interviewing and attention to temporal sequence that the underlying dynamics of anniversary reaction were ascertained. The following cases are illustrative.

CASE REPORTS

Case 1. The patient, a 24-year-old housewife and mother, sought psychiatric evaluation in April 1976 because of severe depressive symptoms. She was sleeping only about 2 hours per night, being awakened by marked anxiety, dreams, or nightmares. She had gained about 9.1 kg and felt she "had to eat everything in sight." She had frequent crying spells and felt as though life had no meaning for her. She had thought of committing suicide and had planned to do it by traffic accident so that it would appear to be accidental.

The mental status examination revealed a woman who was coherent, logical, and relevant and showed no evidence of psychosis or organic brain syndrome. She was profoundly depressed, had marked psychomotor retardation, and admitted to the symptoms noted above. She remarked that she had had similar episodes for the past 2 years, at the same time of the year, and had been told by a physician that she had manic-depressive illness, depressed type. She had been treated in the past with lithium carbonate, with a gradual resolution of the depression, and the lithium had been discontinued after the depression cleared.

The patient was finally able to date the onset of the symptoms of the current depressive episode to around Christmas 1975. Upon reflecting more, she realized that it was December 26, 1975, as well as December 26, 1974 and 1973, that had heralded the onset of the depressive symptoms. The depressive episode in 1973 had lasted for about 2 weeks and the one in 1974 for about 1 month; the current depressive episode was more intense both in severity and length.

The patient realized that her mother, with whom she had been very close, had entered a hospital on December 26, 1972, for a diagnostic evaluation. A diagnosis of carcinoma had been established, and the mother had survived until early June 1973. In spite of the fact that the mother lived another 6 months, it was "as though my relationship with her ended the day she entered the hospital." In many ways this date was more significant for the patient than the date on which the mother actually died.

The husband was interviewed, and he was even more emphatic than the patient about the significance of December 26. He talked of his wife retiring on Christmas night in good spirits, only to awaken the following morning profoundly depressed. He confirmed that this had occurred for 3 consecutive years and that this was the worst episode she had experienced.

It was recommended that the patient be hospitalized immediately, but she and her husband refused. She was started on therapeutic doses of amitriptyline and seen in insight-oriented psychotherapy. It became obvious during the brief psychotherapy that this woman had an unresolved grief reaction to a very significant loss and that her depressive episodes were on an anniversary basis. She obtained some insight into the unresolved grief, worked through many feelings, and clinically recovered from her depression.

Case 2. This patient, a 42-year-old married woman, was seen in psychiatric referral from her family physician. She complained of depressive symptoms, including crying spells, early morning awakening, anorexia with a 6.8 kg weight loss, and suicidal rumination. These symptoms had been present for 6 weeks prior to psychiatric evaluation and were gradually worsening. The referring physician had made a tentative diagnosis of manic-depressive illness, depressed type.

The mental status examination revealed a woman with profound depression. The affect was one of depression; she

cried readily and admitted to the symptoms noted above. She was not psychotic and had no organic impairment. The conversation revealed that she was preoccupied with the death of her son 5 years earlier.

The history revealed that the patient and her son, who was 12 years old at the time, were involved in an auto accident. She was only slightly injured, but her son was killed instantly. Her severe guilt over her son's death was apparent, and she related the onset of her depression and guilt to the fifth anniversary of the boy's death. She remarked that the other anniversaries did not seem to bother her and she was at a loss to explain why the fifth anniversary would do so.

During the course of the interview it was learned that when the patient was 5 years old, a younger male sibling had died suddenly of "summer diarrhea." She recalled that she had been very close to this brother but had not thought of him in years.

It seemed clear that the patient was suffering from an anniversary reaction based on unresolved grief over her son's death and also her brother's death many years earlier. Unconsciously she viewed the fifth anniversary of her own son's death as a double loss, corresponding to the age at which she had lost her own brother.

The patient was encouraged to ventilate her feelings over a period of 4 visits and was treated with appropriate levels of tricyclic antidepressants. She completely recovered from her depression in 1 month and returned to her usual level of functioning.

Case 3. This patient, a 60-year-old married man, was admitted to the urology service for prostatitis and sepsis. Psychiatric evaluation was requested because of his feelings of depression and crying spells. There was a history of similar episodes of depression with crying outbursts for each of the past 10 years, always between January and March. The patient's symptoms were more intense and severe this year than ever before. He could offer no explanation as to why he experienced the recurrent depressions in such a pattern.

The mental status examination revealed a man who had no symptoms of psychosis or organic impairment. He had a depressed affect and would burst into uncontrollable crying when he discussed reaching age 60 or the death of his mother, father, and brother. There was no sleep disturbance and he denied suicidal ideation.

The history revealed that the patient had been inducted into military service in early January 1942. At that time he was single and lived on a farm with his mother and father. He tried unsuccessfully to obtain deferment because his mother was critically ill. She died in March 1942, and the patient had had the fantasy since her death that if he had been allowed to remain at home, she would have suffered less or even survived. His father died 8 years later, in February. In addition, the patient's older brother had died at age 60 eight years earlier.

From the interview it became clear that the patient's recurrent depressions were an anniversary response. He had had multiple past traumata in January, February, and March, leading to unresolved grief reactions and recurrent depressions. The current depression was much worse than the others because he had now reached the age at which his brother had died; this, compounded by the previous trauma, deepened the severity.

Antidepressants were not required for the alleviation of the patient's depression; discussion of the genesis of the depression, plus sympathetic support, provided symptomatic relief. He was referred for outpatient psychotherapy in another city upon discharge from the hospital. It is hoped that additional insight into the dynamics of his depression, and an opportunity to grieve his losses in the psychotherapy, will prevent recurrences of the anniversary depressive reactions.

This patient could easily have been diagnosed as having manic-depressive illness, depressed type. The history of recurrent depressive episodes over a 10-year period, occurring at the same time of year, would tend to support that diagnosis. It was only through careful and complete interviewing that the true diagnosis became clear.

DISCUSSION

It was more than a theoretical and clinical exercise to establish the correct diagnosis and to understand the dynamics in these cases. The treatment of manic-depressive disorder with lithium carbonate for an extended period of time is not without hazard. Further, these patients would have benefited little, if at all, from lithium therapy, and it might have erroneously been assumed that they were treatment successes until the next anniversary reaction occurred. The implications of manic-depressive illness, from a social, professional, and financial standpoint, are quite different from the implications of an anniversary reaction.

In our experience the prognosis is good with anniversary depressive reactions if the correct diagnosis is established and proper treatment, consisting of antidepressant medication and insight-oriented psychotherapy, is instituted. We believe that many cases diagnosed as manic-depressive illness, depressed type, are actually anniversary reactions masquerading as manic-depressive illness.

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Urinary Catecholamine Metabolites in Hyperkinetic Boys Treated with *d*-Amphetamine

BY WALID O. SHEKIM, M.D., HAROUTUNE DEKIRMENJIAN, PH.D., AND JAMES L. CHAPEL, M.D.

The authors studied the excretion of 3-methoxy-4-hydroxyphenyl glycol (MHPG), normetanephrine (NM), metanephrine, and homovanillic acid in 7 hyperactive and 12 control children. MHPG was lower but NM was significantly higher in hyperactive children than in controls. Administration of d*-amphetamine for 2 weeks depressed the levels of MHPG, NM, and metanephrine. The authors suggest a decreased central norepinephrine activity, which may be secondary to the interruption of norepinephrine fibers in the medial forebrain bundle.*

RECENT LITERATURE on the etiology of the hyperactive child syndrome (alternately called minimal brain dysfunction) suggests a variety of causes (1, 2), but it is generally agreed that a disorder in the catecholamine system, in the metabolism of either dopamine (DA) (3–7) or norepinephrine (NE) (8, 9) plays an important part in the causation. Since both of these catecholamines produce many of the same behavioral actions, there is some doubt as to which is really at fault.

A number of investigators suggest that 3-methoxy-4-hydroxyphenyl glycol (MHPG) is the main metabolite of central nervous system NE of various species, including man (10). It is also generally recognized that urinary MHPG derives, in part, from the central nervous system as well as from the sympathetic peripheral nervous system, but the percentage of MHPG that comes from the brain has not been determined; it is estimated that 30–50% of urinary MHPG comes from the metabolism of NE in the central nervous system of dogs and monkeys (11). In contrast, most if not all of urinary normetanephrine (NM) and metanephrine comes from the peripheral nervous system metabolism of NE and epinephrine, respectively (11, 12), while urinary homovanillic acid (HVA) derives from both central and peripheral metabolism of DA (13). It has been suggested that simultaneous assays of several of the catecholamine metabolites might allow one to make inferences as to the disposition of NE in central versus peripheral pools (14). *d*-Amphetamine, which has been shown to be clinically effective in controlling the hyperkinesis of about 70% of treated hyperactive children, has been shown to alter NE and DA metabolism (15).

This study attempts to present evidence that will support a relationship between central NE metabolism and hyperactivity and to demonstrate the effects of *d*-amphetamine on the central and peripheral metabolism of the catecholamines as reflected in the urinary metabolites studied (HVA, MHPG, NM, and metanephrine).

METHOD

We considered hyperactive boys, ages 7–12, of borderline intelligence or better and without gross neurological abnormalities for the study. If they met the cri-

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teria for diagnosis of hyperkinetic reaction of childhood by *DSM-II* and by the children diagnostic classification and children diagnostic scale of the National Institute of Mental Health-Early Clinical Drug Evaluation Unit Program (NIMH-ECDEU) Assessment Battery for Pediatric Psychopharmacology, they were admitted to a research ward after having been free of all drugs for at least 2 weeks. (The first 3 children were studied in a preadolescent inpatient unit where W.S. is the clinical director.) Informed consent was obtained from all the children and their parents. All children were given a physical examination, the physical and neurological examination for soft signs, and an EEG. A complete psychological, educational, and behavioral evaluation was also made, the results of which will be given in a future report.

Twenty-four-hour urines on two successive days were obtained from 7 hyperactive children and 12 normal children (mean age=9.5±1.58 years and 10.5±1.25 years, respectively; p=n.s.) to measure HVA, MHPG, NM, and metanephrine. Diet control was not used in this pilot study, since its value in urinary catecholamine metabolites studies has recently been questioned (16, 17). Every effort was expended to make the urine collection as complete as possible (e.g., a Texas catheter was used on the one enuretic child). The biochemical assays were done by H.D., who had no knowledge of whether the urines were from normal children (controls) or hyperactive children (subjects). The methods employed for assay of metabolites have been described previously (11, 18). The subjects were discharged on *d*-amphetamine (0.5 mg/kg of body weight daily) and readmitted at the end of 2 weeks, and the metabolites were measured again. The controls, matched for age and sex, were compared with the subjects for metabolite excretion but were not given medication or placebo. Four 24-hour urine samples were collected from each normal and hyperactive child.

RESULTS

Values are expressed in micrograms per 24 hours and micrograms per milligrams of creatinine. Gitlow and coworkers (19) have shown that the excretion of catecholamine metabolites varies according to age, weight, and body surface; this variability can be decreased by expressing the results in micrograms of metabolite per milligram of urinary creatinine, which eliminates the problem of incomplete 24-hour urine collections. Mean creatinine values were 652±184 mg for controls and 560±147 mg for subjects; the subjects were physically smaller than the controls (mean weights were 27.5±5.4 kg and 31.04±6.66 kg, respectively). The differences in creatinine and weight means were statistically nonsignificant.

The differences in metabolite levels (expressed in micrograms per 24 hours) between subjects and controls were nonsignificant for HVA and metanephrine but were significant for MHPG and NM (table 1). Subjects excreted significantly lower levels of MHPG and significantly higher levels of NM than controls did. This finding is of some interest, since urinary NM is derived exclusively from the peripheral nervous system and is metabolized exclusively to MHPG. Increased metabolism of NE in the peripheral nervous system would lead to an increase in urinary NM and to an increase in the urinary MHPG coming from the metabolism of NE and NM in the periphery; therefore, a normal or low total urinary MHPG would indicate a decrease in the MHPG derived from the metabolism of NE in the central nervous system. The ratio of MHPG to NM may be considered an indication of central noradrenergic activity relative to its peripheral noradrenergic activity. The MHPG to NM ratio in our study was 6.9±1.7:1 for subjects and 11.4±3.2:1 for controls. The difference between the ratio was statistically significant. Others have used the MHPG to VMA (4-hydroxy-3-methoxymandelic acid) ratio similarly (20).

The administration of daily *d*-amphetamine significantly depressed the excretion of MHPG, NM, and M in hyperactive children compared with excretion of these metabolites in a drug-free state (table 1) and in controls. MHPG to NM ratio and HVA levels were not significantly changed by *d*-amphetamine.

DISCUSSION

Wender and associates (21) studied catecholamine metabolites in 9 children with minimal brain dysfunction and did not find significant differences between them and 4 control subjects. They also could not demonstrate any effect of *d*-amphetamine administration. Their study, however, did not describe the criteria for selection of the subjects; it included boys and girls, and amphetamine dosage and duration of treatment varied within the group. Shaywitz and associates (7) demonstrated a decrease in HVA accumulation in the CSF of 6 children with minimal brain dysfunction after probenecid ingestion, suggesting a decreased DA turnover in the central nervous system. Furthermore, they selectively reduced DA in rat brain by injecting 6-hydroxydopamine in an experimental animal model for minimal brain dysfunction and produced rat pups who were significantly more active than controls and who exhibited avoidance learning deficits (5).

Our finding of a low MHPG to NM ratio in subjects compared with controls suggests depressed central NE activity in the hyperactive child syndrome. The finding of significantly lower urinary MHPG levels in a group of depressed patients compared with controls while their urinary NM and metanephrine were the same as that of controls was interpreted by Maas and associates (22) as being consistent with and supportive of the catecholamine hypothesis of the affective disorders. This may also suggest cortical depletion of NE and subsequent depletion of its metabolite MHPG.

In animals, decreased urinary MHPG levels have

TABLE 1
Urinary Metabolite Levels of Hyperactive Boys, Drug-Free and After 2 Weeks on *d*-Amphetamine, and Control Boys

Metabolites	Drug-Free Hyperactive Boys (N=7)		Control Boys (N=12)			Hyperactive Boys (N=6)**				
						Drug-Free		After 2 Weeks on *d*-Amphetamine		
	Mean	SD	Mean	SD	Significance*	Mean	SD	Mean	SD	Significance*
MHPG										
μg/24 hours	806	153	1044	264	p<.05	820	163	481	155	p<.01
μg/mg of creatinine	1.51	0.36	1.66	0.33	p=n.s.	1.45	0.35	0.92	0.19	p<.01
NM										
μg/24 hours	125	40	94	24	p<.05	116	35	64	12	p<.01
μg/mg of creatinine	.234	.098	.150	.038	p<.02	.205	.06	.125	.028	p<.01
MHPG to NM ratio	6:9	1.7	11.4	3.2	p<.01	7.4	1.2	7.5	2.2	p=n.s.
Metanephrine										
μg/24 hours	85	25	74	19	p=n.s.	85	28	48	17	p<.05
μg/mg of creatinine	.154	.031	.117	.030	p<.05	.146	.026	.094	.028	p<.02
HVA										
μg/24 hours	2828	490	3212	736	p=n.s.	2907	485	2606	1020	p=n.s.
μg/mg of creatinine	5.2	0.92	5.15	1.32	p=n.s.	5.16	1.0	5.01	1.3	p=n.s.

*Two-tailed Student's t test.
**There were only 6 hyperactive boys in this group because 1 boy developed tic-like movements, his hyperactivity worsened on *d*-amphetamine, and he was removed from the study before any data could be obtained for him while medicated.

been produced by the intraventricular administration of a neurotoxin that is believed to destroy catecholamine neuronal processes. For example, Breese and Traylor (23) found that an 82% depletion in rat brain NE stores following 6-hydroxydopamine administration resulted in a 29% decrease in total urinary MHPG. In nonhuman primates, Maas and associates (24) found that a 72% decrease in brain NE following 6-hydroxydopamine administration resulted in a 32% decrement in total urinary MHPG. Lesions to NE fibers in the medial forebrain bundle in animals have been shown to result in cortical depletion of NE and subsequent depletion of its metabolites (25).

Our findings and Shaywitz and associates' finding (5–7) of decreased DA levels and turnover in the brain may very well be related, since it has been suggested that NE may play a modulator role in the control of motor activity and its action may be facilitation of the nigro-neostriatal DA system of the brain (26). Andén and Grabowska (27) presented pharmacological evidence for a stimulation of DA neurons by NE neurons in the brain. Donaldson and associates (28, 29) presented evidence consistent with a proposed ceruleus-nigral NE pathway that enhances impulse flow in dopaminergic nigrostriatal system.

The reduction of MHPG levels by *d*-amphetamine in our study is in keeping with similar findings in some adult depressed patients demonstrated by Fawcett and associates (30) and Beckmann and associates (15). Amphetamine acts at the catecholamine-containing nerve terminals and results in an increase in the availability of neurotransmitter to postsynaptic receptors (31). Corrodi and associates (32) hypothesized that this increase in neurotransmitter level leads to an inhibition of catecholaminergic cell activity via neuronal feedback circuits. Bunney and Aghajanian (33, 34) presented evidence to support this hypothesis; from their data there is reason to believe that feedback control is present in the NE system and originates in the locus ceruleus, since amphetamine decreases activity in the locus (35). Bunney and Aghajanian (35) have submitted electrophysiological evidence supporting the view that central catecholaminergic neurons possess presynaptic receptors for their respective neurotransmitters and that these receptors are responsive to specific agonists and antagonists. Stimulation of presynaptic NE receptors through microionophoresis of NE or its agonist, clonidine, onto the soma of NE neurons inhibits the activity of these cells. Moreover, Walters and associates (36) demonstrated that both *d*- and *l*-amphetamine inhibit the firing of locus ceruleus neurons. Amphetamine may exert its action in hyperactive children through feedback inhibition of brain stem NE neurons.

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In Vitro Half-Life of ^{14}C-Tryptamine in Whole Blood of Drug-Free Chronic Schizophrenic Patients

BY EDWARD F. DOMINO, M.D., AND SHEILA GAHAGAN

The authors found higher in vitro ^{14}C-tryptamine half-lives in the blood of 10 drug-free chronic schizophrenic patients than in the blood of 13 mentally normal control subjects. This finding was negatively correlated for both groups with platelet MAO activity. Repeated determinations of in vitro ^{14}C-tryptamine half-lives over a period of 5 months were positively correlated for the patients, although some blood samples showed statistically significant variability.

THE LITERATURE REGARDING platelet monoamine oxidase (MAO) activity in chronic schizophrenic patients is controversial. Many investigators have reported that platelet MAO activity is reduced in such patients (1–6), but others report normal values (7–10). At least four studies have shown that brain MAO activity in deceased chronic schizophrenic patients is normal (11–14), yet evidence that MAO inhibitors exacerbate psychotic behavior suggests that biogenic amines must be involved in such disorders (15). Of special interest is tryptamine, a substrate for both A and B type MAO (16) and a hallucinogen when given intravenously to human volunteers (17). One would predict that some drug-free chronic schizophrenic patients would have prolonged tryptamine blood half-lives both in vitro and in vivo. Inasmuch as an in vitro study was much simpler and would not have the potential to temporarily exacerbate the patient's illness, we decided to test this obvious hypothesis.

METHOD

We studied 10 drug-free chronic schizophrenic patients and 13 mentally normal adult men. Only patients and control subjects who signed informed consent forms were included. This study was approved by the Institutional Review Board of the Lafayette Clinic and the Technical Advisory Committee for Research of the Michigan Department of Mental Health.

All patients were diagnosed independently as chronic schizophrenics by three to five psychiatrists and were hospitalized in a special drug-free chronic schizophrenic ward at Lafayette Clinic. These patients had originally been selected for this ward because they failed to respond to antipsychotic drug therapy (dopamine antagonists). Most of the patients had been drug-free for approximately 10 years and had been mentally ill since adolescence or young adulthood. All patients exhibited thought disorder, disturbance of association, autistic behavior, and altered affect. The patients were between 30 and 46 years old and were on a diet of 2,300 calories and one vitamin pill (Myadec) a day. Recreational therapy included vigorous exercise each day.

The control subjects were 20- to 50-year-old men, chosen from Lafayette Clinic personnel. They had normal medical histories and psychiatric status. All controls were drug-free from 1 to 2 months before the test, excluding occasional use of acetylsalicylic acid, alcoholic beverages, and cigarettes. Acetylsalicylic acid and alcohol were not allowed for 24 hours before the test.

All patients and control subjects fasted overnight before blood withdrawal. Venous blood, 35 ml, was taken between 7:30 and 8:00 on the morning of the test. Heparinized whole blood was immediately transferred in 3-ml aliquots to 11 beakers in a Dubonoff shaker waterbath. The blood was incubated at 37°C in an atmosphere of 95% O_2 and 5% CO_2.

At the starting time 0.1 ml of 10^{-5} M ^{14}C-tryptamine was added to 10 of the 3-ml aliquots of blood. The total amount of tryptamine added to each sample was 1.1 μg containing .02 μCi or 44,000 disintegrations per minute of radioactivity. The 11th sample was a control to which no ^{14}C-tryptamine was added.

The blood samples were incubated for 0, 5, 30, 60, and 120 minutes. At each of these time intervals 5 ml of 10% trichloroacetic acid (TCA) was added to 2 of the blood samples to stop the reaction. The samples were then homogenized with the 10% TCA, and 4 ml 10% TCA was added in two successive washings. The total 12 ml of blood and TCA were basified with 2 ml of

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10 N NaOH. The ^{14}C-tryptamine was extracted into a 50%-50% mixture of ethyl acetate and toluene and reconcentrated in 2 ml of 0.1 N HCl. This was again basified and extracted in ethyl acetate and toluene. At this point 2 ml of the final organic layer was mixed with 10 ml of Aquafluor and counted in a Beckman scintillation counter. Earlier, many of the patients and controls were studied for their platelet MAO activity using the same substrate, ^{14}C-tryptamine. The methods used have been described previously (5).

The data were analyzed on an IBM 1800 Process Control Computer. First the total disintegrations per minute counted were converted to nanograms of ^{14}C-tryptamine per milliliter of blood, which was then plotted against time. Half-life of ^{14}C-tryptamine in human blood was calculated using Gauss's nonlinear regression analysis.

RESULTS

The in vitro blood ^{14}C-tryptamine disappearance data for the 13 controls and 10 drug-free chronic schizophrenic patients are summarized in tables 1 and 2. The amount of ^{14}C-tryptamine in the blood of controls tended to decrease more rapidly. When each individual's data were used to determine in vitro half-life, the scattergram showed much greater variability among the patients than the controls (see figure 1). The mean half-life was significantly longer for patients than for controls—110.9±18.4 minutes versus 69.5±8.0 minutes, respectively ($p<.01$).

The mean platelet MAO activity for 9 patients was 3.34±.19 nmol/mg of protein per hour and 6.38±.83 nmol/mg of protein per hour for 8 controls. This difference, which was statistically significant ($p<.01$), has been reported previously (5). When platelet MAO activity of each subject was correlated with his whole blood ^{14}C-tryptamine half-life, the correlation coefficient was negative, as might be predicted ($r=-.45$; $p<.07$, two-tailed test; $p<.035$, one-tailed test).

Six of the 10 patients were retested 5 months later. Three showed very similar tryptamine half-lives on the first and second tests, but 3 varied markedly. The mean ^{14}C-tryptamine half-lives were 112.5±22.9 minutes and 181.3±37.2 minutes, respectively ($p<.02$). The two values for each subject correlated positively ($r=+.87$, $p<.03$).

TABLE 1
^{14}C-Tryptamine in Whole Blood In Vitro of Mentally Normal Controls and Drug-Free Chronic Schizophrenic Patients

Incubation Times (minutes)	^{14}C-Tryptamine (ng/ml per minute)				
	Controls (N=13)		Patients (N=10)		
	Mean	SE	Mean	SE	Significance
0	25.3	0.7	27.8	0.5	$p < .05$
5	22.9	0.6	23.8	0.4	$p > .10$
30	16.1	0.9	20.3	0.9	$p < .005$
60	11.6	1.0	16.5	1.4	$p < .01$
120	7.2	1.0	11.1	1.6	$p < .05$

TABLE 2
Rate of Disappearance of ^{14}C-Tryptamine in Whole Blood In Vitro of Mentally Normal Controls and Drug-Free Chronic Schizophrenic Patients

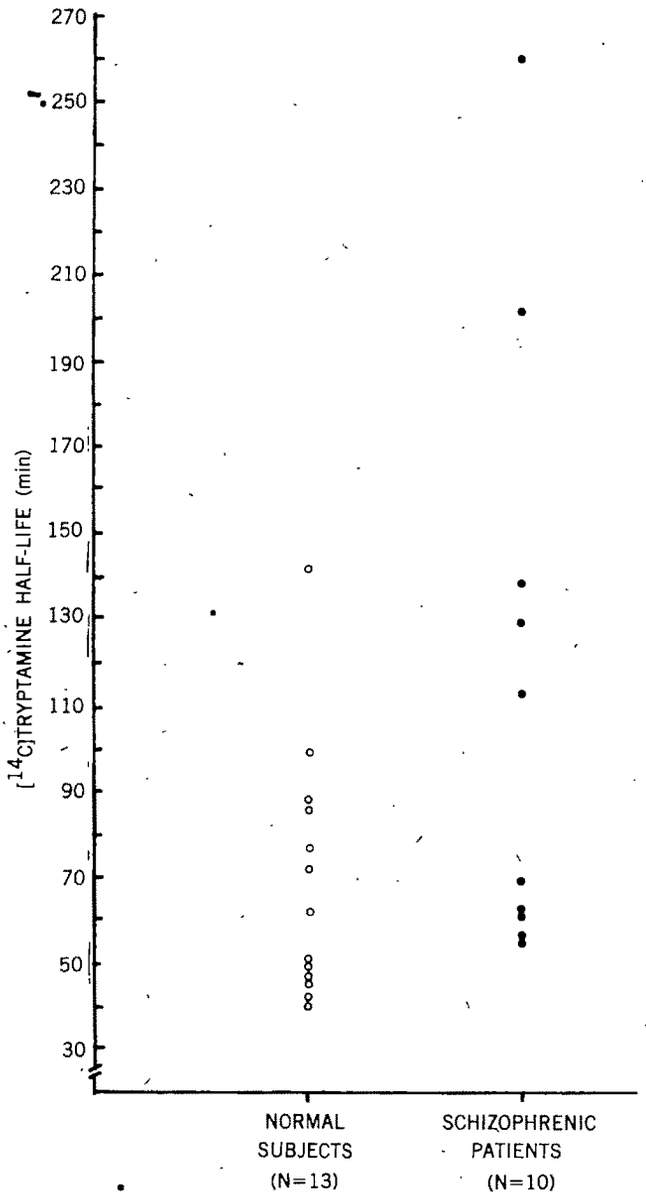
Time Period (minutes)	Change in ^{14}C-Tryptamine (ng/ml per minute)				
	Controls (N=13)		Patients (N=10)		
	Mean	SE	Mean	SE	Significance
0 to 5	.73	.09	.80	.10	$p > .10$
0 to 30	.35	.02	.25	.03	$p < .01$
0 to 60	.25	.01	.19	.02	$p < .05$
0 to 120	.16	.01	.14	.01	$p > .10$
5 to 30	.28	.02	.14	.02	$p < .001$
30 to 60	.16	.01	.13	.02	$p > .10$
60 to 120	.07	.00	.09	.01	$p > .10$

DISCUSSION

The present findings stress again a biologic difference in the way the blood of chronic schizophrenic patients oxidizes a known hallucinogen, tryptamine, which is a trace endogenous brain amine (18). The fact that 3 of the 6 patients had widely different blood half-lives on test-retest conditions indicates that nongenetic factors must be very important. Wyatt and associates (2) have shown, using monozygotic twins divergent for schizophrenia, that platelet MAO activity was reduced in each twin. Although platelet MAO activity may be genetically predetermined, environmental factors must also be involved. The recent studies of Belmaker and colleagues (10) as well as Owen and associates (9) and others (7, 8), in which no difference in platelet MAO activity was found in chronic schizophrenic patients and normal controls, indicate that low platelet MAO activity cannot be regarded as a genetic marker for schizophrenia.

Inasmuch as whole blood contains a plasma amine oxidase as well as platelet MAO, in vitro studies of whole blood are relatively crude because one is measuring at least two different enzyme activities. Future studies of the activity of plasma amine oxidase should be compared to other monoamine oxidases such as those in the platelets of schizophrenic patients. However, the purpose of our study was to demonstrate an in vitro difference in tryptamine half-lives. Measurement of the presumed end product indoleacetaldehyde and its subsequently oxidized product indoleacetic acid would be a direct test for the involvement of oxidase enzymes. Disappearance of substrate is only indirect evidence that oxidases are involved since other products may be formed from tryptamine.

FIGURE 1
Scattergram of ^{14}C-Tryptamine Blood Half-Life In Vitro for Mentally Normal Controls and Drug-Free Chronic Schizophrenic Patients*



*The mean half-life was 110.9±18.4 minutes for patients and 69.5±8.0 minutes for controls ($p<.01$).

The half-life measurements we obtained in both normal subjects and chronic schizophrenic patients seemed to us surprisingly long. This would suggest our conditions for incubation were not ideal. Nevertheless, our study clearly indicates that further research, especially measuring in vivo tryptamine half-life in chronic schizophrenic patients and normal controls, is warranted. By using small intravenous doses of labeled tryptamine of high specific activity, one should be able to avoid hallucinogenic reactions (17) and do pharmacokinetic studies in schizophrenic patients. A tryptaminergic metabolic abnormality in some chronic schizophrenic patients resistant to antidopamine neuroleptic therapy still seems tenable.

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The Hearst Trial and the Confidentiality of Residency Records

BY LENORE C. TERR, M.D.

Review of the Hearst trial transcript reveals that the subpoena of Dr. Joel Fort's residency files met with very little effective resistance on the part of the hospital that had trained him. There are few legal mechanisms to stop such a subpoena. The only truly effective solution may be the abstraction and destruction of residency files once the residency has been successfully completed.

NOW THAT THE Hearst trial is over, one question remains that must not escape the attention of all physicians: Why did Herrick Hospital give Dr. Joel Fort's residency files to F. Lee Bailey's team without a legal fight? Joel Fort, M.D., an expert witness for the government, had served his psychiatric residency at Herrick Memorial Hospital, Berkeley, Calif., in 1957 and 1958. In his cross-examination of Fort, Bailey produced some negative opinions about Fort's competence as a resident from his 18-year-old file. These opinions had been written by supervising psychiatrists on the hospital staff. The residency records were subpoenaed by the Hearst lawyers.

THE EXPERT WITNESS AND THE LAW OF EVIDENCE

An expert psychiatric witness must establish that he or she has skill, knowledge, and experience in the field of psychiatry. These qualifications must be demonstrated in direct testimony so that inferences and opinions made by the psychiatrist are admitted into evidence. Expert opinion and inferences are allowed by the courts in order to aid the judge and jurors with their understanding of the facts and to help them reach true and just conclusions.

On cross-examination of an expert, the attorney has the legal right to attempt to "elicit answers which will impeach the veracity, capacity to observe, impartiality, and consistency of the witness . . ." (1). Furthermore, the attorney has the right to ask questions that will impeach the witness' qualifications to aid the jury at all or make him appear to be less qualified than the psychiatric witnesses appearing for the cross-examining attorney.

Psychiatrists planning to appear in court are well-advised to read the chapters on cross-examination by Goulett (2) and Ziskin (3), both of whom are attorneys. Both authors explore in detail various methods of discrediting psychiatrists. Goulett, relying upon methods other than attacking the psychiatrist "within the heart of his field" (i.e., *why* he made the diagnosis), suggests impeaching the psychiatrist's credibility by asking about failed Board examinations, lack of forensic experience, too much forensic experience, or contradicting writings by eminent psychiatrists. Ziskin's cross-examination suggestions are aimed at discrediting the general field of psychiatry altogether. Although these authors recommend a thorough search for impeaching questions, neither suggests opening the residency file, as was done in the Hearst case. While this new tactic may be acceptable to the courts, it is most disturbing to training directors, department chairmen, and psychiatrists in general. In the future, it is very likely that many attorneys will use residency records in their armamentarium of cross-examination techniques.

THE CHAIN OF EVENTS

The following chain of events was reconstructed from the transcript of the Hearst trial.[1] On March 9, 1976, at about 9 a.m. the Fort file was subpoenaed, to be delivered to the court by about noon. Herrick Hospital's assistant administrator, Mr. Passama, appeared in court with two attorneys, Mr. Vlahos and Mr. Kessenick. In addition, a representative of Herrick Hospital phoned Mr. Davis, an attorney whose name had been mentioned in the newspapers in connection with some legal transactions of Dr. Fort's, to ask him to appear in court to represent Dr. Fort. Dr. Fort stated during cross-examination that he had not been notified by the hospital and did not know that an attorney would appear on his behalf (4, 3693:3–18). It is not usual and is not good court "etiquette" for an expert witness to be represented by counsel because it might detract from the main issues of the case.

At the time the records were delivered on March 9, the attorneys discussed the potential use of the residency files. The jury was not present. Dr. Fort's lawyer objected and attempted to quash the subpoena be-

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[1]Citations are to the page and lines of the reporter's verbatim transcript of *U.S. v. Hearst* (4).

cause it was too broad, too remote in time, and was an abuse of confidence and privilege (4, 3483:11–25, 3484:1–25). In explaining what confidence was involved, Mr. Davis objected to the presence of another resident's name in Dr. Fort's file. The court and Bailey agreed that the other psychiatric trainee, Dr. "X," would not be mentioned by name. Judge Carter pointed out to Mr. Davis that there was no federal privilege extended to trainees, and there was no doctor-patient privilege in such circumstances. He denied the motion to quash the subpoena. The government attorney moved to quash the subpoena because Dr. Fort's file was a "side-issue, which has little to no bearing on the case whatsoever" (4, 3498:2–3). Judge Carter upheld the government's motion (4, 3501:14).

Unfortunately, there was a turn of events the next day, March 10. Mr. Browning, the government's attorney, in questioning Dr. Fort before the jury, asked him about his training. Mr. Bailey seized the opportunity to ask Judge Carter to rule again on the admission of Dr. Fort's residency file. This time Carter ruled that it could be admitted as evidence. There were no hospital attorneys or counsel for Dr. Fort present on March 10 when the residency file was used in open court in an attempt to discredit Dr. Fort's psychiatric opinion.

What was the role of the two attorneys for Herrick Hospital in the deliberations about using the residency files? They raised no serious objection. First Mr. Vlahos made the following policy statement: "We on behalf of the hospital were concerned only in that we felt any party who wanted to make his objections as to privacy and other privilege should make them" (4, 3487:8–10). Thus the hospital was allowing others to object but did not object itself to the use of its residency files. Mr. Vlahos asked that Dr. X not be mentioned, and all the attorneys agreed. Mr. Vlahos did not ask that a patient, Mrs. M., whose name and illness were disclosed in Dr. Fort's file, be protected. Finally the hospital attorney stated, "I would like to call to the Court's attention, without making any motion of any kind, a provision in California law which may or may not be applicable to these proceedings, Sections 1156 and 57 of the Evidence Code relating to medical staff records, and a privilege under California law with respect thereto" (4, 3490:2–8).[2] These rules relate primarily to medical malpractice suits. The minutes of peer review committees cannot be used in California to prove that a hospital was negligent. Thus the rules cited by the hospital did not apply. Furthermore, the hospital made "no motion of any kind" to protect highly sensitive material in its files.

On March 10, with full permission to use Dr. Fort's residency file, Bailey cross-examined Dr. Fort. Even though the judge and all of the participating attorneys had already read it the previous day, Dr. Fort had never seen his record before (4, 3693:19–25, 3694:1–19). Eleven staff psychiatrists whose statements were in Dr. Fort's record were named and discussed in the cross-examination. Mrs. M., a patient who had suffered a serious mental illness in 1958, was also named and discussed (4, 3688:11–12, 3689:13–16).

THE ISSUES

There are several issues involved in the use of residency records. First, there is the unique teacher-student relationship during residency, which is at least morally privileged if not legally privileged. Second, there is the issue of doctor-patient privilege, which applies when residency files contain patients' names and a discussion of their cases. Finally, there is the issue of the safekeeping of residency records.

The single most important factor necessitating the protection of psychiatric residency files is the quasi-patient relationship that exists between the resident and the senior staff. The resident often experiences this quasi-patient role when he or she tells the supervisor about personal problems as they relate to countertransference (5). In fact, at least one training program achieves a true doctor-patient relationship by encouraging the resident to use "an 'undirected, free-associational' technique" in supervisory hours (6), thus offering the trainee the "opportunity to use his supervisory time as therapy" (7). Returning to the more ordinary program, however, it is clear that when practicing psychiatrists work with psychiatrists-in-training, personal discussions, observations, and interpretations are made. These analyses often find their way into the psychiatrist resident's record at the training institution (8).

What are the results of exposing residency records in the courtroom? First, the lay jury will not automatically understand that supervisors' comments made during the formative residency years are meant as constructive criticism, not as a permanent blot on the psychiatrist's subsequent career. Second, because of the highly personal comments in residency files, most practicing psychiatrists will become more and more reluctant to testify. The risk of a full disclosure of material from training is serious enough to stop psychiatrists from serving as expert witnesses. Third, mental health professionals on the permanent staff of teaching hospitals may become less than candid in evaluating residents for fear of hearing their comments read in public at a later date. If this occurs, groups and institutions that rely on data from residency files (e.g., certification boards, medical organizations, hiring groups) will not receive the kind of useful information they now do. Finally, in an age of generational conflict, the disclosure of private and personal files can only add to the distrust of the young for the medical establishment.

The exposure of a patient's name was a tragic out-

[2]Section 1157 of the California Statute states, "Neither the proceedings nor the records of organized committees of medical or medical-dental staffs in hospitals having the responsibility of evaluation and improvement of the quality of care rendered in the hospital or medical and dental review committees of local medical or dental societies shall be subject to discovery."

come of the Bailey subpoena. No one protected the patient's right to confidentiality, a legally established privilege. One can only imagine the impact on Mrs. M. and her family when her mental illness of 1958 was brought up in the most highly publicized trial of the 1970s.

The third issue related to hospital records is the safekeeping of residency files. The immediate type of subpoena issued by Bailey caught the hospital unprepared. The officials of Herrick Hospital could have moved for a continuance (for a delay until their motions were prepared), but they did not. It is unlikely that Bailey would have subpoenaed this record without some clues. Bailey stated, as he attempted to offer proof to the court of the relevance of the records, "We have information he [Fort] was found not to be qualified to administer psychotherapy. . ." (4, 3488:14–15). Davis, Dr. Fort's attorney, replied, "from the information I have, that is an entirely false statement as to the fact and as to what is contained in these records. . ." (4, 3488:10–21). Bailey concluded, "I am sorry, Mr. Davis, we have talked with the administrator of the hospital. I think some of the documents I am talking about, you haven't seen" (4, 3489:1–3). All of these statements were made before the review of the residency files. From the colloquy, it is not unreasonable to infer that Bailey had been given sensitive information by a hospital administrator before the court discussion. Did Bailey actually see the record before its subpoena?

THE POSSIBLE SOLUTIONS

The use of residency files to discredit expert witnesses must be dealt with by psychiatry. Individual psychiatrists, training institutions, and organized psychiatry must be alert to the problem. The individual psychiatrist should inspect his own record if it is to be used in cross-examination. In examining it he can prepare himself for questions concerning supervisors' comments. The Buckley amendment (9, sections 438, 513) requires that the educational institution notify the student before it complies with a subpoena of its records. U.S. Department of Health, Education, and Welfare regulations for the enforcement of the Buckley amendment state that an educational institution must make "a reasonable effort to notify . . . the eligible student of the order of subpoena in advance of compliance therewith . . . (10, section 99.31 [a] [9]). Thus the Buckley amendment helps the expert witness in two ways—by allowing him to see his file in advance and by giving him prior notification that his file has been subpoenaed.

Training institutions have few, if any, effective legal grounds to quash subpoenas of their files even though they might wish to vigorously fight. However, legal delay tactics can be very effective. Particularly because the residency file is a side issue, the subpoenaing attorney may not pursue the matter if there is a legally obtained delay.

In the Hearst case, Federal Rules 611[3] and 403[4] might have been cited. Rule 611 protects the witness from harassment or undue embarrassment, and Rule 403 eliminates irrelevant evidence. State courts have similar rules (1). However, the courts tend to overrule attorneys' objections based on these rules when *expert* witnesses testify. The courts are not particularly protective of the expert, who the courts assume is appearing voluntarily and is being paid for his or her services. Furthermore, most judges would consider that information concerning residency training is relevant, regardless of how many years have passed, because the expert justifies his presence in court on the basis of his training and experience. Thus it is extremely difficult for a training institution to put up an effective fight.

At the psychiatric facility, training directors must handle residency files more carefully. Patients' names must be eliminated. Institutions must place residency files under lock and key and make them available to only a few authorized individuals.

Organized psychiatry needs to consider asking legislatures for an extension of privileges to cover trainee-institution relationships. The relationship between the psychiatric resident and the psychiatric supervisor is unique among medical specialty training relationships in that the trainee is "seeing a psychiatrist" when he or she visits his or her supervisor. It would be helpful to extend privilege to cover this relationship.

Finally, if such privilege rules are not forthcoming, psychiatric residency files must be destroyed once the residency has been completed. At present, it is extremely difficult to quash a subpoena of a residency file. Although files are useful for the first few years after graduation, they can be abstracted to include the subject matter covered, general quality of the resident's performance, and the dates the residency was begun and completed. If training institutions cannot effectively protect such records from use and misuse by investigating agencies and courts, then they must be destroyed.

To summarize, F. Lee Bailey's cross-examination of Dr. Joel Fort is a worrisome episode for all physicians, particularly psychiatrists. In the future, residency files must be zealously guarded by training institutions. If it is not possible to keep such records out of the courts, then it may be preferable to eliminate them altogether.

[3]Federal Rule 611 states, "The Judge shall exercise reasonable control over the mode and order of interrogating witnesses and presenting evidence so as (1) to make the interrogation and presentation effective for the ascertainment of the truth (2) to avoid needless consumption of time and (3) to protect witnesses from harassment or undue embarrassment."

[4]Federal Rule 403 states, "Although relevant, evidence may be excluded if its probative value is substantially outweighed by the danger of unfair prejudice, confusion of the issues, or misleading the jury or by considerations of undue delay, waste of time, or needless presentation of cumulative evidence."

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Suicide Among Police

BY MICHAEL F. HEIMAN, M.D.

The author presents and comments on recently acquired police suicide data from several cities in the United States and from the Metropolitan Police Department of London and on extensive archival police suicide files. He also explores some of the psychosocial factors that may affect the high incidence of suicide among people who actively specialize in police work.

IN RECENT YEARS considerable interest has been shown in the phenomenon of suicide (1). Suicidal events have been observed in almost all cultural, religious, and political settings in spite of the diversity of attitudes and the variety of feelings enshrouding these settings (2, 3). One setting that is highly visible and intimately involved with such issues as death and violence but is not usually considered as harboring active participants in the process of termination by self-destruction is the police department. However, the standardized mortality ratio among law enforcement officials (police, sheriffs, and marshals) is one of the highest of any occupation studied by the U.S. Department of Health, Education, and Welfare (4)—183, compared with 100 for working men in all occupations (women were excluded from the study because at the time [1950] active police work was an almost exclusively male experience). Other studies have directly or indirectly come to a similar conclusion, i.e., American policemen kill themselves in proportionately greater numbers than do members of other public service occupations (5, 6).

Although Nelson and Smith (6) focused on the sociological aspects of police work from the theoretical perspective of Durkheim, most studies are primarily statistical in nature, providing little information as to the personal qualities and experiences of law officers who have committed suicide. Consequently, there appears to be a need for assessing suicide among police not only from sociometric and sociological perspectives (perhaps with a special focus on regional variation, the nature of the police institution, and the alternatives of Durkheim) but also from a psychological viewpoint, with more emphasis on the psychological autopsy and psychodynamic speculations of the single case study.

METHOD

Under the aegis of Dr. Edwin Shneidman, I obtained access to the original files and records of 93 New York City policemen who were alleged to have committed suicide between 1934 and 1940 (7). These files and records make up a small but exciting part of the Zilboorg-Friedman Archives in the Laboratory for the Study of Life Threatening Behavior at the University of California, Los Angeles. They were given to Dr. Shneidman by the estate of the late Dr. Paul Friedman. Dr. Friedman's description of the history of these archival ma-

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terials and his work with Dr. Zilboorg and the Committee for the Study of Suicide, Inc., has been published elsewhere (8).

The police files studied are pre-World War II equivalents of what has now become known as the psychological autopsy (9). They include not only the actual police department records but also extensive interviews with any significant friends, coworkers, and family members who could shed some light on the events immediately preceding the police officer's self-inflicted death.

A second part of the research involved corresponding with the chiefs of police or the police commissioners in approximately 30 major cities throughout the United States and with the Metropolitan Police Department of London. I did this to gain some idea of the incidence of police suicides since 1960 and to compare data from these cities with Friedman's data for New York City (7).

CASE REPORTS FROM THE ZILBOORG-FRIEDMAN FILE

Case 42.[1] This patrolman had been married twice, drank a lot, was educated through the eighth grade, was Catholic, and had one younger sibling. He joined the police force during the heyday of Jimmy Walker's mayoralty in New York City. His first marriage was turbulent; he and his wife were flagrantly unfaithful to each other. During one drunken episode, he shot himself in the leg after he and his first wife had a heated argument. He said of that incident, "I almost shot her at that time." About a year and a half before his suicide and eight months after his divorce he remarried. This marriage was also unrewarding, and his second wife's behavior may have contributed to his increased irritability.

At work he was one of the most surly, irascible patrolmen in the precinct. He felt his colleagues singled him out for being derelict in his duty and for being intoxicated while on patrol. He especially focused on a sergeant as the prime architect of the disciplinary actions taken against him.

On the day of his suicide, after a very trying day with this particular sergeant, the patrolman went to a local bar and waited for the sergeant to come there. He told some fellow drinkers that he would kill the sergeant when he saw him. He was agitated and frequently walked out of the bar looking for the sergeant. On his final return to the bar he wrote a note addressed to the sergeant: "To Whom It May Concern: Goodbye, you old prick, and when I mean prick you are a prick. Hope you fall with the rest of us, you yellow bastard." He shot himself in the head after finishing this note.

Case 43. This patrolman was a "good cop." He had been on the force for 12 years and, in spite of the rampant corruption of the department during the 1920s, had never taken a bribe. He was the eldest of five siblings and had been raised in a good Catholic household. He had taken on the sole responsibility of raising his younger siblings when his mother died during his teenage years. He had managed to go through one year of high school.

During the period near his death, he was suffering from uncontrolled diabetes and was probably going blind. He may have taken an overdose of insulin in a suicidal gesture just a few days before his actual death. At that time he was revived and told to return to work. The night before his death, he asked his wife if she knew another man to fall back on. During the day of his death, he seemed preoccupied with thoughts about dying. All of his debts had recently been paid up. He shot himself in the head, leaving a note indicating despondence over his uncontrolled diabetes.

[1]This case was also described by Friedman (7).

RECENT SUICIDE STATISTICS

Much of the descriptive and statistical data collected on New York City police during the heyday of Tammany Hall and in the early years of Fiorello La Guardia's mayoralty can be found in Friedman's work (7) and in the Zilboorg-Friedman Archives. More recent statistical data from the Metropolitan Police Department of London and from the New York City Police Department have been published elsewhere (10, 11). Unpublished data on the method of suicide, number of years on the police force, age, and marital status of the London police suicides since 1960 are shown in table 1.

It is clear from the London data that the typical police suicide for that city was married, in his early 40s, had been on the force for nearly 20 years, and died by gas inhalation or drug ingestion. None of these policemen used a gun as a means of committing suicide. The current situation for the United States is more nebulous, as table 2 shows. There seems to be a regional difference in the police suicide patterns in the United States: the east and west coasts seem to have more than the midwest and the south. The use of handguns was the overwhelmingly most common means of self-inflicted death.

According to data from police departments for which additional information was available, the average policeman who suicided was in his late 40s, was divorced or seeking divorce, was at or near retirement after 15 or more years of service, and had difficulties surrounding the use of alcohol. This picture is different from that in London in marital status and the method of suicide.

COMMENT

One interesting sidelight of this research is that it pointed out the difficulty of obtaining consistent and reliable data for useful comparisons among police departments in the United States. The Zilboorg team in New York City had the blessing of Mayor La Guardia for their research activities. Modern police departments, however, have a spectrum of opinions concerning the usefulness of this type of research; consequently, they are often reluctant to admit that such information is available. Even if the information is available, it is usually incomplete. Perhaps the best way to obtain such information in the future is for a

TABLE 1
Suicide Data from the Metropolitan Police Department of London

Suicide	Year	Method of Suicide	Marital Status	Age	Number of Years of Service
1	1960	Not recorded	Married	43	24
2	1960	Not recorded	Married	55	29
3	1960	Coal gas poisoning	Married	47	25
4	1961	Coal gas poisoning	Married	46	24
5	1961	Coal gas poisoning	Single	53	14
6	1961	Coal gas poisoning	Married	39	14
7	1964	Sodium amytal poisoning	Married	26	7
8	1965	Overdose of drugs	Married	31	9
9	1967	Hanging	Single	42	20
10	1967	Barbiturate poisoning	Married in 1956; divorced in 1960; remarried in 1962	43	14
11	1967	Threw himself under a train	Married	34	11
12	1968	Knife wounds	Married	47	23
13	1969	Hanging	Married	51	22
14	1970	Inhaled exhaust fumes	Married	40	15
15	1971	Asphyxiated in plastic bag	Married	47	23
16	1972	Not recorded	Married	22	3

TABLE 2
Suicide Data from Police Departments of 23 Major U.S. Cities*

City	Number of Suicides	Comment
Albuquerque	0	1 former policeman committed suicide
Atlanta	0	"Unaware of any since 1960"
Boston	4	All by handgun
Dallas	0	No suicide of active department member in 20 years
Denver	Unknown	46 officers met violent death between 1890–1971; none appeared to be suicides; 77% were homicides
Detroit	About 1 a year	Information confidential
Honolulu	5	
Little Rock	0	
Los Angeles	Unknown	Department does not keep statistics
Miami Beach	2	". . . during entire existence of department"
Minneapolis	At least 2	No accurate records but at least 2 known
Newark	5	4 by handgun; 1 by drug overdose
New York	About 74	See Heiman (10, 11)
Philadelphia	At least 1	Data not available; 1 by memory
Phoenix	2	1 by firearm; 1 by car crash
Portland	1	5 others who had been retired for a short period of time committed suicide with firearms
Rochester	1	There were others before 1960; 1 after 1960 was by gun
Salt Lake City	0	1 officer killed himself by gun in 1949; he had cancer
San Diego	2	Both by handgun
St. Louis	Unknown	Suicide statistics not available
Seattle	5	All by gun
Topeka	0	
Washington, D.C.	Unknown	No statistical material kept

*Eleven other major cities did not respond to the research inquiry.

physician to become a member of a law enforcement agency and work simultaneously at primary prevention and research (e.g., Bruce Danto [12]).

In spite of the relative paucity of information and the guarded postures of several police departments, some observations can be made. The majority of American police officers used a handgun as the means of suicide. However, it may not be because handguns are used that the suicide rates are high but, rather, that the choice of a handgun reflects patterns of socialization. The use of a handgun is part of the patrolman's work. Marks and Stokes made a similar observation in their analysis of suicide in the south (13). They said their finding that southerners who committed suicide used firearms more often than nonsoutherners and were consequently more successful in killing themselves does not suggest that southerners have a higher lethal intent but that the use of firearms is a function of the

normative patterns of socialization in different regions and sections of the United States.

Although it is tempting to posit a psychoanalytic interpretation of the excessive use of handguns by suicidal police officers, I know of no published study of a large number of suicidal policemen in psychoanalysis that would lend credence to such speculation. Thus the availability of handguns and the inherent socialization process for their use as part of the policeman's work may be the greatest factors in determining the use of a handgun in police suicides in the United States as contrasted to London, where police officers do not carry guns as a rule.

Many psychodynamic formulations have been presented in the literature to account for suicidal behaviors (14–16). Several of these psychodynamic patterns were elaborated by Hendin in his study of the death fantasies and dreams of suicidal patients in analysis (17). One must realize, however, that the psychodynamic patterns of people who attempt suicide might not adequately account for the patterns of those who commit suicide; that is, those who attempt suicide may represent a different population from those who commit suicide (18). Nevertheless, in considering the history of patrolman 42, one can easily ascribe to him Hendin's idea of suicide as retroflexed murder. The intrapsychic processes of this patrolman may have been similar to Stekel's description at the 1910 meeting of the Vienna Psychoanalytic Society:

> *No one kills himself who has never wanted to kill another, or at least wished the death of another. . . .* The secret court of the unconscious follows the principle of talion—"an eye for an eye, a tooth for a tooth"; it declares itself guilty of the death wish and condemns itself to death. (19, pp. 87–89)

For patrolman 43 one can suggest another of Hendin's suicidal mechanisms, i.e., a possible narcissistic injury with death as a process that in an affective sense has already taken place. Although several psychodynamic mechanisms have been suggested to retrospectively and tentatively explain the suicidal deaths of patrolmen 42 and 43, other factors may influence the high suicide rate in some police departments.

Many people consider the role of the police in American society as subsuming criminal investigation and identification and as the vanguard in the war against crime. However, this image greatly underestimates the importance of such nonenforcement roles as social worker, crisis intervener, paramedic, community liaison officer with minority groups, and crime preventer (20). Farber (21) posited that the inability of a patrolman to fulfill these nonenforcement roles, with their caretaker aspects, constant exposure to human misery, and unrelenting demands for "interpersonal giving" and outlays of concern and nurturance, increases the probability of suicide.

One could also consider the modern American policeman as an alienated worker who has little control over the means of production and distribution of law enforcement. That is, legislatures, judges, and lawyers interpret the statutes defining criminal deviance, while the competing pressure groups and lobbying agencies buffet and manipulate the patrolman's use of discretion; these forces are only partially reconciled by political interventions. This situation, coupled with the nonenforcement role demands already described, enables one to readily appreciate that a policeman may, like Goethe's Werther, find no exit from a labyrinth of contradictory forces and choose to die.

Although there appear to be regional variations in suicide rates among police, the individual policeman's life, not a suicide rate, is at stake. Police departments could provide some sort of counseling like that of the Boston Stress Program (22). The policeman's role could also be made less alienated by having community leaders attend to the four basic psychological needs of patrolmen outlined by Denyer and associates (23)—physical security, belonging, efficacy of work, and self-transcendence. Perhaps if these suggestions were implemented, fewer policemen's lives would be lost through suicide.

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Inhalant Abuse in an Indian Boarding School

BY MARY F. SCHOTTSTAEDT, M.D., AND JOHN W. BJORK, M.S.W.

The authors describe inhalant abuse among children in an American Indian elementary boarding school. They discuss the initial interventions made to control this problem, such as providing a lower adult-child ratio and improving staff morale and child management skills and developing a volunteer program to improve the quality of the children's experience after school hours. They suggest that such preventive efforts be made early to interrupt the destructive cycle of dependence on external agents.

CASE REPORTS of deliberate inhalation of gasoline vapors by children and adolescents appeared during the 1950s and early 1960s (1–4). In 1962 and 1963 Glaser and Massengale (5) and Massengale and associates (6) drew attention to a rapid escalation of glue-sniffing among adolescents. The majority of their patients (in Denver, Colo.) were boys from large, low-income, disorganized Spanish-American families, who had poor peer relationships, low school achievement, and delinquent behavior. These authors commented on the similarity of these children to adult alcoholic subjects and postulated that many of the boys would shift from glue-sniffing to alcohol use as they grew older. Brozovsky and Winkler (7) and Rude (8) reported alcohol use in the groups of glue-sniffers they studied.

The serious physiological effects of glue-sniffing as well as deaths due to asphyxiation have been documented (9–11). An additional danger for glue-sniffing children is the establishment of habit patterns and ways of coping that foster dependence on external agents. For American Indian children the external agent will most likely be alcohol, a well-documented major problem for these people (12). Particularly vulnerable are Indian groups suffering recent and severe disruptions of their culture and lifestyle, such as the Plains tribes (13). If glue-sniffing is a precursor of alcoholism, identification and treatment of glue-sniffing children becomes an important strategy for preventing alcoholism.

The Bureau of Indian Affairs (BIA) runs 77 boarding schools for American Indians and an additional 18 dormitories from which Indian students attend public schools (14). These schools were originally designed "to separate a child from his reservation and his family, strip him of his tribal lore and mores, and prepare him for never again returning to his people" (15). Children were deliberately sent to boarding schools located far away from their homes, given English names, and forbidden to speak their own languages. The Merriam Report in 1928 urged a fundamental change in these destructive policies (15). However, in 1965 and 1966 Krush and Bjork (16) and Krush and asso-

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ciates (17) documented the continued deleterious impact of the boarding school experience on Indian children.

One effort to improve the well-being of Indian boarding school children was instituted in 1967—the model dormitory project on the Navaho reservation (18). Decreasing the adult-child ratio, training counselors in child care techniques, and developing pride in the Indian heritage resulted in positive effects on the intellectual, physical, and emotional development of model dormitory children (19, 20). However, this project has been terminated.

THE INDIAN BOARDING SCHOOL

The school reported on in this paper is one of two BIA elementary boarding schools in the state of Oklahoma. It was founded in 1871 to serve children of the Western Plains tribes, which were defeated and confined to reservations about 100 years ago after more than a century of independent, nomadic, warlike existence in a buffalo-hunting economy. The Dawes Act of 1887 reduced the amount of land assigned to Oklahoma Indians by awarding each Indian 160 acres and allotting the remainder for white settlement (21). Oklahoma Indians continue to live throughout the state, not on reservations. The Indians are an impoverished group largely dependent on unskilled employment or forms of relief. In the 1972–1973 school year, 90% of the children enrolled in our school came from western and central Oklahoma.

Data on our students were gathered by direct observation, participation in staff meetings, and interviews with staff members over a 2-year period (1972–1974). One of us (M.F.S.) served as psychiatric consultant to the Indian Health Service Unit and was asked by the school superintendent to consult at the school one day a month. We regarded this as an opportunity to initiate primary and secondary prevention (as defined by Caplan [22]) in a circumscribed, high-risk, young population group. Although glue-sniffers were the focus of our attention because of the hypothesized relationship between glue-sniffing and alcoholism, our overall goal was to alter the boarding school experience favorably for all of the children.

The school's capacity was 256 students in grades 1–8. Total enrollment for a school year ran around 350. Although the sole criterion for admission was that a student be at least one-quarter Indian, most children came to the school because of serious family disruption. Eighty of the 291 children enrolled by November 1973 (27%) had last names different from their next of kin's. Academic deficiencies were demonstrable in many students. More than 33% of the children in grades 4–8 were reading 2 or more years below their grade level.

The school was located on a prairie in western Oklahoma 40 miles west of the state capital. The physical plant, completed in 1968, had school rooms using open space with learning centers that were well-equipped with modern learning devices and resources. Indian heritage was emphasized in the classrooms.

The dormitories were one-story buildings separate for boys and girls. According to their age, 4 children were assigned to one sparsely furnished room from which the doors had been removed. None of the children had any personal possessions. Even clothing was kept in a central laundry area and doled out as needed. In contrast to the classrooms, nothing about the dormitories indicated the Indian heritage. Although the large central living and recreation rooms were always accessible, hobby rooms and kitchenettes were kept locked because of the limited number of staff. Food was served cafeteria-style in a separate building with no chance for selection. Children ate at long tables separated from the staff. Evening snacks were rarely available except from coin-operated candy and soft drink machines.

The dormitory staff included 4 college-educated supervisors and counselors (3 of whom were Indian) and 18 Indian aides charged with the immediate care of the children and their living area. Two aides were on duty at a time, resulting in an adult-child ratio of 1:64. To keep order, supervise housekeeping tasks, and meet the many physical needs of 64 children was an overwhelming assignment, leaving little time and energy to meet the emotional needs of the youngsters. Aides could be observed to retreat to their glass-walled offices as children poured into the dormitories after school. The aides posted written directions such as "Go take showers" on the glass wall. They were constantly responding to crisis situations, and they were frustrated because physical punishment, which they had experienced as boarding school students, was strictly forbidden. They could give "hours" for misbehavior, but then they had to see that children "worked off" the hours. There was no system of rewards.

Children not only brought to school overwhelming personal problems and needs but found themselves in a setting in which problems of group living were rampant. A pecking order was quickly established; status was maintained by brute force. A negative value system was promulgated: those who did well academically tended to drop out due to harassment. Scapegoating was common, and peer pressure to participate in mischief, including glue-sniffing, was ever present.

INHALANT ABUSE AND INTERVENTIONS

It is difficult to accurately document such behavior as glue-sniffing even in a boarding school population. In November 1973 2 counselors identified 37 habitual sniffers among 291 students enrolled that fall (12.7%). According to a personal communication from T. Voskuhl at the National Institute on Drug Abuse, it is reasonable to assume that the actual number was higher. The children identified as glue-sniffers included 11 girls and 26 boys in grades 6–8 (4 in grade 6, 13 in grade 7,

and 20 in grade 8). Sixteen of these children had last names different from their next of kin's (43%, compared with 27% of the total school population). An additional 4 of these children used alcohol habitually, and 15 were chronic smokers.

Other kinds of escapist behavior erupted in the fall of 1973. Nineteen girls engaged in knocking themselves out by twisting towels around their necks. Children obtained spray paints, glue, and solvents for sniffing from stores in nearby towns and the maintenance shop on campus. They stole gas from unlocked gas tanks of cars or tractors in adjacent fields. They tried shoe polish, ink, lighter fluid, and nail polish remover. The gas jets in the science classroom had to be disconnected because children used them to fill plastic sacks for sniffing. Supplies were hidden ingeniously in dormitory rooms—in vents, light fixtures, beds, and behind molding or paneling. Sniffing was usually a group activity done in nearby woods or after lights out in dormitory rooms.

School staff had already taken steps to limit the accessibility of inhalants through locking up supplies and car gas tanks. Plastic sacks were cut up before being discarded. A local Indian youth organization conducted campaigns to get stores in nearby towns to stock paint and glue supplies out of reach because sniffing was also a problem among public school children.

Our additional methods of intervention were based on the premise that glue-sniffing is a symptom of emotional distress resulting from multiple deprivations. We sought ways to meet basic emotional needs more adequately and to provide nondestructive mechanisms for release of tensions. Decreasing the adult-child ratio and improving staff morale and child management skills were initial steps. The staff schedule was changed to maximize coverage in evening hours. One of us (M.F.S.) met regularly with girls' dormitory aides to explore specific problems. Permission was granted for a social worker from the state's drug abuse program to spend one afternoon a week at the school seeing individual children in counseling sessions. The superintendent and counselors supported the development of a volunteer program in which 10 students and faculty from the Oklahoma Health Sciences Center participated. All of the volunteers attended weekly supervisory sessions; the medical students in the group were given elective credit for these sessions. The volunteers spent one evening a week in dormitories getting to know children individually and participating with them in recreational activities and at the evening meal. They introduced new games, crafts, and reading materials. Despite the prediction that books would be destroyed if left in the dormitory, 37 of the 40 books placed in the boys' dormitory were accounted for at the end of the year. The volunteers furnished new kinds of interpersonal experiences and role models for both students and aides. Ultimately, the school staff obtained permission to hire two Indian recreation directors. Their presence allowed evening use of the gymnasium and school library as well as further expansion of sports, arts, and crafts programs.

The counselors commented that there was less sniffing after the volunteer program was initiated than there had been in previous school years. By March 1974 only 5 additional children had been identified as sniffers. Counselors initiated the effort to hire the two recreation directors. Thus, indirect evidence supported the value of the program. Despite tribal antipathy toward research, school staff and tribal leaders cooperated in developing a research and demonstration grant proposal. Funds from this source are now supporting a research project to study the problem of sniffing and the effects of additional interventions.

DISCUSSION

Background factors known to be associated with inhalant abuse were readily identifiable in these Indian boarding school children: minority status, cultural upheaval, low socioeconomic level, family disruption, significant losses, neglect, delinquency, and poor school achievement. In addition, the boarding school experience itself compounded their problems: they were separated from their families and their home communities and placed in an institutional setting. Serious interpersonal deprivation occurred due to the limited number of dormitory aides and the impossible demands made on them. Organized recreational activities were inadequate. These children were denied the usual accoutrements of a home, toys, personal possessions, and reading materials. In such a setting, inhalant abuse offered solace, escape, entertainment, camaraderie, prestige, and gratification. Peer pressure enabled it to spread like a contagious disease.

All of these children needed to relate to warm, accessible, caring adults. They needed a chance to develop individuality, self-esteem, and pride in their Indian heritage. With one day a month of psychiatric consultation time available, the challenge was to set in motion some mechanism to change the system. Although a few identified sniffers were singled out for separate management, their environment was our prime focus of attention. Two main means of intervention were used: improving the morale and child management skills of existing staff and finding additional sources of adult contact through whom to introduce new attitudes and ideas. The latter included the development of a volunteer program, which had additional potential for interesting health care professionals in the needs of American Indians; and identifying additional sources of funding to improve the quality of the children's experiences in after-school hours. The active cooperation of school and health service staff was forthcoming in this quest.

The boarding school experience of American Indian children can be modified to provide healthier influences on the emotional growth and development of these students. Dependence on external agents is a

major problem for American Indians. Preventive efforts must start early to interrupt the destructive cycle that perpetuates family disruption and consequent boarding school rearing for yet another generation.

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This section contains reports of new clinical or laboratory observations, single cases of unusual interest or importance, descriptions of pilot studies, suggestions for future research, etc. Reports should be submitted in duplicate, typed double-spaced throughout, and may contain 1,000 words, no more than 1 table, and a maximum of 10 pertinent references. Figures cannot be used. Reports will be reviewed by at least one referee and the Editor; the author will receive a postcard informing him/her of the disposition of the submission. Reports will be edited for clarity and conformance with Journal *style.*

Timing and Risk of Bone Marrow Depression by Psychotropic Drugs

BY LAURENCE DUCOMB, M.D., AND ROSS J. BALDESSARINI, M.D.

We recently encountered a case that illustrates the clinically important point, accepted in good medical practice but rarely documented, that the period of risk for the suppression of bone marrow function by a medication begins anew each time another agent is introduced in the treatment. In addition, we will present further data to support the impression that severe depression of bone marrow function ascribable to psychotropic agents, while potentially life-threatening, is unusual.

Case Report

A 21-year-old male bank teller with no previous psychiatric illness developed an acute psychotic illness characterized by agitation, grandiosity, paranoid delusions, and auditory hallucinations. His mother had had recurrent depressions. He was admitted to our general hospital psychiatric service and treated for a month with haloperidol, 30–50 mg/day. Neurological and general medical examinations, including leukocyte counts, were normal during that period. In the second month of treatment with haloperidol he developed increasingly bothersome motor restlessness (akathisia), which disappeared after discontinuation of the butyrophenone. However, a week later his mental status deteriorated markedly. Thioridazine (600 mg/day) was used for 2 days but was ineffective. The medication was changed to chlorpromazine in increasing doses up to 2000-3000 mg/day, with a good response over the next 6 weeks. At that point, the patient complained of malaise and sore throat. Since the total time of exposure to neuroleptic agents had been almost 3 months and since this young man was not considered to be at high epidemiologic risk for agranulocytosis, it was somewhat surprising that a leukocyte count was only 1,600 cells/mm^3. Chlorpromazine was stopped and the patient was immediately transferred to a medical intensive care unit. The white blood cell count (WBC) continued to fall for several days to 400 cells/mm^3, with no granulocytes in the peripheral blood and a hypoplastic iliac bone marrow containing some myeloblasts. There were only small decreases in circulating erythrocytes and platelets and their marrow precursors. Fever and staphylococcal skin infections were treated with antibiotics. For 10 days his clinical condition remained precarious. Then, over 72 hours, immature granulocytes appeared in the blood and the hematologic condition returned toward normal. His psychiatric status remained stable enough to permit continued withholding of antipsychotic medication.

Discussion

This case illustrates the point that one must be alert to the possibility of toxic suppression of the bone marrow for at least several weeks following the initiation of treatment or *after a change to a new agent* and whenever the medical picture presented by this case is encountered. Antipsychotic agents, especially low-potency phenothiazines (notably chlorpromazine), are

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among the psychotropic agents most commonly associated with drug-induced agranulocytosis (1–3). The reported period of maximum risk is at about 3–4 weeks but continues to at least 8–12 weeks (1–3). Since this reversible condition evolves rapidly over a few days and is potentially fatal secondary to sepsis, a high index of suspicion, immediate discontinuation of drug treatment with appropriate medical treatment can be life-saving. Occasional routine WBCs are rarely helpful in predicting agranulocytosis (1, 3).

The current incidence of frank agranulocytosis or serious leukopenia during treatment with psychotropic agents is not clear. Previous estimates for the phenothiazines have been as low as 1/250,000 with short-term treatment (1, 3), but rates of about 1/1,000 to 1/2,000 have been typical in many surveys of the 1960s (1–3), and the figure may be higher in older patients (especially women) exposed to high doses of low-potency phenothiazines for prolonged periods. Similar reactions to other antipsychotic agents and to the chemically related tricyclic antidepressants have been reported very infrequently, possibly because of the use of smaller doses than of the low-potency phenothiazines and the typically short-term exposure to antidepressants. Leukopenia has been only rarely associated with other psychotropic agents such as the sedative-tranquilizers (1). Recent studies have suggested that the incidence of agranulocytosis associated with antipsychotic drugs may be lower than was reported previously. For example, only 1 case associated with chlorpromazine plus imipramine (and 3 other cases of milder leukopenia) was found in a 1,552-bed psychiatric hospital under careful surveillance in 1968 (4). More recently, only 1 or 2 cases possibly associated with phenothiazines were found in 1972 in a total population of 44,430 medical and psychiatric outpatients (5), although the number of patients actually at risk (i.e., exposed to phenothiazines) was not stated.

In the course of the present study we obtained, by a computer-assisted search, information on all instances of leukopenia associated with any medication in the period 1969–1976 (360 cases) among all patients admitted to the Massachusetts General Hospital. In most cases the probable cause of the bone marrow suppression remained obscure. Of the 91 cases we found that bore a highly probable association with specific medications other than cytotoxins (chemotherapy in cancer and organ transplantation), more than half were associated with antibiotics and almost 40% with various other drugs. Including the present case, we found only 5 patients with significant leukopenia (WBC below 4,000) associated with antipsychotic or antidepressant drugs, and only the present one had "frank agranulocytosis" (WBC well below 3,000) as defined by Pisciotta (2). One other patient had counts as low as 2,900 with very small doses of chlorpromazine (25 mg h.s.) plus alphamethyldopa. Of the 3 other cases, 1 involved a WBC close to 3,000 following small doses of chlorpromazine, a second had very mild transient pancytopenia associated with imipramine, and a third involved WBCs between 4,000 and 5,000 in an 81-year-old woman given amitriptyline. Thus, at most, there were only 2 cases that might be considered agranulocytosis (including the present one), and both were possibly associated with chlorpromazine. The population at risk is unknown since we could not determine how many patients were exposed to antipsychotic or antidepressant agents, but the total number of patients (out of 233,610 admissions) from whom this group was gathered was estimated to be 205,554, suggesting that the problem either occurs infrequently or is infrequently noted in the record. Arguing against the latter hypothesis was our observation of extremely assiduous hematologic evaluation of relatively mild leukopenias, which might be expected in a teaching hospital.

Although these results and recent observations of outpatients (5) cannot provide true incidence figures because the number of patients at risk by exposure to the drugs in question is unknown, they suggest that the incidence of serious leukopenia associated with antipsychotic and antidepressant drugs may have been declining since the late 1960s. Such a change might reflect an increased use of high potency (low dose) neuroleptic agents in recent years. Although it is not clear that the incidence rises in a close relation to daily drug dosage, it seems to increase as a function of the dose-time interaction (1, 2). Moreover, the current trend toward increasingly high doses of antipsychotic medications and prolonged use of antidepressants may increase tissue pools of slowly cleared potentially toxic drugs and might, if continued, eventually lead to higher incidence rates of agranulocytosis.

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Proven Asymptomatic Eosinophilia with Imipramine

BY NEAL R. CUTLER, M.D., AND DOUGLAS J. ANDERSON, M.D.

The *Physicians' Desk Reference* lists eosinophilia as a potential adverse reaction to imipramine, but a survey of the literature revealed only three reports of this response. Penick and Carrier (1) described a case of asymptomatic eosinophilia accompanied by leukocytosis during imipramine therapy. The total white cell count reached 23,200 with 56% eosinophils; 8 days after withdrawal of imipramine the count had decreased to 6,400 with 12% eosinophils. Joynt and Clancy (2) reported on a patient who showed a maximum total white cell count of 30,000 with 61% eosinophils during treatment with imipramine. When the patient developed a stiff neck, severe generalized headache, and nausea, imipramine was discontinued. Results of a lumbar puncture were normal and the symptoms abated within 2 days. By the 25th day after imipramine was discontinued, the total white cell count had decreased to 11,500 with 3% eosinophils. Hippius (3) followed the relative eosinophilia in 25 patients treated with imipramine. During 9 weeks of administration of the drug the relative percentage of eosinophils peaked at an average of 4% at 6 weeks and returned to the pretreatment average level of 2% by the end of the 9 weeks. Data for individual cases were not provided, and generalized leukocytosis was not mentioned.

All of these reports were either based on coincidental rise and fall of the eosinophil count during imipramine therapy or coincidental decline of a marked eosinophilia on withdrawal of the drug. The case we will describe, in which we followed the eosinophil count through two consecutive periods of imipramine administration separated by a drug-free interval, is to our knowledge the first to confirm in this way the correlation between imipramine and asymptomatic eosinophilia.

Case Report

A 57-year-old man was admitted to the hospital following a suicide attempt. A diagnosis of psychotic depression was made based on a 6-week history of depressed affect, feelings of worthlessness and hopelessness, and vegetative signs and symptoms, including insomnia, anorexia (6.8-kg weight loss), constipation, psychomotor retardation, and decreased libido. Physical examination on admission was within normal limits. On the 2nd hospital day the patient was started on 75 mg of imipramine, increased over the next 6 days to 300 mg/day. On the 12th hospital day the patient developed a nonproductive cough with chest pain. His CBC revealed a total eosinophil count of 10,750, or 45% of the total white count of 23,900.[1] A diagnosis of viral pneumonia was made, and the eosinophilia was thought to be Löffler's syndrome (pulmonary infiltration with eosinophilia), eosinophilic leukemia, or secondary to imipramine therapy.

The pulmonary symptoms and physical findings resolved in a few days, but the eosinophilia persisted. An extensive medical workup was undertaken, including bone marrow aspiration and biopsy, bronchoscopy with bronchial biopsy, and whole body gallium scan. These studies ruled out any suspected neoplastic disease. Other studies were done to rule out parasitic infestation and collagen vascular disease. The bone marrow aspiration showed 51% eosinophils. Pulmonary symptoms cleared in a few days and a chest X ray was normal, but because of persistent depressive symptoms imipramine was continued after resolution of the pneumonia to the 27th hospital day. At that time the total white cell count was 17,500 with eosinophilia having reached a maximum of 66% (11,550 total eosinophil count).

Two days following discontinuation of imipramine, leukocytes and eosinophils had fallen to 8,800 and 32%, respectively (2,830 total eosinophil count). Nine days after discontinuation the total white cell count was 6,100 with 10% eosinophils. The patient was discharged and healthy on the 47th day on no medication. Seven days later the white count was 7,300 with a normal eosinophil count (140 total). In order to confirm our impression of drug-induced eosinophilia, the patient gave informed consent to restart 100 mg of imipramine daily as an outpatient. One month later on a dose of 250 mg/day the white cell count was 7,000 and the eosinophil count had again risen to a high of 11%.

Discussion

The incidence of asymptomatic eosinophilia with imipramine is unknown. Our case adds to the reported occurrence of a marked or relative eosinophilia with this drug. Moreover, this is the first case we know of to confirm a causal relationship by a second course of administration. It is of interest that there was no total leukocytosis in the second course compared with the first, and the eosinophilia was only 15% as severe. This diminished eosinophilia response with a repeated administration of the drug is unlike that seen with certain other drugs. For example, gold therapy in rheuma-

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[1]The normal range of total eosinophil count at our hospital is 0–450.

toid arthritis has been shown to produce a type 1 hypersensitivity reaction. The eosinophilia response in these patients remains about the same with a repeated administration of gold (4). Our case suggests a gradual desensitization to imipramine-induced eosinophilia with repeated administration. Although the precise mechanism behind asymptomatic eosinophilia with imipramine is unclear, our case leaves no doubt that it does occur.

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A Case of Oral Dyskinesia Associated with Imipramine Treatment

BY JEFFREY J. DEKRET, M.D., IRADJ MAANY, M.D., T. ALAN RAMSEY, M.D., AND JOSEPH MENDELS, M.D.

Reports of oral dyskinesias associated with the administration of antidepressants are scarce. There have been reports of choreoathetoid movements as a toxic manifestation of acute tricyclic antidepressant poisoning (1) and of two patients who developed dyskinesia while receiving tricyclics concurrently with neuroleptics (2). The following case strongly suggests a causal relationship between imipramine treatment and an oral dyskinesia.

Case Report

The patient, a 52-year-old man, was hospitalized for complaints of depression, nervousness, diminished appetite with a 7-kg weight loss, and guilty ruminations that had persisted for over a year. There was no evidence of manic disorder, alcohol or drug abuse, formal thought disorder, or organic brain syndrome. Six months before his admission, the patient had been hospitalized elsewhere for 10 weeks and had been given haloperidol, 0.5 mg t.i.d., which he was still taking at the time of his admission. He had not taken any other psychotropic medication for years. The patient also had a history of hypertension that had been treated with methyldopa and hydrochlorothiazide for the preceding 3 years.

Physical examination, including a neurological workup and laboratory studies, was unremarkable on admission. The patient received no active medications for the first 3 weeks in the hospital and he remained significantly depressed (17-item Hamilton Rating Scale scores were in the mid-20s). Imipramine HCl, 100 mg/day, was started and increased by 50 mg every other day until a dosage of 250 mg/day was reached. Three days after the 250-mg dosage was started, the patient developed abrupt involuntary buccal contractions about every 5 seconds while speaking. These contractions apparently involved the soft palate, larynx, and diaphragm and disturbed his speech. His symptoms were worse when he was anxious but slight and infrequent when he was not speaking. A fine tremor of the tongue and involuntary tongue rolling and lip licking were present at rest. There was also a fine resting tremor of the upper extremities. During sleep he had myoclonic jerks of his extremities and grimacing, chewing motions that resulted in mouth ulcerations.

A neurology consultant noted no other findings and felt the condition was related to the medication. The patient was videotaped twice weekly while the condition was severe and weekly when it improved. Physostigmine, 1 mg I.V., was given intravenously over a 5-minute period during one videotaping, and the abrupt oral contractions almost disappeared for an hour. Other signs mentioned above were difficult to evaluate because of rapidly emerging signs and symptoms of cholinergic excess, which required atropine administration.

The patient had an excellent response to imipramine, so it was decided to reduce the imipramine dosage rather than change medications. Two weeks later, on a dose of 150 mg/day, the speech disturbance had disappeared, but the fine tremor of the tongue and upper extremities, tongue rolling, and lip licking persisted. One week later, on 100 mg/day, only the fine movements of the tongue remained. The next week the imipramine dosage was increased to 250 mg/day over 3 days. His oral dyskinesia reappeared but was less severe. The imipramine was again reduced to 150 mg, and the dyskinesia disappeared within a week. The patient was discharged on this dosage, and there was no recurrence of the dyskinesia or depression after 6 months.

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Discussion

The oral dyskinesia described herein appears clinically very similar to tardive dyskinesia, and we hypothesize a similar pathogenesis. Current theories regarding the pathogenesis of tardive dyskinesia rest on the emergence of denervation supersensitivity of dopamine receptors following the prolonged blockade of these receptors by neuroleptics (3). This leads to a relative hyperdopaminergic and hypocholinergic imbalance in the basal ganglia (4).

The same imbalance may have developed in this patient. Supersensitivity of dopamine receptors may have developed from the previous haloperidol treatment or even from the mild postsynaptic dopaminergic blocking properties of imipramine (5). Finally, the strong anticholinergic properties of imipramine could increase the relative hyperdopaminergic-hypocholinergic imbalance sufficiently to produce the described oral dyskinesia. Imipramine seems to be the primary causal agent because the onset and severity of the dyskinesia correlated with the onset of treatment and the dosage of imipramine.

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L-Tryptophan in Obsessive-Compulsive Disorders

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Obsessive-compulsive disorders are usually characterized by dwelling on thoughts that one cannot reject, inability to make decisions, rituals (e.g., hand washing), double-checking, and repetitive questioning. Secondary symptoms are anxiety, depression, and hostility. The etiology of the obsessive-compulsive disorders is believed to be psychological (1), but psychobiological treatments have failed to yield satisfactory responses. We have had success in clinical trials using chlorimipramine, a potent serotonin (5-HT) reuptake blocker (2). Further studies supported our hypothesis that some obsessive-compulsive disorders are organic in nature and may be related to a central disturbance in 5-HT metabolism (3). In this paper, we will report on the clinical efficacy of the precursor amino acid L-tryptophan in obsessive-compulsive disorders.

Method and Results

Seven obsessive-compulsive patients (4 male and 3 female) ranging in age from 13 to 40 (mean=22.9 years), gave informed consent to participate in the study. Subjects were placed on L-tryptophan, 3 to 9 g (mean=6.28) daily in divided doses given orally. In addition, all patients received oral daily divided doses of nicotinic acid, 1000 mg b.i.d., and pyridoxine HCl, 200 mg b.i.d. The patients showed considerable improvement after 1 month of therapy and after 6 months to 1 year of therapy their conditions were stabilized. In 2 cases in which there was a history of aggressive behavior, such behavior increased after L-tryptophan administration (doses of 3 g and 7 g, respectively). One patient who was given 5 g/day of L-tryptophan experienced headaches.

Discussion

The data presented here demonstrate the therapeutic effectiveness of L-tryptophan in controlling obsessive-compulsive symptoms. The rationale for the use of L-tryptophan was based on our successful experience with chlorimipramine in obsessive-compulsive disorders. Chlorimipramine is a potent 5-HT reuptake blocker and its effect therefore is presumed to be mediated by a serotonergic mechanism. There are other means by which an increase in the steady state levels of serotonin in specific areas of the brain may be accomplished. In this study, we took the approach of using the naturally occurring amino acid L-tryptophan, a precursor of 5-HT. Serotonin synthesis in the brain is regulated by the availability of this amino acid (4, 5), so an increased synthesis may be expected after administration of an excess of the precursor. To ensure that significant amounts of tryptophan would be available for 5-HT synthesis, we also used nicotinic acid supplements. It has been shown that excess nicotinic acid can inhibit the major metabolic pathway of tryptophan by way of tryptophan pyrrolase and thus make more tryptophan available for 5-HT synthesis (6). Be-

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cause pyridoxine, as the coenzyme pyridoxal-5'-phosphate, is involved in numerous pathways related to amino acids, neurotransmitters, etc. (7), it was considered helpful to provide adequate quantities of this vitamin in the treatment regimen.

The observed increase in the aggressive behavior of two patients is similar to our previous unpublished findings with aggressive patients who were given tryptophan. We can only speculate, in the absence of pertinent biochemical data, that it is the tryptophan per se or the balance between tryptophan and 5-HT that may regulate aggressive behavior. An imbalance may precipitate or exacerbate the aggressive behavior. Whatever the mechanism, our experience indicates that tryptophan treatment in such patients is contraindicated.

The data obtained in this preliminary study are very encouraging and suggest the potential use of this type of approach in other disorders.

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Pediatric Bone Marrow Transplantation: Psychosocial Aspects

BY BETTY PFEFFERBAUM, M.D., M. MURIEL LINDAMOOD, M.S.W., AND FRANCES M. WILEY, R.N.

Many authors have investigated the multifaceted impact of serious childhood illness (1–6). One recent article addressed the emotional stress of bone marrow transplantation in adults and adolescents (7). In our experience with children undergoing the bone marrow transplantation procedure, we have been equally impressed with the importance of psychosocial factors in this highly vulnerable age group.

The procedure involves supralethal doses of radiation and chemotherapy. The destroyed bone marrow is then replaced by an infusion of marrow from a compatible donor. The process and the expected complications of graft-host disease, infection during marrow suppression, bleeding, and toxicity from radiation and chemotherapy necessitate a lengthy and complicated hospitalization (mean=8.8 weeks). The child spends most of his/her time in reverse isolation, frequently in a critical condition (8, 9).

Nineteen children between the ages of 7 and 16 received bone marrow transplants at the University of California, Los Angeles, from 1973 through 1976 for aplastic anemia and leukemia. Because of the high mortality rates for these diseases (approaching 100% without this procedure), bone marrow transplantation was often considered the last hope in an otherwise hopeless situation. Five children died during the procedure.

From its inception the transplant team emphasized attention to the psychosocial aspects of care. As we gained more experience in working with those involved with the procedure, it became clear that an increasingly vigorous participation of mental health personnel could be of significant benefit in dealing with the intense emotional stress involved. Consequently, a child psychiatry consultant joined the social worker and nurses as a regular participant in the weekly transplant rounds held by the oncologists. In addition, case-centered and issue-centered psychiatric conferences were held in which the entire staff had the opportunity to discuss their feelings of frustration with problem areas, intrastaff conflicts, and the ever difficult emotional aspects of working with terminally ill children and their families. In addition, support services such as play therapy and a schoolteacher were readily available. Physical therapy, dietary, and professional services were all geared to the needs of children. Weekly thanatology conferences gave the transplant team an opportunity to view pediatric death in a larger framework.

An objective assessment of all those involved in the

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The authors wish to acknowledge the support and cooperation of Dr. Stephen Feig, Associate Professor and Chief, Division of Pediatric Hematology-Oncology, and Dr. Peter Falk, Assistant Professor of Pediatrics, University of California, Los Angeles, Medical Center. They also wish to thank the pediatric bone marrow transplant nursing team, headed by Debby Rose, R.N., for valuable insights.

care of the children was not possible. We decided to focus our investigation on the parent-child interaction, although we also collected many subjective impressions of all those concerned. We chose this focus because in the day-to-day life of the hospitalized child the familial interaction seemed to play so large a part in the child's well-being. We suspected that family interaction and involvement might be predictors of the child's survival during the procedure. In order to determine whether emotional factors had a direct bearing on survival, retrospective subjective ratings were done by the physicians, nursing staff, and social worker on various aspects of family interaction. Parents were rated as to the frequency of visiting, the degree to which they seemed to offer emotional support to the child, and their general enthusiasm for the procedure. In addition, the child's motivation for the procedure was rated. Data were analyzed by chi-square with Yates' correction.

None of the factors investigated was found to be a significant predictor of survival, either during or after hospitalization. We speculate that the overwhelming nature of the illness far outweighed any other single factor.

Although the psychosocial factors did not affect outcome in terms of survival, we believe that these factors were critical in the management of many cases. Therefore, even though direct correlations between survival and psychosocial factors could not be demonstrated, we strongly suspect that psychosocial factors may have had an indirect influence that was difficult to measure. Certainly, the team's awareness of such factors improved their management of these children and their families as is illustrated in the cases discribed below.

Case Reports

Case 1. Bobby came from a highly disturbed family with an alcoholic father and a histrionic mother. The parents' ambivalence toward the procedure, especially manifest in times of crisis, led to such drastic intrafamilial interactions that they were unable to support the patient.

Although he was only 10 years old, Bobby gave an initial impression of maturity and control. We considered this to be an adaptive response to a long struggle with leukemia. However, we later recognized this adaptation to be a shaky construct of fragile defenses.

Bobby suffered a very stormy course, during which it was difficult to distinguish physical from psychological components. Problematic signs and symptoms included anorexia, hematemesis, and blurred vision. Behavioral symptoms included rage reactions, hiding under his blankets, and major control struggles over activities, medications, and even staff assignments. He interfered with medical management by turning off his I.V. line and refusing venipuncture. Bobby displayed the fragility of his defensive structure when he related elaborate fantasies and dream material to the child psychiatrist.

After much persistent team effort, a sufficient modicum of cooperation was obtained from the parents and the patient to permit improved medical management. Bobby survived the transplantation. We feel, although we cannot prove it conclusively, that the team effort contributed to his survival.

Case 2. Susan's parents had a strong marriage, seemed to share responsibilities, and agreed fully about the decision for her transplantation. Susan's home was many miles from the medical center, so the parents decided that her mother would move to Los Angeles to be with Susan while the rest of the family remained at home. Family "reunions" were held every weekend in Susan's hospital room, and she received a call from home every night.

The mother's insight enabled her to convey to the staff both Susan's and her own needs. This became a positive working relationship in which the staff focused on helping the mother to care for her own child. Susan allowed the child psychiatrist only occasional glimpses of her fears of death, but she confronted her mother directly with these fears. Her mother was able to seek guidance and support from the staff and the child psychiatrist worked with her to help Susan deal with her fears and concerns. In this case, the team was able to channel the positive forces already present and thereby further ensure smooth medical management.

Conclusions

We have observed that attention to psychosocial factors favorably affects medical management of children undergoing bone marrow transplantation. We reasoned that the attention paid to these factors might well affect mortality rates during and/or after hospitalization, particularly after the addition of a child psychiatric consultant to the team. Our pilot study of 19 children focused on parent-child interactions, which seemed of paramount importance. No statistically significant correlation could be made in this small sample between psychosocial factors of parent-child interaction and survival rates for the procedure or the post-hospitalization course. However, since we were clinically impressed by the salutory effect of psychosocial intervention on medical management, we postulate that a larger, more detailed study using other measures might demonstrate a direct or indirect effect on mortality rates, at least with respect to survival of the procedure itself.

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The Effect of Benztropine Mesylate in the Rabbit Syndrome and Tardive Dyskinesia

BY ROBERT SOVNER, M.D., AND ALBERTO DIMASCIO, PH.D.

The "rabbit syndrome," a term coined by Villeneuve (1), is a late-onset neuroleptic-induced extrapyramidal syndrome characterized by rhythmic 5–5.5 per second involuntary movements of the oral and masticatory musculature that mimic the chewing movements of a rabbit (2). The rabbit syndrome is readily reversible when neuroleptic drug therapy is discontinued (1), and it responds to antiparkinsonian drug therapy (1, 2) These clinical characteristics and the experimental observation that the rabbit syndrome persists during stage I non-REM sleep while tardive dyskinesia does not (2, 3) suggest that the former is a distinct neuroleptic-induced extrapyramidal syndrome and not just a form of the latter. The following case report provides additional support for this hypothesis.

Case Report

Ms. A, a 54-year-old single woman, has had eight psychiatric hospitalizations in the past 10 years for a late-onset paranoid psychosis and has received approximately 8 months of intermittent drug therapy with several different neuroleptic agents. She has no history of neurological illness and has not received ECT.

Involuntary movements were first noted at the time of her most recent hospitalization for treatment of her recurrent psychosis. On the day of admission she was given fluphenazine decanoate, 25 mg I.M., and within several days her psychotic symptoms resolved and the involuntary movements decreased in severity. She was discharged on the fifth hospital day and was referred to the Tardive Dyskinesia Clinic. At each clinic visit, her involuntary movements were assessed by R.S. on the Abnormal Involuntary Movement Scale (AIMS), developed for use in the National Institute of Mental Health Collaborative Study of Fluphenazine Decanoate. The severity of rabbit syndrome movements, when present, was assessed with a global rating scale using the same anchor points employed in the AIMS. Results of ratings are presented in table 1.

At her first clinic visit a definite diagnosis of tardive dyskinesia was made by R.S. on the basis of undulating (myokimic) tongue movements, involuntary chewing movements, and axial hyperkinesia (all of mild severity). There was also a suggestion of perioral involvement. The severity of her dyskinetic symptoms was increased by arousal. The patient was not aware of the movements. She was requested to return in 2 weeks for reevaluation.

When she returned to the clinic, the patient reported that she had gone to an emergency room 3 days after her last visit when severe mouth movements suddenly developed. The doctor on call prescribed benztropine mesylate, 2 mg b.i.d., and these movements disappeared within 2 days. Examination in the Tardive Dyskinesia Clinic revealed that the tongue movements were of moderate severity. Perioral movements were definitely present and choreoathetoid movements of the extremities were observed for the first time. There was no sign of the new movements for which she had been treated. The patient was told to discontinue the benztropine mesylate.

She returned to the clinic 8 days later in acute distress. Mouth movements identical to those for which she was given the benztropine mesylate had returned, involving the masticatory and oral musculature. She rapidly opened and closed her mouth several times per second and had bitten her tongue frequently during the previous week. These new movements had obliterated the more typical perioral and lingual movements of tardive dyskinesia described previously. The choreoathetoid movements of her extremities had disappeared. Both authors concurred on a diagnosis of the rabbit syndrome. Benztropine mesylate, 2 mg I.V., did not affect these new movements but brought out the choreoathetoid movements of her extremities. Because of her previous response to oral benzetropine mesylate she was restarted on the drug at a dose of 2 mg b.i.d. A week later, rabbit syndrome symptoms were not present, but symptoms of tardive dyskinesia were again exacerbated.

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TABLE 1
Results of AIMS and Rabbit Syndrome Ratings

Time	Medication at Time of Exam*	Rabbit Syndrome Rating	Facial Expression	AIMS Ratings**					
				Lips and Perioral Area***	Jaw***	Tongue	Upper Extremities	Lower Extremities	Neck, Shoulder, Hip
Day of discharge	—	0	0	1	2	2	0	0	2
12 days postdischarge	Benztropine mesylate, 2 mg b.i.d. for 4 days	0	0	2	2	3	2	1	2
20 days postdischarge	—	3	0	—	—	2	0	0	2
15 minutes after I.V.	Benztropine mesylate, 2 mg I.V.	3	0	—	—	2	2	2	2
26 days postdischarge	Benztropine mesylate, 2 mg b.i.d.	0	0	3	3	3	2	2	3

*Patient received fluphenazine decanoate, 25 mg I.M., on the first day of her hospitalization.
**0=absent, 1=equivocal, 2=mild, 3=moderate, 4=severe.
***When the rabbit syndrome was present, it was impossible to accurately assess dyskinetic movements of the patient's mouth and jaw.

Discussion

The response of our patient to benztropine mesylate (suppression of the rabbit-like oral movements with a concomitant exacerbation of the dyskinetic symptoms) further demonstrates the clinical independence of these two neuroleptic side effects. To our knowledge, this is the first report describing the effects of an antiparkinsonian drug in a patient simultaneously afflicted with both these drug-induced extrapyramidal syndromes. Although the effects of benztropine mesylate on the rabbit syndrome have been described by Villeneuve (1) and systematically examined by Jus and associates (2), they did not administer this drug to a patient who had both syndromes at the same time. The fact that our patient's rabbit syndrome symptoms were not suppressed by intravenous benztropine mesylate may indicate the dose was too small.

The rabbit syndrome is generally considered to be a form of drug-induced parkinsonism (1, 4), but it is in many ways atypical for this side effect. To date, it has only been reported to develop in middle-aged or elderly individuals (many having evidence of brain damage) who have received neuroleptic drugs for several years (1). We theorize that even though drug-induced parkinsonism and the rabbit syndrome are both mediated by a neuroleptic blockade of the same pool of dopaminergic neurons within the extrapyramidal system, the rabbit syndrome results only when this neuronal pool undergoes structural alteration, possibly by a toxic effect of long-term neuroleptic drug therapy. This pool of neurons (or receptors) appears to be different from the one mediating tardive dyskinesia since both the rabbit syndrome and drug-induced parkinsonism can coexist with tardive dyskinesia yet respond differently to anticholinergic agents (5). Such a hypothesis has already been put forward by Klawans (6) and Cools and Van Rossum (7).

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Amitriptyline Therapy in Anorexia Nervosa

BY DANIEL C. MOORE, M.D.

Patients with anorexia nervosa report that they are hungry but use all sorts of techniques, including food refusal, laxatives, and self-induced vomiting, in their pursuit of thinness. The following is a report of the effect of amitriptyline, commonly known to induce weight gain in depressives, in a patient with anorexia nervosa.

Case Report

A thin, attractive 20-year-old college senior first presented with a chief complaint of multiple daily eating and vomiting episodes, which interfered so much with her studies that she chose to drop out of school in December. A thorough medical evaluation failed to reveal any cause for her unusual behavior. Since the patient had been on birth control pills, it was not possible to document amenorrhea.

She described a pattern of going from store to store eating doughnuts, ice cream, pizza, chocolate, and fruit in rapid succession until she felt thoroughly and uncomfortably stuffed, then finding a quiet secluded place to vomit.

Her relentless preoccupation with thinness—which continued unabated even after her weight had gone down to an extremely slender but not emaciated 47.7 kg—began in her sophomore year of college after she met her first real boyfriend, a graduate student. At that time, she was "chubby," having weighed 63.6 kg during and since high school. Her height was 160 cm. They began to date steadily and he exhorted her to lose weight, telling her she was sexually unattractive and "flabby." Soon her failure at dieting joined dissatisfaction with her grades and choice of major as significant frustrations.

It was at this point of extremely low self-esteem and sense of failure that she hit upon the stratagem of vomiting as a way of satisfying both her desire to eat and her pursuit of thinness.

History. The patient's father is a blue-collar worker; her mother has a college education. Her mother has always been overweight and dresses in unflattering clothes.

The pregnancy was marked by bleeding in the third month, and the obstetrician told the mother that the baby would be defective. The delivery was uneventful, but according to the patient, "my mother's milk was bad" and she lost weight from 3.1 kg to 1.36 kg at the end of 2 weeks. She said, "I threw up everything and was crying and crying for 2 weeks. Finally my grandfather recommended goat's milk and I put on weight rapidly and was a chubby baby and have been chubby ever since."

Her mother prepared her for menstruation, and she had an uneventful menarche at age 11 with regular cycles thereafter.

Course of treatment. A sleep EEG ordered to rule out seizure disorder as a cause of the bulimia was negative. Nevertheless, the patient was given a trial of diphenylhydantoin, 100 mg t.i.d., with blood levels of 20 μg/ml. The 2-month trial had no effect on her vomiting pattern. In her psychotherapy the focus was her pervasive sense of ineffectiveness and low self-esteem, reinforcing her awareness of her own wants and needs. A 3-month trial of imipramine, 150 mg/day, had no effect on these symptoms.

Amitriptyline was started 18 months after the patient entered treatment and 2½ years after she began the vomiting, first at 50 mg and later at 100 mg. After the first week, she reported that she felt different and had vomited only twice during the week. During the second week, when the dosage was increased to 150 mg, she felt no desire to vomit but was somewhat anxious because she seemed to be increasing her caloric intake and might be gaining weight.

During the next four months, she vomited a total of four times. Each episode seemed stress-related, was not accompanied by binge eating, and was experienced as ego-alien.

By the end of 4 months on amitriptyline, she had gained 5.0 kg and weighed 52.2 kg, a normal weight for her height. At this point, we tapered her medication over 2 weeks. The day after amitriptyline was stopped, she had a protracted episode of bulimia and vomiting, followed by a week of what sounded like an exaggerated tricyclic withdrawal—malaise, abdominal pains, and nausea. At the end of the second week, she became preoccupied with food, calories, eating, and vomiting to the exclusion of the other therapeutic issues on which she had worked so well for the past 4 months. The next week, she called in tears to say that she had been eating and vomiting continually and would like to resume the amitriptyline. The bulimia stopped within 2 days and her preoccupation with weight became a background issue once again. Psychotherapy continues on issues of personal effectiveness and awareness of wants and needs.

Discussion

This case satisfies the Feighner and associates (1) criteria for anorexia nervosa, although Bruch (2) would probably label it atypical anorexia nervosa because of the prominence of bulimia and vomiting and compulsive rituals about eating. The resemblance of these symptoms to those described by Rau and Green (3)

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as being responsive to anticonvulsants prompted the unsuccessful trial of diphenylhydantoin.

Tricyclic antidepressants were specifically chosen because of their success in some cases of obsessive-compulsive neurosis (4), which anorexic eating behavior sometimes resembles. Needleman and Waber (5) have also used amitriptyline recently in six cases of anorexia nervosa, but their original purpose was to treat the motor retardation and sadness observed. They reported weight gain and normalization of mood.

The lack of effectiveness of imipramine in this case compared with amitriptyline suggests an interesting line of speculation about the mechanism of action. Animal studies by Carlsson and associates (6) on the ability of various antidepressant drugs to prevent depletion of intraneuronal 5-hydroxytryptamine (5-HT) stores caused by 4-methyl-α-ethyl-metatyramine show that the order of potency in preventing 5-HT depletion is chlorimipramine followed by amitriptyline, imipramine, chlordesipramine, nortriptyline, and desipramine. Sheard and associates (7) have shown a similar order of potency in depressing the firing rate of the midbrain raphe, presumably because decreased reuptake causes increased 5-HT availability at the receptor and feedback inhibition of presynaptic neuronal activity. This suggests that amitriptyline is more potent than imipramine in blockading reuptake of 5-HT, and Maas (8) has suggested that the difference may be even larger in therapeutic situations, since amitriptyline is rather slowly broken down to its metabolite nortriptyline while imipramine is rapidly metabolized to desipramine.

Studies have suggested that plasma-free tryptophan is a determinant of brain tryptophan levels (9). Because brain tryptophan concentration is known to be an important factor influencing brain serotonin levels, Coppen and associates (9, 10) have used free plasma tryptophan levels to study patients with depression and patients with anorexia nervosa. Free plasma tryptophan is significantly low in both conditions but returns to control levels in recovered depressives, while remaining low in anorexics after they have returned to near normal weight on a psychiatric ward. The failure of low plasma tryptophan, unusual eating habits, and amenorrhea to return to normal suggests that induced weight gain alone does not attack the basic causes of anorexia nervosa and therefore leaves the patient at risk for frequent relapses.

The success with amitriptyline in this case and the six others reported by Needleman and Waber (5) suggests that drugs which prevent reuptake of 5-HT and decrease its turnover in the brain may be useful in anorexia nervosa. Since chlorimipramine is the most potent of these drugs, it probably deserves a trial in this condition.

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Nuclear Symptoms of Schizophrenia After Cingulotomy: A Case Report

BY JAVIER I. ESCOBAR, M.D., AND VIJAYA CHANDEL, M.D.

The importance of the limbic system in the genesis of schizophrenic symptoms has been highlighted in recent reviews (1, 2). Schizophrenia-like symptoms are reported to occur frequently in temporal lobe epilepsy and other instances of "injury" to limbic structures, e.g., some brain tumors (3).

We wish to describe an illustrative case in which "nuclear" symptoms of schizophrenia (4) first developed after a cingulotomy. The exacerbation and diminution of these symptoms during treatment with imipramine, methylphenidate, and neuroleptics are described. We theorize that schizophrenia-like symptoms may have resulted from the lesion to the cingulum and that

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fluctuations in symptoms during pharmacotherapy were mediated through changes in catecholamine turnover (primarily dopamine) in the mesolimbic system.

Case Report

The patient, a 44-year-old married woman, was referred to the Memphis Mental Health Institute in January 1977 because of "intractable depression" 7 months after a cingulotomy.

Symptoms of depression were first noticed in 1974 and worsened over the next year. In 1975 the patient took an overdose of diazepam. She was hospitalized five times the following year with a diagnosis of "involutional melancholia." She received tricyclic antidepressants at first, but because of "lack of response to drugs" she was given ECT on four different occasions. Response to each course of ECT was transient, and by mid-1976 the patient's situation had become desperate. Her depression was extremely severe and refractory to traditional treatments.

A bilateral cingulotomy was performed in June 1976 in a private hospital. Following an uneventful operation, her symptoms of depression were unchanged, and she developed additional symptoms with a nonaffective quality—nihilistic and somatic delusions, feelings of depersonalization and derealization, and paranoid ideas. According to family interviews and past hospital records, such symptoms had not been present before the surgery.

The patient's medical history included a hysterectomy in 1970 and removal of an ovarian cyst in 1972. Following the 1972 surgery, she developed "gastrointestinal complaints and nervousness" for which diazepam was prescribed. The history is otherwise unremarkable. Family history revealed the presence of depression in the patient's mother and her older sister.

On admission to the institute the patient exhibited affective blunting, anhedonia, nihilistic and somatic delusions, feelings of depersonalization and derealization, and Schneiderian symptoms of schizophrenia (delusions of influence, auditory hallucinations, and thought broadcasting). No evidence of organicity was found. Physical and laboratory findings were unremarkable other than an EEG that showed "mildly irregular and slow diffuse activity; findings indicative of mild underlying cerebral dysfunction." A sleep-deprived EEG did not show any changes over the baseline EEG. A neurological consultation revealed no gross abnormality, and neuropsychological test results gave no evidence of lateralizing dysfunction or deterioration of brain function compared with the precingulotomy test results. The brain scan was normal. After her admission to the hospital, there was a 7-day medication wash-out period for baseline observation, during which time the symptoms of psychosis were unchanged. Therapy with imipramine and methylphenidate was initiated, with maximum daily dosages of 300 mg of imipramine and 30 mg of methylphenidate attained by the second week of treatment. On this combination therapy, her insomnia disappeared and there was a remarkable increase in psychomotor activity. However, bizarre ideation became evident and the patient began to experience cenesthesic hallucinations (i.e., she felt the floor moving under her feet and her body being pulled in different directions). There was also a net increase in auditory hallucinations and she constantly heard "voices and noises." No signs of organic brain syndrome were found at any time.

Because of this exacerbation in her symptoms, methylphenidate was discontinued and a neuroleptic agent, trifluoperazine, was added to the treatment at dosages increasing to 60 mg/day, at which point imipramine was discontinued. With neuroleptic therapy, the patient showed rapid and dramatic improvement, and by 3 weeks later the psychosis had totally faded. Affective blunting and anhedonia were the only remaining symptoms. The patient was able to leave the hospital to be followed on an outpatient basis, and her clinical improvement has been maintained at 2-month follow-up.

Discussion

A connection between lesions of the limbic system and schizophrenic symptoms is supported by the following facts (2).

1. Ablation of limbic structures in animals produces profound changes in affect and emotion, and an impairment in the capacity to "filter out" stimuli (these are thought to be basic disturbances in schizophrenia).
2. Experimental stimulation of the limbic system in humans produces perceptual distortion and feelings of depersonalization.
3. Temporal lobe epilepsy (which commonly involves limbic structures) closely resembles schizophrenia.
4. Encephalitis, a disease that frequently affects the temporal lobes and the basal ganglia, gives rise to schizophrenia-like symptoms.

The dopamine hypothesis, a leading theory in attempts to understand the pathophysiology of schizophrenia, is based on differential effects of dopamine agonists and dopamine blockers on symptoms of schizophrenia. Dopamine agonists such as L-dopa, apomorphine, amphetamines, and methylphenidate produce schizophrenia-like symptoms, while dopamine blockers such as neuroleptics decrease symptoms (1).

Of the dopamine pathways, the mesolimbic dopamine system that connects limbic structures (olfactory tubercle, nucleus accumbens, and stria terminalis) to the midbrain has been called the "seat of schizophrenia" (5). This is also thought to be the relevant tract for amphetamine psychosis, a model psychosis mediated by catecholamines (6). The area of the cingulum is an important part of the limbic system. Tumors involving the cingulate gyrus have been shown to produce florid symptoms of schizophrenia in humans (3). Stimulation of the cingulum produces catatonia-like states in animals (2). Furthermore, frontal lobe lesions

are known to produce schizophrenia-like symptoms, particularly if they involve the cingulum (2).

Psychosurgery causes specific lesions to limbic structures. A cingulotomy involves destruction of white matter with its cingulate fibers above the corpus callosum and approximately 3 cm posterior to the tip of the frontal horn of the cerebral ventricles. Although the "target" lesion encroaches on area 24, adjacent areas of the corpus callosum and calloso marginal gyrus may also be damaged (7). The cingulate gyrus, with its connections to the temporal lobes, the hypothalamus, and other limbic structures, is thought to be a crucial area in controlling emotional and autonomic responses. Cingulotomy is reported to produce beneficial effects in obsessive-compulsive neurosis and depressive disease, but it is thought to be ineffective in schizophrenia (7).

In the case we have presented, a cingulotomy performed to treat a chronic depressive disorder activated schizophrenia-like symptoms. This emergence of symptoms following a cingulotomy is consonant with reports describing schizophrenia-like symptoms in association with different limbic lesions. The worsening of symptoms with methylphenidate administration and their reversal by a neuroleptic drug is of particular clinical interest and suggests the possibility that the symptoms were mediated through catecholamines, mainly dopamine, in the mesolimbic system.

To our knowledge, this is one of the first reports of schizophrenia-like symptoms as a complication of psychosurgery. However, since our observations were primarily retrospective, we cannot rule out the possibility of an insidious schizophrenic syndrome that could have been activated by the surgical procedure.

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The Difficult Defendant: A Case Report

BY HAROLD C. MORGAN, M.D.

Although problems with the behavior of defendants in the courtroom seem to have been increasing during recent years, there has been very little psychiatric literature about this problem.[1] It is my impression that the obstreperous defendant who disturbs, frustrates, and puzzles the court is no longer uncommon. This problem may be manifested in strange or disruptive behavior or threats to the judge, the jury, or to the self. What problems does this present for the psychiatrist who has the responsibility of evaluating the defendant and advising the court? What are his responsibilities to the defendant and to the court? How does he reassure the judge in the face of the defendant's threats of homicide or suicide? How does he reconcile potential conflict of interest between responsibility to the court and responsibility to the defendant? How does the psychiatrist tread the narrow path of neutrality? How does he deal with his own apprehensions?

Answers to these questions do not come easily and there are no absolute standards that can be applied universally, but we can gain some answers from clinical experience with individual cases. One particularly difficult case in my experience recently provided some insights and ideas for resolving these problems.

Case Report

The defendant was on trial for one of several major crimes with which he had been charged. While serving time in prison for another offense, he had been attacked. He was transferred to the state maximum security prison-hospital for his protection and for treatment of emotional problems and was transported daily to court.

During jury selection the defendant, looking dapper and distinguished, "aided" his counsel in the selection process. Soon after the trial was underway, the defendant was described by court personnel as "emotionally upset"; he talked of suicide and switched to prison clothes. I was called to examine the defendant for competency to stand trial.

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[1]A computer search of *Index Medicus* and *Psychological Abstracts* for the past seven years revealed no truly pertinent journal articles.

Before the competency examination a conference was held in the judge's chambers in which counsel for the prosecution and the defense, law enforcement officers, and security personnel were informed of the legal delicacy of the situation and the importance of avoiding a mistrial. The examination itself was conducted with the defendant's attorney and a security guard present. I found the defendant to be angry, scared, and tearful, but competent. The judge and all counsel were informed of these findings and I then informed the defendant personally of my conclusions.

The trial continued with periodic interruptions by the defendant, who threatened suicide and dire harm to others, including his court-appointed attorney and the transporting officers. From time to time, I was called on to assess and assuage the defendant's perturbations and to deal with the apprehensions of all those in court. For the first task, interviews were held with the defendant in the holding room adjacent to the courtroom with security officers stationed nearby. Although it was observed, the conversation was essentially private. He talked freely while I listened and commented supportively. For the second task, group conferences were held in the judge's chambers during which I reported my findings and made suggestions. There was opportunity for questions and answers about the defendant's behavior and the risk of harm to others. This group ventilation afforded a means of relieving anxiety surrounding these risks and fears and drained away part of the hostility that was being focused on the defendant.

Discussion

This two-fold approach to the problem, in which both the defendant's emotions and those of court personnel were examined, served to interrupt the cyclical pattern of acting-out by the defendant and retaliative measures by the court. The bilateral hostility of the opposing camps was relieved and the defendant and his keepers developed more mutually tolerant attitudes. Equally important, the trial was continued to satisfactory completion.[2]

Thus far, I have mentioned only the clinical-administrative difficulties; nothing has been said of my feelings, apprehensions, or misgivings as the psychiatric consultant in this situation. Does the consultant feel in danger when he interviews such a difficult defendant in private? Do the defendant's threats toward others inspire fear that he will similarly threaten—or harm—the psychiatrist? Unequivocally, the answer is yes. Any psychiatrist who denies concern about this risk probably does not understand the situation. The psychiatrist's own apprehensions may prejudice him against the defendant. The consultant must recognize this bias as it arises and try to resolve it immediately in order to have an open mind about the defendant, who will recognize and exploit anything less.

One effective way to resolve this conflict is to recognize the defendant's feelings and to explain clearly the difficult responsibility the psychiatrist has, i.e., being fair and honest with both the defendant and with the court. An understanding of the defendant's legal situation and his emotional reaction to that situation should also be expressed. In short, total fairness and honesty with the defendant is the best protection against threat or assault. It is also the best approach to the sensitive issue of professional loyalties and responsibilities and the best way to remain neutral. As is the case with any potentially dangerous, threatening person, it is wise to have one or more security officers nearby. (I have been assaulted by psychotic offenders, but I have never been attacked or even threatened by a nonpsychotic one.)

In summary, the difficult defendant arouses anxiety and sometimes anger in everyone in the courtroom, from the highest judge to the lowest clerk. The psychiatric consultant to the court is primarily responsible for determining the defendant's clinical state, but he should be aware of the feelings and needs of others in the courtroom. Ideally, the psychiatrist becomes an intermediary. Informing the court of the defendant's feelings along with suggestions for responding to the defendant will usually lead to better understanding of the defendant and diminished apprehension, which will contribute to more suitable behavior by the defendant. It is imperative that the consultant be completely fair and honest in dealing with the defendant. If he is not, the consultant will be professionally ineffective and personally uncomfortable. An honest, unbiased approach of open communication with all concerned generally leads to conformity by the defendant, relief of the court's anxiety and, in the case I have reported, gratitude from both.

[2]After the trial was over, I received handwritten notes of appreciation from both the judge and the defendant.

Transcendental Meditation as an Alternative to Heroin Abuse in Servicemen

BY DOUGLAS J. ANDERSON, MAJ., MC, USAR

Several impressive retrospective studies demonstrate that many former drug abusers who regularly practice Transcendental Meditation (TM)[1] have abandoned their use of marijuana, LSD, amphetamines, barbiturates, narcotics, alcohol, and even tobacco (1–4). This finding suggests that drug abuse might be significantly reduced if abusers could be taught to practice TM as an alternative. I investigated TM as an alternative to drug abuse in young servicemen who had been identified as heroin users. The purpose of this study was to determine whether such a population could be motivated to continue practicing TM and, if so, whether there would be a decrease in drug abuse.

Unpublished prospective studies by Brautigam in Sweden (cited in reference 5) and by G.E. Schwartz in the United States supported this hypothesis by showing a significant decrease in drug abuse in small populations of drug abusers who were taught to practice TM. However, these investigations were biased because the populations studied were particularly interested in learning TM or were novice meditators already. The retrospective studies were also biased in that most of the subjects were highly motivated college students who were well-established in the practice of TM. A more recent unpublished study by Benson and associates (cited in reference 6) of the usefulness of TM in a population of 4,000 high school students showed an insignificant decrease in drug abuse among meditators as compared with controls; in fact, only 1.3% of the subjects could even be persuaded to use meditation regularly. They concluded that TM was not acceptable to high school age populations as an alternative to drug abuse but indicated that the study did not refute the potential usefulness of TM in the treatment of drug abuse in other populations.

It became clear to me that when individuals were motivated to practice TM, a decrease in drug abuse could be expected. The problem was finding out who could be motivated to learn the technique. I therefore decided to determine whether TM might be useful in the treatment of heroin use in young servicemen.

Method

A population of 115 admitted heroin users was studied. The subjects included a few soldiers who volunteered for detoxification, but most were either brought in under the influence of heroin (confirmed by urine drug screen) or were referred for detoxification after a positive result on a random urinalysis ordered by the subject's unit. The subjects ranged in age from 19 to 26 years, with a mean age of 20. Most were weekend users of heroin, and none was physiologically addicted at the time of admission. Detoxification was done on a locked ward, and, although the subjects were given no medication, withdrawal symptoms (if any) were extremely mild. The subjects remained on the ward for 5–7 days. After detoxification, 89 subjects were returned to duty; 26 subjects were in the process of being separated from the service for repeated drug abuse and were evacuated to military hospitals in the United States for final discharge.

All individuals admitted to the hospital for heroin detoxification during the study period volunteered, willingly and with informed consent, to participate in the study. Each subject was taught to practice TM for 15–20 minutes twice each day by a qualified instructor from the Ananda Marga Yoga Society of Berlin.[2] The subjects were also urged to attend Yoga society meetings after discharge. Follow-up was arranged in two ways. The subjects who returned to duty were referred to the Community Drug and Alcohol Assistance Center (CDAAC) for mandatory counseling. All subjects gave permission for confidential consultation with CDAAC counselors who provided follow-up information. The subjects who were in the process of being discharged from the service were given a questionnaire to complete 1 month after returning to civilian life. Basically, they were asked whether they were still meditating and whether they were again using heroin or other drugs.

Results

All of the subjects were enthusiastic about learning TM from their instructor and practiced diligently in the hospital. However, of the 89 subjects who returned to duty, none reported continuing TM after leaving the hospital. Moreover, all returned to some form of drug abuse almost immediately, although most were using drugs other than heroin. Of the 26 subjects who returned to civilian life, only 2 returned their questionnaires. These 2 subjects, who represent 1.7% of the total sample, indicated that they were continuing to meditate and to refrain from drug use. They also were continuing to maintain postal contact with the Ananda Marga Yoga Society after the conclusion of the study.

When this work was done, Dr. Anderson was Chief, Department of Psychiatry, U.S. Army Hospital, West Berlin. He is now Staff Psychiatrist, Long Beach Veterans Administration Hospital, Long Beach, Calif. 90822, and Clinical Instructor in Psychiatry and Human Behavior, University of California, Irvine.

The author wishes to thank Tattvika Amshumalii Brahmacarii of the Ananda Marga Yoga Society of Berlin who served as the meditation instructor for this study.

[1]Transcendental Meditation and TM are trademarks of the World Plan Executive Council-United States.

[2]Although the trademarked term "TM" is used throughout this paper, the Ananda Marga Yoga Society of Berlin is not affiliated with any organization in the United States.

Discussion

The subjects' enthusiasm for learning TM probably resulted from the welcome visits by a nonmilitary instructor who helped to relieve the boredom of confinement. TM was readily practiced by all of the subjects while they were in an environment that provided little else to do. The practice of TM was reinforced in the hospital by regular instruction and peer identification, but after discharge the subjects were on their own. Although additional instruction was available through the Yoga society, none of the subjects made use of it. Therefore, the exposure to TM was unfortunately brief, and this may be one reason for the negative results. Candelent and Candelent (7) have noted that the effects of TM are gradual and usually are not noticeable until after several weeks of regular practice.

The results are interesting and support Benson and associates' conclusion that TM is not an alternative to drug abuse for all populations. TM seems to be of benefit only to those who have the motivation to continue to practice it regularly. If this is true, TM may be no better than any alternative that requires motivation (e.g., a hobby or a sport). Another factor that may have influenced the results is the possibility that this group of young men have not yet completed adolescence and have a need to test limits and challenge authority that might make drug abuse more appealing than any legal alternative.

In conclusion, it seems that TM cannot be offered to any group of drug abusers with the expectation that they will turn away from their drugs and embrace meditation. Further study is needed to determine what differentiates the drug abuser who develops motivation for an alternative from the one who does not.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double-spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Can Dialysis Help the Chronic Schizophrenic?

SIR: The fascinating Clinical and Research Report on "The Use of Hemodialysis in Chronic Schizophrenia," by Herbert Wagemaker, Jr., M.D., and Robert Cade, M.D., (June 1977 issue) prompts me to recount my experience with hemodialysis used to treat a patient with chronic paranoid schizophrenia.

The patient, a 35-year-old single unemployed man, first manifested schizophrenic symptoms at age 21. He had multiple psychiatric hospitalizations in private and state psychiatric facilities, where the diagnosis and illness course were well-documented. While he was an outpatient, the patient developed a water-drinking compulsion-delusion that led to water intoxication and had a major motor seizure with rhabdomyolysis. He then developed acute tubular necrosis and renal failure. The patient had 24 hemodialysis treatments over 8 weeks, with complete return of renal function.

As a psychiatric consultant to the medical ward, I had the opportunity to follow this patient almost daily during and after the dialysis. Although haloperidol, 80–100 mg/day, did decrease his agitation, the patient's psychotic symptoms remained essentially unchanged, with persistent flat affect, looseness of associations, hallucinations, delusions, and suspiciousness throughout the hospitalization. Upon his return to the psychiatric hospital following the completion of dialysis, nursing staff members who were familiar with the patient from previous hospitalizations noted no significant changes in his behavior, symptoms, or hospital course. He did improve enough to be discharged after 7 weeks.

Dialysis did not alter this patient's schizophrenic symptomatology, although it certainly proved useful for his acute renal failure. It seems that he would fall in the "dialysis nonresponsive" category in contrast with most of the "dialysis responsive" patients reported by Drs. Wagemaker and Cade.

I hope that others who have had clinical experience with schizophrenic patients on hemodialysis will report them in order to expand the groups of responders and nonresponders. Carefully controlled double-blind studies are needed to better examine this potentially productive area.

WILLIAM W. WEDDINGTON, JR., M.D.
Durham, N.C.

SIR: I found the article by Drs. Wagemaker and Cade intensely interesting. I would like to detail a professional experience that the authors and *Journal* readers may find provocative. From 1968 to 1973 I was an assistant professor of psychiatry and served as liaison psychiatrist to an organ transplant-hemodialysis unit. I routinely interviewed all patients, both old and new, as well as treating psychiatric complications of all types.

Of relevance to the article by Drs. Wagemaker and Cade was a young man who at 17 had both kidneys removed secondary to nonfunction and blood pressure complications due to glomerulonephritis. A transplant kidney from a related donor did well for about 5 months but was then rejected and removed. Repeated cadaver transplants all failed. When I first saw him, he was receiving hemodialysis every 3 days and had no functioning renal tissue at all. However, he was doing well otherwise. Evaluation showed him to be a reasonably adjusted schizoid personality. Some months later, still on hemodialysis, he suffered an acute paranoid schizophrenic decompensation, his symptoms having gone unnoticed by the staff for about 2 weeks.

Administration of phenothiazines, notably moderate doses of trifluoperazine and chlorpromazine, quickly controlled the acute symptoms. These medications were necessary for 2½ years, after which the patient entered a good remission and psychotropic drugs were discontinued. He remained in good mental health until his death from water intoxication and the complications of long-term hemodialysis about a year later.

During the entire period of his schizophrenic illness, this patient received hemodialysis every 3 days, and no variation in mental status related to dialysis was noted. A change to a more efficient dialysis machine also produced no differences. His remission occurred shortly after he had been dropped as a possible transplant recipient and placed on less frequent and less thorough maintenance dialysis. His mental condition varied, but it did so in relation to the state of renal function, as detailed in a paper by Ferris (1). The role of continuing total absence of functional renal tissue compared with the status of Wagemaker and Cade's patients, who had normal renal function, is a matter for speculation.

If their results can be replicated, I believe the authors have produced a landmark paper. The dialysate content in particular may lead to a major biochemical breakthrough in the understanding of a stubborn and mysterious illness.

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GILBERT N. FERRIS, M.D.
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SIR: The interesting article by Drs. Wagemaker and Cade in the June 1977 issue necessitates some comment. It would be most fortunate if the conclusion that "some chronic schizophrenics are helped by dialysis" stated in this article were true. However, based on physiological reasons and the clinical experience I have accumulated in 13 years of studying these patients, I must express some serious doubts about the possibility of that conclusion being verified.

As to the physiology of kidney function, the ability of hemodialysis to remove both foreign chemicals and normal metabolites is far inferior to that of normal kidneys. Since schizophrenics have normal kidney function, why should their kidneys be less able to remove the hypothetical foreign chemical in their blood in a system superior to man's makeshift artificial kidney machine? As to clinical experience in this area, Reichsman and I intensively studied a group of hemodialysis patients over a period of 4 years at the State University of New York Downstate Medical Center (1). Two patients who were schizophrenic prior to being on dialysis continued to have this illness after they started dialysis and throughout the years of their treatment. In another study that Wynbrandt and I conducted (2), one patient in our series who was schizophrenic prior to the onset of kidney disease was not helped psychologically by dialysis and as a result of the stresses of this procedure had a most arduous course that led to her death.

I have been active in research in this area and have reviewed the literature rather extensively (3). I know of no report of improvement in the psychological state of schizophrenic dialysis patients maintained by this procedure. Of course, this may be due to my failure or the failure of other investigators to make accurate observations. However, another possibility is that the effects Drs. Wagemaker and Cade have reported are either happenstance or a placebo effect of this procedure. I hope that I am wrong.

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NORMAN B. LEVY, M.D.
Brooklyn, N.Y.

Dr. Wagemaker Replies

SIR: We think the substance being removed from the patient's bloodstream is a polypeptide, which probably would not be removed by the kidney. The artificial kidney filters out anything that will pass through the filter.

Dr. Levy mentions three cases of schizophrenics who did not respond to dialysis with psychological improvement. We never claimed that all schizophrenics would respond; in fact, it would seem improbable that all would. Furthermore, the complications of renal failure certainly alter the biochemistry of the body to such an extent that it would be hard to separate out cause and effect.

The placebo effect of dialysis certainly needs to be reckoned with. However, I would not expect this effect to last four years in one case and more than two years in another. We are planning to carry out a double-blind sham dialysis experiment to evaluate this.

Our patients who have responded to dialysis are able to live lives of quality. Many of them are back at work or in school, and they are able to socialize and enter once more into meaningful relationships. Our experiments are preliminary, but we have been greatly encouraged by the improvement in quality of life of our patients following dialysis.

HERBERT WAGEMAKER, M.D.
Louisville, Ky.

Availability of L-Tryptophan

SIR: American psychiatrists must now face another pharmacotherapeutic dilemma. Ernest Hartmann, M.D., in his article "L-Tryptophan: A Rational Hypnotic with Clinical Potential" (April 1977 issue) gives us a very nice summary of already completed research, and it certainly appears that L-tryptophan should have a place in our formularies. We all know, however, that in the United States a physician cannot prescribe L-tryptophan as of this writing. It should be pointed out, however, that L-tryptophan can be purchased at "health food" stores for about 25¢ per 500 mg. It is not clear how a substance can be approved as a "food" and not as a pharmaceutical agent.

Pending approval of L-tryptophan for marketing by the pharmaceutical industry, I would like to ask the following: what are the legal and ethical ramifications if a physician "prescribes" L-tryptophan by advising his/her patient to buy it at the local health food store?

R. HARLAN BRIDENBAUGH, M.D.
JOHN G. LAFFT, M.D.
Washington, D.C.

Dr. Hartmann Replies

SIR: I agree with Drs. Bridenbaugh and Lafft that a dilemma exists: L-tryptophan is readily available as "food" and, in fact, appears on the Food and Drug Administration's (FDA) list of food substances generally recommended as safe. At the same time, it is not available as a drug in the United States, and I have had to conduct many studies under procedures for the investigation of a new drug as if L-tryptophan were a newly synthesized molecule.

I can think of no totally logical reason for the approval of a substance as a food but not as a drug. However, I am sure the FDA can think of some legal reasons. In fact, I have sent a copy of Dr. Bridenbaugh's letter to the FDA, and if I receive an answer I will be happy to share it with him and with the readers of the *Journal*.

To have L-tryptophan approved as a drug would require someone (presumably a pharmaceutical company) to spend a million dollars or more on studies to demonstrate the safety and efficacy of a substance that would then not be patentable. I very much hope a company will accept the challenge and that the FDA will be somewhat accommodating, since the efficacy of L-tryptophan in reducing sleep latency is fairly well established, and widespread clinical experience with its use as an antidepressant should at least partially fulfill the safety requirements. Some studies, including those involving

long-term administration or administration to pregnant animals, will certainly still be required.

In regard to Dr. Bridenbaugh's final question, I do not presently see any ethical problems in advising a patient to obtain L-tryptophan at a health food store. Assuming that the doctor is convinced that there are not contraindications to the use of L-tryptophan, advising the patient to buy it would be similar to advising him to buy vitamins or to obtain a dietary supplement of some kind. I do not know of any legal ramifications, but further clarification of this issue may be forthcoming from the FDA.

ERNEST HARTMANN, M.D.
Boston, Mass.

Editor's Note. Since Dr. Hartmann wrote his response to Drs. Bridenbaugh and Lafft, he has notified us that he received a communication from Stephen McNamara, Associate Chief Counsel for Food of the Food and Drug Administration, portions of which follow.

> Pursuant to the Federal Food, Drug, and Cosmetic Act, (21 USC 321 (s), 342 (a) (2) (C), and 348) if an ingredient used in a food product is not "generally recognized as safe" under the conditions of its intended use, it is a "food additive" and may not be used without prior FDA approval. FDA has promulgated a food additive regulation approving certain limited uses of L-tryptophan. This regulation, however, does not appear to authorize the sale of L-tryptophan in tablet form as a dietary supplement. If, in fact, the "health food" L-tryptophan products to which you refer are not "generally recognized as safe" or encompassed by the food additive regulation, they are subject to regulatory action under the Federal Food, Drug, and Cosmetic Act.
>
> A physician's action in advising a patient to consume, for therapeutic purposes, an article commonly sold as a food is generally not a subject of regulation by the FDA under the Federal Food, Drug, and Cosmetic Act. FDA has stated that the act does not provide the agency authority "to regulate the practice of medicine as between the physician and the patient." Indeed, pursuant to this premise the courts have ruled that the act does not prohibit a physician from prescribing a drug for a new use which has not been approved by FDA.[1]
>
> Of course, if a physician's advice to his patient is not in accord with sound medical practice, the physician may be subject to action under state law. For example, a physician who acts in a negligent manner may be subject to a civil action for damages or to the loss of his license to practice medicine. However, the prescription of an article which FDA has not approved is not necessarily a negligent act.
>
> Your letter expresses doubt that an article may properly be approved by FDA for use as a food but not as a drug. It appears that this circumstance is both reasonable and consistent with law. Speaking generally, the new drug provisions of the Federal Food, Drug, and Cosmetic Act require that a subject article be shown to be both safe and effective for its intended therapeutic use before a new drug application may be approved by FDA. An article that has been approved for a particular food additive use may nevertheless *not* have been shown to be safe and effective for a distinct therapeutic purpose.

[1] *FTC v. Simeon Management Company*, 391 F Supp 697, 706-707 (ND CAL-1975), affd 532 F 2d 708, 716–717 (9th Cir 1976).

Borderline Versus Narcissism

SIR: With reference to the excellent paper "Borderline Rage and the Therapist's Response" by Theodore Nadelson, M.D. (July 1977 issue), I wish to emphasize a distinction between borderline rage (in patients who were sadistically treated by their parents) and narcissistic rage (in patients who were seductively "too well" taken care of by parents who wanted them as companions to counterbalance their own depression and passivity). The borderline person, as Dr. Nadelson pointed out, has not integrated the hostile and caretaking parts of the parental imagoes and projects these hostile introjects onto others. He then has an active fear of his own destructiveness and a passive fear of annihilation.

The narcissistic patient projects rage, but it is secondary to a particular kind of fear. The threat in the narcissistic personality is abandonment and becoming a totally depleted shell in caring for himself. Both borderline and narcissistic patients have intense hate and self-hate, and both types of people engender in the therapist strong desires to retreat or to become overactive and to allow premature termination. Both types of patients accuse us of being ineffectual or rejecting and of feeling hopeless.

Although the symptoms are similar, narcissistic and borderline pathology are distinct and different. The inner needs and dynamics of borderline and narcissistic patients are different, although they may initially present in the same way. The borderline patient needs to control destructiveness mainly by distancing. The narcissist needs to deny separateness and resists demands for autonomy and for self-care and care for others. The rage and self-denigration of the narcissist cover a profound fear of fusion. He feels good only when he feels totally in control but totally and passively taken care of. Action is an intrusion perceived as "too much hassle," but the narcissist complains of being excluded from all the action. Parents of narcissistic children encourage passivity in their children and tell them they are better than and different from the wild and unruly neighborhood children. These mothers are idolized by the child, but later they incur the child's rage when he discovers the world does not reward passivity. Closeness is mistakenly held to be narcissistic fusion and retreat rather than shared activity. At first these "good children" are adored, but later their passivity is treated with annoyance, condemnation, and disgust by the parents. Once again, active coping is bypassed. Revenge and passive withholding then become the life-style, which causes the self-hate and guilt. Separateness causes pain due to loss of a fused self-object. Often these lonely patients inflict isolation on themselves when disappointed and use this pain as a punishment and precondition for their revenge taking.

When the narcissist accuses us of not helping he is saying that we are demanding active coping which exposes his helplessness and intrudes on the blissfully fused narcissistic activities to which he feels entitled. The rage is particularly painful because it is felt against "good parents." For the narcissist, it is not sufficient to delimit hostility and curb projection. The narcissist needs a steady and consistent confrontation with his passive demands when active decision making, sharing, and coping are in order. The rage of being forced to take care of himself must be converted into the pride of being able to give up passive seductive modes and to feel actively in control, including being able to complete

tasks. Covert parental patterns of passivity set up as ideals must be converted to active participation with other people.

Narcissistic and borderline patients are often confused with one another, especially when rage is the presenting symptom. Since they cause very similar countertransference responses, I think it is worthwhile to emphasize their distinction and their different dynamics. Both sets of patients suffer from a conviction of being "unwanted, unloved, unredeemable, doomed, and guilty." The borderline feels this way because of the splitting or lack of integration of his introjects and the projection of both his good and his hostile parts, while the narcissist feels cheated and rejected because of his neediness, passivity, use of people, and rage when he is called upon to act or give. When our patients occasionally combine both of these character pathologies, it is important to interpret appropriately both contexts, to be effective, and to prevent premature termination of therapy.

HELEN STEIN, M.D.
Fresno, Calif.

The Therapist as Participant-Observer

SIR: I very much enjoyed "Psychiatric Eclecticism: A Cognitive View" by Joel Yager, M.D. (July 1977 issue). It is literate, reasonable, and coincides nicely with my own views. I would like to comment on the concept of the participant-observer Dr. Yager mentioned but did not develop.

In Dr. Yager's Venn diagram, the client is seen as a disease by the medical observer, a mind by the psychological observer, an autonomous machine by the behavioral observer, and a cog in a machine by the social observer. It is the therapist as participant-observer who sees the client not as an object but as a person who talks to him and develops the relationship that makes all the rest work. The domain of this observer is the center area of the diagram intersected by all the others. It is from only this position that the therapist can have immediate access to each of the other vantage points, singly or in groups of two or three.

Each of Dr. Yager's four observers plays a fixed role in therapeutic relationships. The medical observer's role is a god and the psychological observer's a father (analysts), mother (social workers), or friend (Rogerians), depending on the therapist's own parataxic distortions. The behavioral observer's role is like an automobile mechanic and the social observer's like a politician.

The participant-observer must be able to use as many roles as possible in order to develop a relationship custom-made for each client. To approach a sociopath in the same way that one would approach a compulsive neurotic or a schizophrenic seems to me to be courting disaster. Nor can one approach two compulsive neurotics in exactly the same way—they are different people and require different relationships.

The exposition of role choices by the participant-observer requires a Venn diagram that I am not yet old enough to draw.

T. STEPHENSON HOLMES, M.D.
Seattle, Wash.

A Neglected Reference

SIR: One of the reasons stated by Nancy C. Andreasen, M.D., and Janusz Bardach, M.D., for presenting "Dysmorphophobia: Symptom or Disease?" (June 1977 issue) was that "A search for references to this term in English yielded only one citation, a definition in a psychiatric dictionary." Hay's overview and report on 17 cases, including 8 case histories (1), was overlooked by the authors and by the reviewer.

This is not to say that Hay's paper gives a sense of closure about dysmorphophobia, which was found to be a nonspecific symptom. However, the picture presented by Hay differed somewhat from that of Drs. Andreasen and Bardach. In short, "Using [Kurt Schneider's] conceptual framework, 11 of the 17 patients were thought to be suffering from severe personality disorder, 5 were schizophrenic, and one had a depressive illness."

The only case that I have treated was a young woman who had an elaborate paranoid delusional system in addition to her continuing concern with the shape of her attractive nose, for which she had had several operations.

REFERENCE

1. Hay GG: Dysmorphophobia. Br J Psychiatry 116:399–406, 1970

KENNETH NAKDIMEN, M.D.
New York, N.Y.

Dr. Andreasen Replies

SIR: My first reaction on learning of Hay's article was chagrin. When conducting our literature search Dr. Bardach and I were surprised to find so little in English, but how could we have missed an article actually titled "Dysmorphophobia" and published in a major journal? I hastened to the library and consulted *Index Medicus* again—first under "dysmorphophobia," then "self-concept," "cosmetic surgery," "plastic surgery," "delusions," etc. Looking under Hay's name to be sure the article was listed in some way for 1970, I found the reference. But how was the subject indexed? As a shot in the dark, I looked under "phobia," and there it was.

I have heard people object to the term "dysmorphophobia" because the disorder is not a phobia, but I have replied that no one takes the term literally. Now I know that indexers do. On the positive side, I also know which haystack to search for this particular needle. In fact, I came away with a veritable pincushion of esoterica with which one could probe the depths of unknowing colleagues' (or Board candidates') "ignorance," if one had a taste for that sort of thing: ergophobia, toilet phobia, entophobia, acarophobia, earthworm phobia, sitophobia, genophobia, traffic phobia, college phobia, neophobia, homophobia, hysterophobia, nosophobia, aerophobia, ornithophobia, and even new-car-buying phobia! In addition to Hay's article, I uncovered articles on dysmorphophobia in many languages and in journals published in Brussels, Moscow, Warsaw, and Paris. Still, our paper could claim to be the first in American, if not in English.

Hay's paper is very fine, and we find that his discussion of dysmorphophobia is strikingly similar to ours. We concur in seeing the common underlying factor as a moderate to severe disturbance in personality and in viewing the personality of the dysmorphophobic as being typically schizoid or obsessional.

As to whether patients with frank delusions or Schneider-

ian symptoms should be classified as dysmorphophobic or some other way, an excellent term used by Riding and Munro (1) has come to my attention. They distinguish between dysmorphophobia and "monosymptomatic hypochondriacal psychoses," the latter being the psychotic equivalent of dysmorphophobia (1), and described seven patients with this syndrome who responded to pimozide.

REFERENCE

1. Riding J, Munro A: Pimozide in the treatment of monosymptomatic hypochondriacal psychoses. Acta Psychiatr Scand 52:23–30, 1975

NANCY C. ANDREASEN, M.D.
Iowa City, Iowa

Psychotropics and Glaucoma

SIR: Stanley W. Hyams, M.D., and Claudette Keroub, M.D., in "Glaucoma Due to Diazepam" (Clinical and Research Report, April 1977 issue), recommended use of evaluation procedures before starting systemic administration of anticholinergic drugs for patients over the age of 40. These procedures are unrealistically time-consuming, expensive, likely to be alarming to the patient, and not justified by current scientific evidence. There are certainly other possible explanations for their single case report of closed-angle glaucoma in association with diazepam, such as intrinsically increased adrenergic tone or decreased cholinergic tone. However, weaknesses in the authors' causal interpretation of the limited data are not my main concern here.

As the authors acknowledge, not a single case of glaucoma caused by diazepam has been reported previously. The benzodiazepines have few or no anticholinergic effects. The incidence of glaucoma has never been shown conclusively to be higher in groups taking psychotropic drugs than in the general population (1). The risk among the adult population of developing acute glaucoma is only 1 in 4,000, even when challenged with strong topical anticholinergics such as atropine (1). With drugs of lesser anticholinergic potency, and recognizing the much milder autonomic effects of orally administered drugs, the figure must be even lower.

A recent comprehensive review of these issues (2) included the following realistic recommendations for evaluation before the use of anticholinergic drugs in patients over the age of 40.

1. Check pupil size before and occasionally during medication to observe for dilation.
2. Check for a personal or family history of glaucoma, as well as eye examinations that included dilation.
3. Check for a history of "halos" around lights associated with eye pain.
4. If any of the above areas are suspicious, referral to an ophthalmologist for further examination is indicated.

Even if glaucoma is present, it is generally safe to proceed with psychotropic medication, provided antiglaucoma treatment is prescribed and the patient is followed by an ophthalmologist (2). Recent data also have enabled the physician to select a psychotropic drug on the basis of its relative anticholinergic activity (3). Among the tricyclics, amitriptyline has the strongest anticholinergic potency, approximately 17 times that of desipramine, which has the least anticholinergic potency. Therefore, one might choose desipramine when anticholinergic effects would be a liability.

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1. Grant WM: Ocular complications of drugs. JAMA 207:2089–2091, 1969
2. Reid WH, Blouin P, Schermer M: A review of psychotropic medications and the glaucomas. Int. Pharmacopsychiatry 11:163–174, 1976
3. Snyder SH, Yamamura HI: Antidepressants and the muscarinic acetylcholine receptor. Arch Gen Psychiatry 34:236–239, 1977

CHARLES L. BOWDEN, M.D.
MARTIN B. GIFFEN, M.D.
San Antonio, Tex.

Drs. Hyams and Keroub Reply

SIR: We agree with Drs. Bowden and Giffen that glaucoma caused by mild anticholinergic drugs is rare and that glaucoma due to diazepam has never been reported. We emphasized this in our paper. We were also careful to point out that a causal relationship between treatment with diazepam and the development of glaucoma in our case was probable, not certain.

However, having seen the report by Friedman and Neumann (1) of two patients blinded by benzhexol, I feel that one cannot ignore the admittedly slight danger associated with these drugs.

As we and your correspondents stated, a glaucoma patient could probably continue to receive anticholinergic drugs if he was under the care of an ophthalmologist and if a simple procedure could be used to select those patients requiring special observation. The modern classification of glaucoma divides the disease into open-angle glaucoma (which is always chronic) and closed-angle glaucoma (which may be acute, subacute, or chronic). The patient we reported and those reported by Freidman and Neumann (1) had chronic closed-angle glaucoma. Bearing this classification in mind, the criteria for evaluation of patients before the use of anticholinergic drugs, which were suggested by Reid and associates and quoted by Drs. Bowden and Giffen, are not reliable. Dilation of the pupils is not a significant sign of chronic closed-angle glaucoma, except in a very advanced stage of the disease. Family history of glaucoma is more pertinent when one is dealing with open-angle glaucoma, and a history of "halos" associated with eye pain will be obtained very rarely in patients with chronic closed-angle glaucoma. Applanation tonometry, gonioscopy, and examination of the optic disc is a routine that should take less than 10 minutes. This does not seem a long time to devote to a patient who is under prolonged psychiatric care. Provocative tests are rarely necessary.

REFERENCE

1. Friedman Z, Neumann E: Benzhexol induced blindness in Parkinson's disease. Br Med J 1:605, 1972

STANLEY W. HYAMS, M.D.
CLAUDETTE KEROUB, M.D.
Afula, Israel

Maintenance Antipsychotic Dosage and Tardive Dyskinesia

SIR: I would like to underscore the comments made by George E. Crane, M.D., on "The Prevention of Tardive

Dyskinesia" (July 1977 issue) with a brief vignette from my clinical practice.

The patient, a 43-year-old man, had been treated with trifluoperazine, 20 mg q.h.s., for a chronic schizophrenic process. He had been on this dose for 13 years and, when I first saw him, had the most severe oral-bucco-lingual and digital dyskinesia I had encountered. His dyskinesia had been first documented only 2 years previously, but poor record keeping made it impossible to date accurately the onset of symptoms. His AIMS score in August 1975 (done as a part of an evaluation for a research project on tardive dyskinesia) was 18 out of a possible 42. Because he had been asymptomatic for over 10 years, I began to reduce his dose of medication. There was no change in his tardive dyskinesia until he had been on trifluoperazine, 10 mg q.h.s., for 3 months, when his dyskinesia rapidly improved. His AIMS score in May 1977 was reduced to 4, with only minimal oral and lingual movements.

This case shows that decreasing the maintenance antipsychotic dose may actually lead to an improvement in the symptoms of tardive dyskinesia if one waits long enough. In addition, instances of marked improvement in long-standing tardive dyskinesia raise questions about the permanence of the symptomatology. Perhaps tardive dyskinesia is always reversible, if one waits and minimizes dopamine receptor blockade for a long enough period of time.

Dr. Crane did not mention the possible role of antiparkinsonian agents in causing tardive dyskinesia. As this possibility has been suggested by Kobayashi (1), it is another reason why the physician should avoid prophylactic use and minimize the long-term use of antiparkinsonian drugs.

REFERENCE

1. Kobayashi RM: Tardive dyskinesia. N Engl J Med 296:257–260, 1977

KENNETH SOLOMON, M.D.
Albany, N.Y.

Trihexyphenidyl Abuse

SIR: "Abuse of Antiparkinsonian Drugs by Psychiatric Patients," by Katherine MacVicar, M.D. (Clinical and Research Report, July 1977 issue) reinforces observations that I made at San Quentin Prison in 1963. At that time, trihexyphenidyl (Artane) was widely enjoyed by the inmates as a drug of abuse. One man told me, "If I take five Artanes, I can see my mother. If I take 10 Artanes, I can see my girlfriend." The use of trihexyphenidyl as a psychedelic needs to be reinforced and publicized. Dr. MacVicar's paper is a valuable contribution.

THOMAS P. LOWRY, M.D.
Imola, Calif.

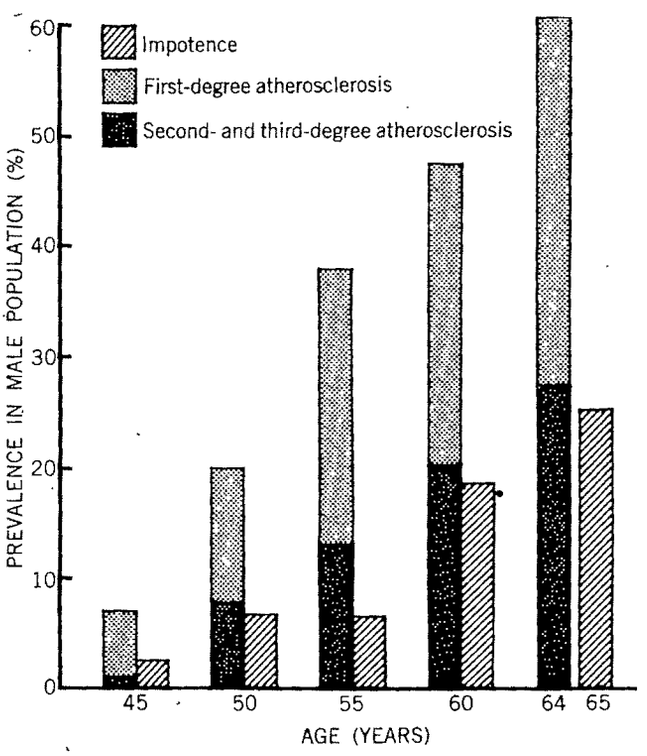
SIR: We read "Abuse of Antiparkinsonian Drugs by Psychiatric Patients" with much interest and would like to comment on another area of trihexyphenidyl abuse that has come to our attention. In the course of consulting at a local prison, we learned that the inmates crush the trihexyphenidyl tablets, mix the powder with tobacco, and smoke the mixture in order to obtain a high. The tablets serve as prison currency for which the inmates gamble.

We mention this form of trihexyphenidyl abuse so that clinicians who work in prisons will consider this etiological possibility when confronted with euphoria or a toxic psychosis in inmates. Clinicians should be cautious about prescribing this medication to an inmate; they should be certain that an extrapyramidal reaction is in fact present and that the inmate swallows the tablet when it is dispensed.

ALVIN M. ROUCHELL, M.D.
STROTHER P. DIXON, M.D.
New Orleans, La.

Correction

In the August 1977 issue, figure 1 in "Impotence Responsive to Glyceryl Trinitrate" by John W. Mudd, M.D. was incorrect. The corrected figure appears here.



BOOK REVIEWS

Modern Synopsis of Comprehensive Textbook of Psychiatry/II, *by Alfred M. Freedman, M.D., Harold I. Kaplan, M.D., and Benjamin J. Sadock, M.D. Baltimore, Md., Williams & Wilkins Co., 1976, 1,336 pp., $35.00.*

In the belief that the two volumes of the second edition of the *Comprehensive Textbook of Psychiatry* (Williams & Wilkins Co., 1975) are a bit voluminous, especially for medical students, the authors decided to condense, abbreviate, and modify its contents and publish this synopsis. The synopsis of the textbook's first edition was published in 1972; this second edition is double the size of the first, and, due to the many changes in the discipline, over 75% of the book has been rewritten. Some of the material deemed not so important for the immediate use of medical students has been eliminated, new and more cogent material has been added, and certain key material has been brought up to date. The expansion in psychiatric knowledge has been explosive, and graduate, postgraduate, and continuing education demand more attention and hold promise of even greater requirements.

Everyone is aware that there is a perplexing array of new drugs available in psychiatry. Students and practitioners must of necessity understand their properties, modes of action, and effects on the CNS. As these authors note, there is a need for the sophisticated dispensing of these agents, lest the condition they inflict be worse than the condition that called them forth. In addition to the new pharmacological agents and new advances in neurophysiology, advances in knowledge in the social sciences also clamor for attention because the environment and surroundings of the individual influence other aspects of his or her life. Along with all of this, the advances in clinical psychiatry are also noteworthy. Continuing education for all professionals is a present-day watchword as plans for national health insurance move into the foreground. More and more demands are being made on the psychiatrist as Professional Standards Review Organizations (PSROs) take form.

Thus, there is ferment in the profession as it seems slowly and painfully to wend its way back in the direction of clinical medicine, from which it evolved and by which it has been nurtured. This book takes cognizance of this fact. The authors realize that in the present state of the discipline an eclectic approach is needed; none of the components that make up the specialty can be neglected. The authors hope that this book will be "an organizing statement for the multitude of relevant variables and an initial approach to a complex model." Although its basic orientation seems to be similar to that of the first edition, the plethora of new subjects that require attention made this new book a necessity.

Random sampling of the various subjects indicates that the authors have succeeded in their efforts. There are interesting descriptions of almost everything the student requires. Not only the student but the experienced clinician will be able to refresh his or her memory of the multitude of subjects he or she is required to know. In addition, there is an excellent glossary, along with a comprehensive index. All of this goes together to make a first-class, useful synopsis of a field that is rapidly becoming more complex. One can certainly recommend this book without hesitation. It is particularly well done.

F.J.B.

My Analysis with Freud: Reminiscences, *by A. Kardiner, M.D. New York, N.Y., W.W. Norton & Co., 1977, 123 pp., $6.95.*

Dr. Kardiner's lively and frank account of his analysis with Freud in 1921 and his reflections about its effect on his life reveal intriguing aspects of the work and character of both men. Dr. Kardiner, whose early life had been traumatic and unpromising, was amazed and excited to have the opportunity to be analyzed by Freud. He expected to be awed by the great man but was pleasantly surprised by Freud's friendliness, modesty, and humor and by his willingness, on occasion, to talk about himself and his colleagues. Kardiner has never understood why Freud discussed personal matters with him; he learned from Freud's other analysands that this was not Freud's practice.

The humility and critical self-appraisal that Freud conveyed to Kardiner contrasted with Freud's expectation of loyalty and with the authority he assumed for the growth and development of psychoanalysis. Freud controlled the economic fate and professional status of analysts. Although Kardiner has enormous admiration for Freud's genius, he thinks it unfortunate that he had so much power because it led to rivalry and infighting among Freud's followers. In spite of their competitiveness, however, they worked together diligently and learned from each other; Dr. Kardiner states that he feels privileged to have learned from them. He describes with enthusiasm his participation in the activities of the Vienna Psychoanalytic Society. While he was in Vienna, the first series of didactic courses ever offered by a psychoanalytic group was organized.

Dr. Kardiner is grateful for the salubrious effect on his personal and professional life of his contacts with Freud the man, the therapist, the investigator, and the teacher. Freud's insights and understanding helped Kardiner to overcome his fears and inhibitions and to become more confident and assertive. Thus, his personal life was enriched and he was more free to pursue his career as an analyst.

Kardiner believes that Freud's momentous contributions to psychoanalysis can be divided into two categories: the delineation of intrapsychic processes and the emphasis on the importance of the environment in human development. It is likely that Dr. Kardiner's appreciation of the effect on his life of early trauma and his capacity to adapt to it accounts in part for his interest in the role of culture in human development. His outstanding contributions in this field are well known.

Dr. Kardiner ends his book with a plea for more field investigations of a variety of cultures. He believes that from such studies we will be able to develop directives for child

rearing and identify successful patterns of adaptation; he feels that this information will help us cope with the ills of our times. Although I agree with Dr. Kardiner that such studies should be encouraged, I am not as optimistic as he about what we can expect from them. In his enthusiasm for field studies, he recommends that we stop studying people on the couch. I disagree with this recommendation and wonder how Dr. Kardiner reconciles it with his own valuable experience on the couch.

REBECCA Z. SOLOMON, M.D.
Hartford, Conn.

Chemotherapy in Psychiatry, *by Ross J. Baldessarini, M.D. Cambridge, Mass., Harvard University Press, 1977, 196 pp., $9.95.*

This is a very well organized, extremely readable book that is a must for every psychiatric resident and for the practicing psychiatrist. It will also be an extremely useful addition to the reference library of the internist and the family practitioner. Dr. Baldessarini has organized the information in this volume by drug types, starting with the antipsychotic agents and continuing through the lithium salts, antidepressant agents, and antianxiety drugs. In the concluding chapter he comments briefly on a series of special issues, such as geriatric and pediatric psychopharmacology.

The information provided is given in adequate detail and in a sufficiently condensed form that it is accessible and useful for rapid review in a given clinical situation. Each chapter starts with a description of the various chemicals in each class of therapeutic agents, including their chemical structure, followed by a discussion of their pharmacological activity. The author provides detailed directions for clinical use of these drugs, including a careful discussion of toxicity and side effects, an assessment of the relative dosage strengths, and the efficacy of currently available psychoactive compounds.

I believe that most skilled psychopharmacologists will agree with the various recommendations made in this volume regarding the use of the different chemical agents. Dr. Baldessarini draws a fine line between an adequately conservative approach toward the use of these compounds and the necessary emphasis on their significance in the management of serious psychotic and neurotic illnesses. If there is a fault to be found with the volume it is that, although the bibliography is nicely organized by topics, it is not referenced to specific statements in the body of the text. However, the bibliography is comprehensive and will provide the serious student of psychopharmacology with an excellent set of references for detailed information on the subjects covered in the book.

BERNARD C. GLUECK, M.D.
Hartford, Conn.

The Annual of Psychoanalysis, Vol. IV, *edited by the Chicago Institute for Psychoanalysis. New York, N.Y., International Universities Press, 1976, 500 pp., $17.50.*

Like the three volumes preceding it, this book covers a wide range of topics in psychoanalysis. Some aspects are covered more completely than others; child analysis, for example, is given relatively little attention. Although most of the articles are from the Chicago Psychoanalytic Institute, some individual chapters are written by other distinguished authors.

This book holds the reader's interest in each chapter, regardless of the area dealt with. Most of the chapters contain good historical background, complementing the first section, Psychoanalytic History. Part two, Clinical Theory, deals with the narcissistic disorders as differentiated from oedipal fixations. In various subsections the authors discuss in some detail the beginnings of Freud's work and compare conditions in Freud's era and now. The chapter by Benedek is particularly intriguing: she discusses not only women analysts but also some of the latest thinking about feminine and masculine psychology. This is interesting reading in the light of various liberation movements.

Section three, Developmental Psychology, presents a fascinating exchange of ideas between Freud and Piaget. The book on the whole is not totally Freudian; it deals with other areas in which psychoanalysis could be or has been involved. The section titled Clinical Psychoanalysis devotes itself primarily to the postencephalitic syndrome, about which we know very little but to which psychoanalysis can contribute a good deal. There is a pertinent section titled Psychoanalytic Education, which is concerned mainly with the selection of analytic candidates. This is an issue about which there is still disagreement; the discussion of the usefulness of single and group interviews presented here indicates that both seem to be of value.

The section on Interdisciplinary Research includes two chapters, one on psychoanalysis and communication science and the other on aspects of the dynamics of prejudice. The final section has two chapters that would be of great interest to the scholar, "The Theme of Incest in the Myth of Osiris" and "The Suicide of Richard Corey," an in-depth analysis of a poem.

I feel that this volume would be an excellent addition to one's library. Some of the reading is a bit tedious in parts, but it is nevertheless an enticing book and well worth reading.

STUART M. FINCH, M.D.
Tucson, Ariz.

The Unconscious God: Psychotherapy and Theology, *by Viktor E. Frankl. New York, N.Y., Simon & Schuster, 1975, 159 pp., $6.95.*

Although the most recent of Dr. Frankl's books to be published in the English language, *The Unconscious God* is actually one of the first books by the renowned Austrian existential psychiatrist and founder of logotherapy. First published as *Der Unbewisste Gott* in 1947, the present edition contains both an English version of the original work (essentially unchanged except for minor alterations) and an extensive postscript in which the author discusses the development and maturation in his thinking over the intervening decades. The two sections of the book serve to complement each other; they are followed by an extensive updated bibliography.

The first section of the book, the English version of the original German edition, consists of several chapters. Here the author develops his thesis that a religious sense is rooted in the unconscious depths of every man and that only by awareness of this dimension can man be fully understood in terms of his uniqueness, his capacities, and his response to

life and a meaning beyond himself. The chapter titled "The Spiritual Unconscious" is especially pertinent; it contains a concise and helpful discussion of the concept of dimensional ontology that is invaluable in unifying any multidimensional approach. Other chapters are devoted to the essence of existential analysis, existential analysis of conscience and dreams, the transcendent quality of conscience, unconscious religiousness, and psychotherapy and theology.

The second part of the book contains Dr. Frankl's reflections on the tenets proposed in the first section in the light of his present thinking. He also presents supportive evidence gained by studies assessing the value of logotherapy as a treatment modality. This becomes rather tedious at times because many studies are cited that arrive at what appear to be gross generalizations in support of logotherapy. This is not to question the value or efficacy of logotherapy but, rather, to alert the reader to be discerning of the more substantial evidence in support of this treatment approach.

This is not an easy book to read, and the problem seems to derive as much from aspects of the author's presentation as from the complexity of the subject matter. Although this edition is written in English, much of the author's thinking remains in German, often creating a sense of unfamiliarity until one becomes aware of the style and makes the necessary adjustments. Of more concern, however, are aspects of the author's reference to other treatment modalities, particularly psychoanalysis. His statements in this regard seem overgeneralized, challenging, and, at times, provocative. As a result the author tends to detract from his own thesis and runs the risk of alienating readers who would otherwise be quite open to serious consideration of the many profound ideas in this book.

HALLIE E. MOORE, M.D.
Hartford, Conn.

Psychotherapy Versus Behavior Therapy, *by R. Bruce Sloane, Fred R. Staples, Allan H. Cristol, Neil J. Yorkston, and Katherine Whipple. Cambridge, Mass., Commonwealth Fund in cooperation with Harvard University Press, 1975, 259 pp., $10.50.*

As the variety of therapeutic interventions available to psychiatrists continues to increase, it is essential that criteria be developed for fitting the proper treatment to the proper patient. Ultimately, these criteria will come out of research. Until the last two decades, research in psychiatry provided little practical guidance for the clinician. Anecdotal studies in which various practitioners described the impact of a particular intervention on a particular group of patients provided general guides to treatment but proved nothing. Later studies using control groups and documenting the effectiveness of particular interventions as compared with no intervention were much more useful. However, these still did not provide the clinician with guidance as to the best treatment for the individual patient. Recently, behavioral scientists have begun to study the relative effectiveness of different interventions on people with similar disorders. These studies will ultimately contribute to the development of effective and rational psychiatric interventions.

In *Psychotherapy Versus Behavior Therapy*, Sloane and associates concisely but thoroughly describe their efforts to discover the effectiveness of two modes of intervention with neurotic patients. They approach their task with considerable sophistication. In the first chapter, "How Psychotherapy Has Been Studied," they review the methodological problems involved in psychiatric research. Their description of the task of defining a patient population, a treatment modality, and outcome variables is excellent. This chapter alone should definitely be read by the psychiatrist.

Fully aware of the pitfalls of psychiatric research, the authors devised an elaborate project in which they compared patients receiving classical psychoanalytically oriented individual psychotherapy with patients receiving behavior therapy and with a control group of patients who were waiting to be placed in therapy. The 11 major features that the authors consider to be distinctive to their study were designed to avoid the pitfalls characteristic of so much psychiatric research. In this study the authors were careful, patient, and exhaustive. They were scrupulously fair in not structuring the study so that the results would favor one intervention. The methodologies used to define the patient population, treatment modality, and outcome criteria are carefully described, as are methods for using independent assessors and test modalities.

The results of the study are interesting and may even be surprising to some, but they do not come close to resolving the question of what is the best way to treat neurotic patients. Psychotherapy and behavior therapy turned out to be more or less equally effective in treating the research population, and both modalities were more effective than no treatment. However, the value of this book does not lie in its presentation of results. *Psychotherapy Versus Behavior Therapy* is an important contribution to psychiatry because it beautifully demonstrates how interventions in psychiatry can be studied if researchers are dedicated enough and patient enough to commit themselves to thorough follow-up research. It is the best available brief guide to dealing with methodological problems in research in psychiatry and should serve as an inspiration to those willing to commit themselves to the difficult but absolutely essential task of studying which interventions work best with which patients.

SEYMOUR L. HALLECK, M.D.
Chapel Hill, N.C.

Current Psychiatric Therapies, Vol. 16—1976, *edited by Jules H. Masserman, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1976, 334 pp., $28.50.*

This 16th volume of what has deservedly come to be an essential part of any psychiatric library is introduced by Judd Marmor with a chapter on "The Psychotherapeutic Process." The rest of the book covers a broad range of subjects and treatment modalities with an almost cookbook practicality. Dr. Marmor demonstrates his own eclecticism when he lists the common denominators of psychotherapy as a good patient-therapist relationship, release of tension, cognitive learning, operant reconditioning, suggestion and persuasion, identification with the therapist, reality testing, and emotional support.

The remainder of the book is divided into sections labeled Childhood and Adolescence; Adult Psychotherapies; Drug, Hormonal and Somatic Therapies; Addictions; Family, Group and Institutional Therapies; and Psychiatric Vectors in Population Control. Some pathologies appear in more than one section; the mentally retarded and their treatment possibilities are discussed in part one and again in part two. It is heartening to note that George Tarjan's insistence that this neglected group of patients has potentials that can be

developed seems to have been adopted. Menolascino and Simmons and Tymchuk are anything but pessimistic in their contributions about the possibilities of dealing therapeutically with these people. Similarly, Cancro's chapter, "Comprehensive Therapy of the Schizophrenic Syndrome," and Ban's "Pharmacotherapy of Schizophrenia," offer succinct and practical therapeutic suggestions for the management of the most prevalent of the psychiatric illnesses.

In the second section there is a series of brief discussions of various types of adult psychotherapies, including television therapy, primal therapy, and existential therapy; there is even a short chapter on humor in psychotherapy. There are good clinical illustrations for many of the points the authors make.

The section on drug, hormonal, and somatic therapies is especially to the point; the two chapters on the indications for lithium therapy, its use, its management, and methods of preventing untoward side effects should be required reading now that more and more conditions are being treated with this chemical. Equally useful are the chapters on haloperidol and injectable fluphenazines.

The sections on addictions and on psychiatric vectors in population control round out the broad coverage provided in this volume, but an especially interesting series of chapters appears in the section on family, group, and institutional therapies. Dr. Masserman gets this section off to a good start by a chapter that includes a case report of a sophisticated female patient whose endless dynamic ramblings were helpfully interrupted when he insisted on treating a puncture wound of her foot with tetanus antitoxin before he would let her proceed. Some of the succeeding chapters recognize the changing loci of appropriate treatment by discussing psychotherapy in the home, hotel therapy, and the use of folk healers as associate therapists. The chapter on correctional psychiatry understandably is directed more at stating the problems than at offering solutions, but an unusual aspect of the discussion by Dy and Herzberg is their emphasis on the importance of the psychiatrist's understanding the problems of the guards and the institution as well as those of the inmates.

The editor and the authors are to be commended for this extremely useful series.

C.H. HARDIN BRANCH, M.D.
Santa Barbara, Calif.

Foundations of Biochemical Psychiatry, *edited by David S. Segal, Ph.D., Joel Yager, M.D., and John L. Sullivan, M.D. Boston, Mass., Butterworths, 1976, 337 pp., $19.95.*

This volume adds further support to the biochemical basis of psychiatric illness. The book consists of seven major sections and is multiauthored. It is a compilation of a series of very important and classic review articles dealing with various aspects of biological psychiatry. Most of the material has been previously published; the virtue of this volume is that it makes the articles available in a handy form. Clearly the editors have made a contribution by bringing these papers together. It is a good introduction to the biochemical foundations of the discipline for residents in psychiatry.

The book begins with an introduction to fundamental concepts: abnormal behavior, biochemical considerations, and research strategies. The second section begins with a clinical description of schizophrenia. L.L. Heston reviews the genetics of schizophrenic and schizoid disease, and R.J. Wyatt, B.A. Termini, and J.M. Davis review the literature from 1960 to 1970 on biochemical and sleep studies in schizophrenia. A review by E.H. Ellinwood, Jr., on amphetamine psychosis as a multidimensional process appears next, followed by S.H. Snyder, S.P. Banerjee, H.I. Yamamura, and D. Greenberg on the effects of drugs, neurotransmitters, and schizophrenia. In addition, L. Stein and C.D. Wise review their data on the possible etiology of schizophrenia in relationship to the noradrenergic reward system by 6-hydroxydopamine.

The third major section of the book deals with the affective disorders. E.S. Gershon, D.L. Dunner, and F.K. Goodwin review the genetic contributions of affective disorders; J.J. Schildkraut reviews the catecholamine hypothesis of affective disorders; and F.K. Goodwin, D.L. Murphy, H.K.H. Brodie, and W.E. Bunney, Jr., review the relationship of L-dopa, catecholamines, and behavior of depressed patients. B. Shopsin, S. Wilk, G. Sathananthan, S. Gershon, and K. Davis critically review the catecholamine hypothesis, and A. Coppen then reviews the role of indoleamines in affective disorder. A review by D.S. Janowsky, M.K. El-Yousef, J.M. Davis, and H.J Sekerke relating a cholinergic-adrenergic hypothesis of mania and depression follows.

Section four deals with the neuroses and their classification, including G.D. Miner's review of the evidence for genetic components in neuroses and D.M. Warburton's discussion of modern biochemical concepts of anxiety. Section five reviews briefly the hyperactive child syndrome. S.H. Snyder and J.L. Meyerhoff suggest how amphetamine acts in minimal brain dysfunction, and L.E. Arnold, P.H. Wender, K. McCloskey, and S.H. Snyder compare the comparative efficacy of levo- and dextro-amphetamine in the hyperkinetic syndrome.

Section six deals with the subject of alcoholism. A review by D.W. Goodwin, F. Schulsinger, L. Hermansen, S.B. Guze, and G. Winokur on alcohol problems in adoptees raised apart from alcoholic biological parents highlights this section. Section seven deals with the subject of psychoendocrinology, including E.J. Sachar's discussion of hormonal changes in stress and mental illness. In addition, references and a glossary are provided.

In summary, this convenient volume contains a large selection of important reviews published in various journals. Although it contains little that is new, the book serves a useful purpose. It is recommended for those with a special interest in biological psychiatry and for psychiatric hospitals and medical libraries.

E.F. DOMINO, M.D.
Ann Arbor, Mich.

Drug Interactions: Clinical Significance of Drug-Drug Interactions and Drug Effects on Clinical Laboratory Results, 3rd ed., *by Philip D. Hansten, Pharm.D. Philadelphia, Pa., Lea & Febiger, 1975, 378 pp., $11.50 (paper).*

This book delivers more than the title promises; in addition to drug interactions, which are covered in the first half of the volume, drug effects in clinical laboratory tests are described in part two, which is somewhat shorter than part one. Coming at a time of increasing consciousness of the importance of drug interactions, increasing use of potent pharmaceuticals, and increasing reliance on laboratory tests, this work fills an obvious need. Even on cursory review one can understand why it has had three editions since 1971 and has already been translated into Japanese and German.

This is a reference book based on the use of the index; the author states in the first paragraph that one must use the index. Following the instructions one finds the book logical in arrangement and easy to use because the print is clear and the paper of good quality. Part one consists of sections 10–20 lines long covering drug interactions. Each section contains a few lines on the mechanism of interaction, a statement on clinical significance, and a statement on management; if no special management is required this is stated. Virtually every assertion is documented with one or more references; the total bibliography is extensive.

To become acquainted with the volume the reader might turn to a familiar drug group, such as the phenothiazines, in the index. There he or she will find that some 23 drugs interact with this group and that the phenothiazines have an effect on 16 tests of blood and urine. Because bold type is reserved to identify well-documented interactions of major significance, it is important that none of the names of the interacting drugs is printed in bold type. Six of the interacting drugs are listed in italic type, which means that they produce interactions of moderate significance; the names of the rest of the drugs in this list are printed in ordinary type because they are considered of minor significance.

On the other hand, the list of interactions found under tricyclic antidepressants shows bold type for interactions with bethanidine, epinephrine, guanethidine, and levarterenol, and the monoamine oxidase inhibitors as a group have seven drugs in bold type in their listing. Similarly, we see at a glance that disulfiram requires special attention when given with diphenylhydantoin or oral anticoagulants.

There is much more of similar interest in this book, but one must remember that it is not a general text on adverse reactions and that the descriptions are limited to interactions among drugs, even when one might expect some comment, e.g., concerning the three-way interaction that occurs when a person taking diphenylhydantoin to control epilepsy is given phenothiazines. Only the interaction between the two drugs is mentioned, so the clinician will have to keep in mind the more significant reaction between the convulsive tendency of the patient and the phenothiazines. Similarly, the description of the interaction between ethyl alcohol and diphenylhydantoin ends with the comment that "epileptics receiving diphenylhydantoin (DPH) who drink heavily should be watched more closely for decreased anticonvulsant effect." This relates only to the interaction between the two drugs; it does not extend to the very serious interaction between epilepsy and alcohol because epileptic patients who drink heavily represent a special problem.

The psychiatrist who reads carefully might raise such questions as why chocolate and banana are listed in the index but processed cheese is not (at least I did not find it), but these are minor details. The language of the text is such that it more than compensates for any quibbles that might be raised; it is clear and explicit without any hint of package-insertese (to coin a term), and it alerts the clinician but still leaves a healthy degree of flexibility for exercise of judgment. Keeping these and other current matters in mind one can give thanks as he or she wanders through the pages of this book that "this adverse potential [for drug interaction] is realized in a relatively small proportion of cases receiving the interacting combinations." However, one cannot forget that the lists of interactions for many drugs are long and that many of the names are printed in bold or italic type. In the nature of such things these lists can only grow longer and more demanding of attention as time goes on.

As documentation grows it will be less and less possible to attribute adverse effects to simple drug idiosyncrasy or underlying disease. We have learned in the past few years that we must ask every new patient what drugs he or she has been taking. This promises to become an ongoing question in the context of treatment. For dealing with this source of growing complexity in medicine there will be an increasing need for the type of reference provided by *Drug Interactions*.

HENRY BRILL, M.D.
Islip, N.Y.

Pharmacology: Drugs Affecting Behavior, *by Conan Kornetsky. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1976, 258 pp., $18.95.*

Knowledge about the effect of drugs on behavior is burgeoning at such a rapid rate that writing about it immediately introduces problems of focus and organization. Dr. Kornetsky is not deterred by these two challenges. He states at the outset that he is aiming his remarks at a nonmedical audience. However, he feels, correctly, that many physicians and some psychiatrists could also benefit from this overview of psychopharmacology. He first sets out to devise a primer of the principles of general pharmacology; this is a model of clarity and succinctness. He describes such fundamentals as absorption, distribution, receptor sites, dose-effect curves, and the impact of tolerance, hypersensitivity, idiosyncratic reactions, allergic responses, and genetics.

Following the exposition of these general pharmacological principles, which apply to any drug, the author goes on to put in proper perspective the role of placebo and nonspecific factors in determining drug response. Because most of the drugs used in the treatment of mental illness have actions involving the autonomic nervous system, Dr. Kornetsky devotes an entire chapter to reviewing this system and the pharmacology of the drugs that act primarily on it. A brief and somewhat spotty chapter on the neurophysiology and biochemistry of the CNS completes this concise primer of fundamentals, which the reader needs in order maximally to understand the remainder of the volume.

Dr. Kornetsky chooses to categorize psychotropic drugs as antipsychotic, antidepressant, and antianxiety medications, narcotic analgesics, hypnotics, and sedatives, with an additional section on the amphetamines. This choice differs little from the choice of most other investigators and teachers writing about psychotropic agents. In addition, however, he provides chapters on alcohol, anti-epileptic medications, and drugs for the treatment of minimal brain dysfunction (the hyperkinetic syndrome) as well as an extensive chapter on the nonmedical use of drugs. These are welcome supplements and serve to make this a truly well-rounded volume about drugs that affect behavior. An additional attractive aspect of the volume is the glossary of street terms. Such colorful terms as "cartwheels" for amphetamine tablets scored crossways, "double trouble" for Tuinal (a barbiturate capsule containing both secobarbital and amobarbital), "TNT" for heroin, and "Acapulco gold" for high-grade marijuana are only a few of the very descriptive and authentic terms used to describe various forms of drugs.

It might be argued that Dr. Kornetsky could have focused in greater depth on some of the areas he writes about rather then attempting to touch so many bases. Actually, the broad panorama covered by the author is one of the book's strengths. Although many students and investigators who

are interested in antipsychotic and antidepressant agents might not be particularly interested in anti-epileptic drugs or minimal brain dysfunction, the material is here for them if they need it. On the other hand, if the reader does not want to review so many disparate fields, he or she can learn a great deal without reading all 13 chapters because most of the chapters stand alone.

This monograph is not for the seasoned expert or experienced researcher. However, it is certainly an invaluable addition for medical students, psychologists, nurses, social workers, and enlightened laypeople who want to obtain a broad overview of the most current knowledge about drugs and behavior.

SIDNEY MALITZ, M.D.
New York, N.Y.

Current Developments in Psychopharmacology, Vol. 2, *edited by Walter B. Essman and L. Valzelli. New York, N.Y., Spectrum Publications (Halsted Press, John Wiley & Sons, distributor), 1976, 248 pp., $20.00.*

This volume is one of a series of annual publications consisting of invited contributions from distinguished scientists and clinicians actively working in different areas of psychopharmacology. Volume 2 has seven chapters, some of which are better and/or more interesting than others. One of the most informative and timely chapters is that of Kuhar, Simon, and Rommelspacher on the location, function, and pharmacology of central cholinergic neurons. This chapter is followed by two considerably less timely papers on the pharmacology and clinical use of flurothyl. Although the use of flurothyl in the treatment of psychiatric disorders as an alternative to ECT has been virtually abandoned by now, Adler states that flurothyl is still a useful investigative tool in the evaluation of compounds with potential anti-petit-mal efficacy, in studying the various processes involved in learning and memory, in quantitating cerebral excitability, and in exploring the biochemical and neurophysiological mechanisms underlying the seizure process.

A new general theory suggesting that amphetamine increases the repetition rate of all motor activities with the ultimate result of increasing response rates within a decreasing number of categories is put forward by Lyon and Robbins. If the new theory is correct, complex behavioral chains or behaviors requiring longer pauses, such as sleep, feeding, and certain types of social behavior, will be eliminated first under the influence of amphetamine; behaviors capable of repetition without long pauses will dominate for a transient period. Whether this new theory of amphetamine action offers any advantages over the existing ones remains to be seen.

There is a short and concise chapter on the relationship between the animal pharmacological profile and clinical effects of neuroleptic drugs. On the basis of a careful analysis of experimental data on 81 neuroleptic drugs, Janssen and Van Beuer suggest a relationship between the ED_{50} on the apomorphine antagonism test in dogs and the potency and/or duration of action of neuroleptic drugs. Similarly, the ratio of palpebral ptosis to catalepsy may serve as a useful index of relative sedative versus neurological effects, the ratio of norepinephrine antagonism to amphetamine antagonism as an index for relative adrenolytic activity versus antipsychotic activity, and the ratio of the jumping box test to the apomorphine antagonism test as an index for relative neurological versus antipsychotic activity. It would be of great practical significance if these observations could be supported by further evidence.

This volume also contains a long-overdue review of sensory psychopharmacology by Robinson and Sabat. The literature they review covers a period extending from 1958 to 1973, with an emphasis on major psychophysical findings and results of evoked response studies. The final chapter reviews some evidence indicating a relation of brain dopamine with depression and mania.

This is obviously not a book for the student but for the teacher, not for the practicing psychiatrist but for the research worker.

THOMAS A. BAN, M.D.
Nashville, Tenn.

The Effect of the Man-Made Environment on Health and Behavior, *edited by Lawrence E. Hinkle, Jr., M.D., and William C. Loring, Ph.D. Atlanta, Ga., U.S. DHEW Center for Disease Control, 1977, 315 pp., $3.25 (paper).*

Physicians have long maintained an interest in understanding environmental factors that affect health and disease. This book explores what is known about factors in the residential environment that might offer opportunities to reduce risk or promote health. It considers housing from the standpoints of physical design and social patterns.

The authors review a range of data and models and attempt to use Leighton's concept of community disintegration to understand the social distress of some large public housing projects. Kasl's chapter summarizes some of what is known about crowding, noise, size of domestic unit, division of residential space, control of residential space, proximity to a range of services, mass relocations, and demographic match between individuals and the local social context.

Much of this work is in such an early state that it provides only a suggestive basis for public policy decisions. The authors adduce evidence showing that a territory which functions as a neighborhood, with social attachments and organizations, yields an environment with a lower risk of accidents, violence, and disease than does the socially undefined space around some large housing projects. They suggest that certain factors of spatial arrangement can promote the development of affiliations and neighborhoods, but the situation is far from clear. Administrative actions that promote simultaneous mass migrations, or "relocations," appear to impede neighborhood development and can yield significant risks.

Cassel's review of the data concerning the risks of a range of physical diseases in various residential environments suggests a model involving individual affiliations, status hierarchies, degree of population adaptation to a particular context, and range of group support systems.

Hinkle's chapter suggests that some residential environments are able to shorten life, sicken it, damage babies, and interfere with the bonding process but that our knowledge is not sufficient to define environments that would reliably reduce such risks. Apparently a residential environment should be warm, commodious, quiet, private, peopled with kith and kin, and serviced with a range of amenities and assistances to daily activities. It should also be free of lead paint and accident hazards.

Damon's chapter tries to determine some limits. How noisy can the environment be before a hazard exists? How crowded can rooms be before damage is regular? It is not

clear whether true thresholds exist or whether the hazards act in a summative or interactive manner.

In *The Effect of the Man-Made Environment on Health and Behavior* Hinkle and Loring provide a helpful overview of an area of knowledge just barely opened. The tabulated data and exploratory models should be of interest to physicians and to those concerned with public housing policy.

NORRIS HANSELL, M.D.
Champaign, Ill.

Support Systems and Mutual Help: Multidisciplinary Explorations, *edited by Gerald Caplan and Marie Killilea. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1976, 315 pp., $19.50.*

This book, which consists of 13 chapters by 11 different authors, brings together ideas, research findings, and practice principles that relate to support systems and mutual help groups. As a whole, it serves as a reminder that competence and mental well-being can be increased by understanding and making use of the wide variety of helping systems that are in existence but are usually not included in program planning; in fact, their techniques are often ignored or spurned by professionals.

In the first chapter, titled "The Family As Support System," Gerald Caplan reminds us that the extended family is not so dead as we supposed, that the majority of older people either live with or very close to one of their children, and that more than 75% of people over 65 feel themselves to be in good health and can play an active role in public and private affairs. He goes on to give a clear and concise listing of the ways in which the family can help in the growth and development of its members. In the final section of his chapter Caplan pleads for suitable housing so that members of extended families can live close to each other; he also decries the underutilization of retired people.

The next several chapters could be titled collectively, "Everything You Always Wanted To Know About Mutual Help Organizations But Were Afraid To Ask." The second chapter is a review of the literature and cites 220 references. After a rather confusing beginning, it outlines the characteristics of mutual help groups, suggesting that the typologies range from the analytic on one end of the scale to the repressive-inspirational on the other. Another way of organizing the groups places those which provide crisis help in one category and those which aid the permanently stigmatized in another. A third group would be those which aid people trapped in some form of self-destructive behavior. This very comprehensive chapter reminds us that social movements attempt to bind people together and provide meaning to life as well as support. There is also a useful comparison between orthodox psychotherapy and self-help group therapy.

The next chapter, "Religious Aspects of Support Systems," suffers the usual fate of the conference report that is not very well edited: because the reader was not at the conference there are vital gaps that are not bridged by the written word. However, the chapter is interesting, with discussions ranging from the dependent immersion that takes place in Pentecostal groups to the religious ritual of the Academy Awards.

In "Self Help Treatment Programs," Dumont reminds mental health professionals that "unless we can accommodate to and find some common ground with this [self-help] movement, we will become increasingly cloistered, self-serving and irrelevant." In "Going Public and Self Help," Spiegel clearly articulates the dilemma of the stigmatized patient who alternates between being a victim and denying his or her handicap. The addition of the role of teacher for others with a similar handicap helps the individual come out with the message, "I am this, but I am more." This applies to gay people, paraplegics, and a host of other silent and stigmatized minorities.

Deathbed scenes through the ages are the subject of a well-written chapter by Ruth Caplan, followed by two chapters describing self-help organizations for middle-class people. Parents Without Partners provides support for many single parents, and Recovery, Inc., binds together, encourages, and inspires former mental patients. The description of this semireligious group is particularly interesting.

In his chapter, "Transition States," Weiss gives details of his Seminars for the Separated, which sound very useful. Chapters 11 and 12 discuss various aspects of a program for widows, including a valuable analysis of various styles of helping. The chapter by Richards on "Peer Consultation Among Clergy" reminds us of the marked stress felt by the modern-day clergy, who find themselves with few unique skills, waning status, and an occupational identity crisis. It describes the supportive interaction possible between dyads of bishops in the Episcopal Church and suggests that this model could be effectively reproduced between clergy at all levels.

The last chapter, largely derived from Caplan's experiences in Israel during the Yom Kippur War, discusses the organization of civilians in time of war or disaster. It is worth reading as soon as one overcomes the temptation to dismiss the content as irrelevant because "It couldn't happen here."

Overall, this is a worthwhile book for psychobiosocial caregivers who are secure enough to admit and embrace the power of the people as an effective force to improve community mental health.

N. MICHAEL MURPHY, M.D.
Albany, N.Y.

Psychopharmacology in the Practice of Medicine, *edited by Murray E. Jarvik, M.D., Ph.D. New York, N.Y., Appleton-Century-Crofts, 1977, 516 pp., $20.50.*

Some books are known in the trade as "how-to" books. This one should properly be considered a "what's-it" book. Unlike most multi-authored books on psychopharmacology, which abound these days, this one has been well planned as an appreciation of psychopharmacology. It provides a broad view of what psychopharmacology is concerned with, what some of its experimental techniques are, and what impact psychopharmacological knowledge has made on clinical problems—not a small ambition but one that has been realized in a most satisfactory way.

Thirty-five "minireview" chapters are grouped into four major sections. The initial section of 3 chapters is introductory; the second considers the experimental use of these drugs in 12 chapters; the third has 13 chapters devoted to various uses of psychotherapeutic drugs; and the fourth section consists of 7 chapters describing social drugs. Each section and each chapter is introduced by brief editorial comment pointing out the salient points to be covered and setting the context. A very short section of concluding editorial remarks completes the book. Each chapter reads very

well and hews to the main theme, no doubt reflecting excellent editing. Although the list of contributors includes many of the most accomplished researchers in the field, each has submerged his or her special interests to the cause of reviewing the entire field; the book is not a collection of individual papers. References are limited in number but provide an adequate base for expanded reading on any given subject.

This format serves admirably for examining the many aspects of the field. The price is that what is gained in breadth is lost in depth. This deficiency is most evident in the sections on clinical applications of psychotherapeutic drugs: some areas are covered far too sketchily. When a number of chapters are grouped together to cover a single topic, such as the 5 chapters concerned with affective disorders or the 2 concerned with opiate abuse, these subjects are given reasonably full treatment.

The most obvious use for this book is to form a basis for a course on psychopharmacology suitable for students or psychiatric residents. Each chapter could provide the advance reading for a more detailed discussion in lectures. The book could also well be read by those working in the field, simply to expand their appreciation of its diversity. Reading it fills one with a sense of awe for what has been learned during the past two or three decades as well as a sense of humility about what still needs to be known.

LEO E. HOLLISTER, M.D.
Palo Alto, Calif.

Computed Brain and Orbital Tomography: Technique and Interpretation, *by Carlos F. Gonzalez, M.D., Charles B. Grossman, M.D., and Enrique Palacios, M.D. New York, N.Y., John Wiley & Sons, 1976, 270 pp., $29.00.*

Computerized axial tomography has been hailed as the greatest advance in radiology since Roentgen's discovery of X rays. The technique uses an extremely small beam of X rays and replaces film with radiation detectors that measure the attenuated intensity of the beam as it passes through the skull and brain. The beam is moved to and fro as well as being rotated. A computer solves a large series of simultaneous equations to create a density value for each point in a two-dimensional matrix with anywhere from 160 to 320 points per side. When this is displayed on a cathode-ray oscilloscope, the picture is that of a horizontal section of the brain showing the ventricles, the sulci, and other anatomical structures. Pathological processes appear as areas of increased or decreased density. The use of intravenous contrast media will "enhance" certain lesions, increasing their apparent density. The process of scanning is carried out in several sections usually separated by 13 mm, thus allowing visualization of the brain from the posterior fossa to the vertex. The process has the advantage of low dosage of radiation and of being a noninvasive, completely safe procedure.

This new radiological procedure has progressed in its application so rapidly that it is difficult to keep up with its progress. A new journal has recently been organized dealing solely with this technology. This book is one of several that have appeared recently. It opens with a relatively brief section on basic physical principles, followed by a section on correlative anatomy and computerized transaxial (CT) scanning. The remainder of the volume is dedicated to changes in specific diseases, e.g., tumors, vascular disease, and trauma.

In the short time of its existence, CT technology has revolutionized approaches to neuroradiological diagnosis. Although it does not eliminate angiography or pneumoencephalopathy, it has greatly reduced the requirement for these procedures, both of which carry a measurable risk. It has also made it possible to literally look into the head without risk and to yield information not obtainable by any other radiological method.

This volume is well printed and contains a wealth of illustrations covering most of the common and some of the uncommon neurological disorders. Because of the rapid progress in this field, it may very well be outmoded in a short time, but for the moment it is an adequate statement of present capability.

DAVID D. DALY, M.D., PH.D.
Dallas, Tex.

The Right to Treatment for Mental Patients, *edited by Stuart Golann and William J. Fremouw. New York, N.Y., Irvington Publishers (Halsted Press, John Wiley & Sons, distributor), 1976, 239 pp., $16.95.*

In recent years the mental health establishment has witnessed an increasing intrusion on the part of insurance carriers, government agencies, and judicial actions. These intrusions have ostensibly been justified on the basis of upgrading and improving mental health care. Although the actions have generally been well meaning, they have often created additional problems and fallen far short of their original goals.

One of the recent controversies that has drawn attention is the right to treatment issue for involuntary patients. This issue was first clearly enunciated in the *Wyatt v. Stickney* decision (1–3) and later expanded in *Donaldson v. O'Connor* (4, 5). It is in this context that the Amherst Conference on Right to Treatment was convened; this book is the result of that conference.

The contributors to the book represent a variety of disciplines including psychology, psychiatry, sociology, and the law, indicating the far-reaching aspects of this legal question. The papers themselves deal with the historical, legal, social, and political perspectives and their impact on mental health delivery systems.

Of particular interest is a chapter by Stickney, the Commissioner of Mental Health of Alabama and defendant in *Wyatt v. Stickney*. He presents an enlightening view of the case itself and highlights the struggle of political and legal forces, with the mental health system a victim in the middle. This theme is expanded in Mechanic's essay, in which he notes the conflict between ideology and pragmatism in the situation. Although the courts might mandate changes, the funding might not be forthcoming; legislators might see a more economical way of dealing with fiscal dilemmas by abolishing state institutions and not providing aftercare arrangements. Mechanic's conclusion is that litigation has only short-range benefits, but social change is more valuable in the long run. Halpern raises a further issue of interest, namely, the care of the mentally retarded, who are often voluntary patients and are in need of basic rehabilitation services.

Overall, the reader comes away from this book with a greater understanding of the issues but probably with more questions than answers. One additional feature of the book is the inclusion of court decision source material on the significant cases for reference. This book informs well without being overwhelming regarding a topic of increasing importance.

REFERENCES

1. Wyatt v Stickney, 325 F Supp 781 (MD Ala 1971)
2. Wyatt v Stickney, 344 F Supp 373 (MD Ala 1972)
3. Wyatt v Aderholt, 503 F 2d 1305 (5th Cir 1974)
4. Donaldson v O'Connor, 493 F 2d 507 (5th Cir 1974)
5. O'Connor v Donaldson, 43 USLW 4929 (1975)

THOMAS C. BOND, M.D.
Belmont, Mass.

Origins of Concepts in Human Behavior: Social and Cultural Factors, *by Mark D. Altschule. Washington, D.C., Hemisphere Publishing Corp. (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1977, 200 pp., no price listed.*

This collection of essays on the most diverse of subjects is drawn together by the interest and sensibility of Professor Altschule. The papers are glittering nuggets gleaned from his reading in the history of medicine over some 20 years. His aim in this collection is to illustrate the ineluctable interplay between medical ideas and *Zeitgeist*. Here, in "The Pneuma Concept of the Soul," he traces the peregrination of the idea of soul=breath through most of the major cultural systems of the world. Another variation of this theme, that of ethnicity and mental disease, is explored in the case of "George Cheyne and His English Malady." Derived largely from his own experience, Cheyne's description of hypochondriacal depression fell on receptive ears in 1733, crystallizing some sentiments about Englishmen general to the time (i.e., that they were self-destructive, moody, and predisposed to insanity). Altschule shows how these notions persisted well into the nineteenth century despite the lack of evidence and the irritation expressed by some English medical writers.

In "Huron Ideas About the Unconscious Mind" and "The Early History of Psychiatric Treatment," Altschule delights in the demonstration of J. L. Borges' thesis, namely, that all great ideas (e.g., Freud's dream theory) create their own precursors. Here are the titles of some other chapters to give a further idea of Altschule's range: "Acupuncture in the Western World Up to a Century Ago," "The Singular Case of James Tilly Matthews, A Clear Paranoid (Whose 19th-Century Mechanical Delusions Are Those of a Newton Gone Mad!)," "Acedia—Its Evolution from Deadly Sin to Psychiatric Syndrome."

Altschule's style is scholarly but not pedantic. There are moments when he is more bookish than literary, but these are few and far between. Furthermore, Altschule possesses a sharp wit that he unlimbers on occasion. I think this volume will be of interest to those who pursue the history of ideas or who seek to broaden their perspective on current clinical phenomena. I wish this book into the hands of those who usually do not read "that sort of thing." Not only is it useful reading, it is enjoyable as well.

JOHN R. NEILL, M.D.
Lexington, Ky.

Dialogues for Therapists: Dynamics of Learning and Supervision, *by Margaret J. Rioch, Winifred R. Coulter, and David M. Weinberger. San Francisco, Calif., Jossey-Bass, 1976, 233 pp., $12.95.*

Margaret Rioch is a distinguished psychologist well known for her pioneering work in the field of group relations. A practicing psychotherapist, she has drawn from personal knowledge of such teachers as Harry Stack Sullivan, Frieda Fromm-Reichmann, Edith Weigert, Martin Buber, and Zen Buddhist scholars Daisetz T. Suzuki and Hubert Benoit. As leader of a seminar for beginning psychotherapists, she has achieved a creative union between her expertise in psychotherapy and her knowledge of group theory. She and her colleagues Winifred Coulter and David Weinberger have compiled this book detailing the most meaningful elements of a series of her seminars, including comments on the ongoing process between Dr. Rioch and her students as the seminars developed. The result is a valuable addition to our knowledge of psychotherapy and how it might be taught.

The book is organized into dialogues between instructor and students centering on critical elements in the therapeutic process as exemplified in case presentations. Subjects such as initial contracts, transference and countertransference, therapeutic strategies, and termination are discussed. This familiar material is refreshed by Dr. Rioch's unbiased approach—she seems as comfortable in behavioral theory as in analytic—and by her steady insistence that the underlying feelings and process between both participants in the therapy relationship must be examined. These dialogues go beyond the therapist's feelings in his or her relationship with the patient to include the group process generated within the seminar.

Rioch's point is that "in order to be 'fully present,' to use Martin Buber's term, it is necessary to clear away the fears and preoccupations, the blind spots and the prejudices, with which beginning therapists, as well as more experienced ones, are heavily burdened. . . . We have to wipe the mirror clean before we can see what is reflected." She believes that the seminar's process, including the students' relationship with her, will inevitably mirror the difficulties and successes of the therapeutic work under discussion. What is especially unique here is that because of the dialogue form we can actually see the process through which the students learn to work with the complex interrelationship between their therapeutic work and the group supervision, between their patients' feelings and attitudes toward them and their own feelings and attitudes toward their seminar colleagues and Dr. Rioch. The art of a skilled and experienced teacher is palpable as each episode unfolds. Her methods are gentle yet incisive, a model for clinical supervisors in any setting.

Dialogues for Therapists is an excellent volume for beginning therapists; many of the cases represent classic difficulties in the therapeutic encounter. Additionally, teachers of psychotherapists may want to try Dr. Rioch's group model in their own supervisory work. Those who already are involved in group supervision will benefit from seeing how an expert in group process can use this knowledge to enhance the learning opportunities in a seminar setting. Of particular note are two remarkably frank evaluative chapters, one on supervision failures and the other on the "antitherapeutic" aspects of the seminar itself. Both of these demonstrate how competition, jealousies, and anger can be magnified by the group context and how unspoken negative fantasies about the instructor can limit creativity and support "no-risk" learning. As one student commented when the seminar members were briefly contemplating how much they were influenced and inhibited by Dr. Rioch's presence, "The real problem is not how nice it would be *without* her, but how to live *with* her. And not only with her, with all the other authorities, too."

Perhaps the most valuable aspect of the book for me was

the opportunity it afforded to learn more about what this very wise woman understands to be the essential elements in the therapeutic process. The book is full of statements that go beyond her immediate task in the seminar yet provide its best teaching. For example, in the course of talking about specific goals of therapy she says, "In the back of my head, I keep the idea that if he [the patient] gets over the few hurdles in the interpersonal world, he may go on to something much better, namely, a lack of concern for himself altogether." The experienced therapist may find himself or herself scanning the more elementary and task-oriented parts of this book for more of these gems. This is at once the strength and weakness of this fine volume.

ARTHUR D. COLMAN, M.D.
Sausalito, Calif.

Alcoholism Problems in Women and Children, *edited by Milton Greenblatt, M.D., and Marc A. Schuckit, M.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1976, 274 pp., $19.50.*

This book is another excellent choice in the series of Seminars in Psychiatry. The book is divided into two sections, the first dealing with alcoholism in women and the second with adolescent drinking problems.

Beginning with a general overview of alcoholism in women, the chapters in the first section explore in detail the problems, theories, and treatment of alcoholic women. In contrast to the traditional view that many alcoholic people drink to gratify passive-oral dependency needs, Wilsnack presents her theory that conflicts and tensions related to sex roles contribute to drinking problems among women today. Whereas some men drink to satisfy their need for power, some women drink to increase feelings of traditional femininity or womanliness.

Beckman presents an excellent description of the social and psychological aspects of alcoholism and the influence of the spouse and mother on the alcoholic picture, providing negative evidence on the old concept of the "alcoholic personality." The authors of the following two chapters discuss theories concerning the biological aspects of alcoholism as they pertain to women. A few studies point to the stress of the menstrual cycle as a possible precipitating factor for alcoholism in certain women. On the basis of studies indicating changes in alcohol metabolism associated with different phases of the menstrual cycle, the authors hypothesize a relationship between alcohol and depression via changes in MAO levels during the menstrual cycle. The first part of the book ends with a short presentation on lesbianism and alcoholism and a discussion of treatment programs for alcoholic women.

The second part of the book starts with a general review of the extent of drinking problems among adolescents. The authors explore the social, interpersonal, and psychological factors involved in teenage drinking. They make the observation that if an individual is ever going to drink, he or she will likely begin to do so before graduation from high school. They also observe that girls in the 15–18-year-old group are changing in terms of the number who drink, type of beverage consumed, frequency of drinking, and drinking to intoxication. In fact, the female adolescent now surpasses her male counterpart on some variables.

A chapter dealing with parental influences on the drinking patterns of their children presents a model assuming a multilevel process of parent effects, including socioeconomic, sociocultural, family interaction, and the individual parent's behavior. This is followed by a short review by Winokur of several recent genetic studies providing evidence for a genetic causative factor in alcoholism and no evidence to indicate that rearing is important.

The last chapter is perhaps the most interesting. It discusses fully the fetal alcohol syndrome and the seriousness of alcoholism in pregnancy. Several studies and observations made during the past several years provide strong evidence that a congenital syndrome of abnormalities can result from heavy alcohol intake by pregnant women. The syndrome may include the following: short birth length and low birth weight, continuing failure to thrive, craniofacial abnormalities, joint and limb anomalies, cardiac defects, mental deficiency, and delayed motor development. The author emphasizes that because of these characteristic facets, diagnosis is relatively easy but very important because it clarifies etiology and provides the opportunity for recommending against subsequent pregnancies for alcoholic women who continue to drink heavily.

In general this is an excellent review of recent and important contributions in the field of alcoholism. It should be read by those who work in the field.

SEBASTIAN MOZZICATO, M.D.
Farmington, Conn.

Developmental Disabilities: Psychologic and Social Implications, *edited by Daniel Bergsma, M.D., and Ann E. Pulver, M.H.S. New York, N.Y., Alan R. Liss, 1976, 188 pp., $18.00.*

This book presents the proceedings of a conference of the National Foundation-March of Dimes sponsored by the Johns Hopkins Medical Institutions in Baltimore, Md., March 1–2, 1976. The 16 papers are organized into four sections: Medical Aspects, Pre-Adult Disability, Individual and Family Needs, and Societal Reaction. The papers are generally of high quality, timely, and well written. However, the authors have some difficulty in properly classifying the papers into the organizational framework, and there is some redundancy.

The overall theme of the conference was the significance of social factors in the understanding of developmental disability and in planning for service delivery. This is perhaps best expressed by Leon Eisenberg, who draws the distinction between illness and disease, i.e., between organic pathology and the reaction of the patient and society to the patient's illness.

Individual papers include an epidemiological study in British Columbia by James R. Miller; a review of the literature dealing with the reactions of the nondisabled toward those who are by Stephen A. Richardson; a review of the British literature dealing with the reactions of parents to the handicapped child by Sheila Hewett; a survey of physical, intellectual, psychological, and social problems in the United Kingdom by Elizabeth M. Anderson; personal experience with the problems of adolescence in the physically disabled by Thomas E. Strax; the roles of the family and health professionals in the decision-making process by Raymond S. Duff; an analysis of the obstacles to effective supporting psychological services by Albert J. Solnit; a review of health care received by children with developmental disorders and their families by Ivan B. Pless; a history of the medical treat-

ment of the orthopedically handicapped by Saul Benson; an attack on current practices of testing and education by Edmund W. Gordon; alternative forms of health care by Robert Morris; the issues in three lawsuits dealing with the rights of the handicapped by Kathleen G. Ursin; the use and misuse of scientific knowledge in shaping public policy by Elizabeth M. Boggs; recommendations for architectural redesign of environments by Sandra C. Howell; recommendations for public education by Lowell S. Levin; and a discussion of the value of epidemiological studies in designing and evaluating health services for the handicapped by Leon Eisenberg.

This volume will be most valuable to the general physician and health care program administrator. It stresses the importance of social factors and personal relationships with handicapped clients in evaluating and treating the full ravages of their disability. However, the book is long on broad ethical, moral, and theoretical issues and short on immediate, concrete program recommendations for the social engineering required to deal with problems of the handicapped.

GERALD R. CLARK, M.D.
Elwyn, Pa.

A Primer on Chemical Dependency: A Clinical Guide to Alcohol and Drug Problems, *by Joseph Westermeyer, M.D., M.A., M.P.H., Ph.D. Baltimore, Md., Williams & Wilkins Co., 1976, 225 pp., $11.95 (paper).*

In his introduction Westermeyer informs us that this book is intended for "the medical or surgical practitioner, whether generalist or specialist . . . [as well as] your spouse, parent, child, or friend, . . . dependent patients and their families, . . . other workers in the health field—nurses, psychologists, medical social workers, . . . [students in these fields, and] law enforcement officials, clergy, counselors, or sociologists." All things to all men? Hardly. Unfortunately, this attempt weakens several potentially useful books. Focus of both subject matter and audience is lacking; the result is simplification, at times to the point of distortion.

What is striking to me is that hidden within the text is the basis of an excellent simple introduction to the problems of alcoholism. On the basis of an examination of the references, I believe this was the author's original intention. For example, of the 19 references in the chapter titled "Chemical Dependency Syndrome," only two deal with drugs other than alcohol. Similar if less extreme ratios are seen in the references for other chapters. In addition, the author seems most sure when discussing alcohol. He scants the complex pharmacological-personality-social relationships of other drugs and the issue of use as distinct from abuse. An attempt at a unified theory of drug abuse, subsumed under the rubric "chemical dependency," is premature, particularly if it is offered in an introductory text.

WILLIAM A. FROSCH, M.D.
New York, N.Y.

Alcoholism and the Brain, *by A.E. Bennett, M.D. New York, N.Y., Stratton Intercontinental Medical Book Corp., 1977, 86 pp., $9.75.*

The author of this book is the respected director of the neuropsychiatric foundation that bears his name; he has enjoyed a long career in neurology and psychiatry. This slender volume is directed mainly toward physicians with the expressed hope of increasing their understanding of the impact of heavy chronic doses of alcohol on the brain and to balance what the author feels is an overemphasis on psychological and social circumstances in the field of alcoholism.

Part of the book reviews many of the ancient statements made by psychiatrists and researchers. Many authors in the field of alcoholism (myself included) feel compelled to engage in this ritual. Since the same names and statements pop up again and again, we should question whether those of us who write about this subject do not ourselves suffer from repetitive compulsivity that is often a part of the problem in alcoholism.

Dr. Bennett's main thesis is that it is impossible to treat alcoholic people successfully unless one first treats their physical addiction by medical means. He allows that once this is accomplished, psychological and social therapies are appropriate. This book is thus an accumulation of old material that describes in detail the acute and chronic effects of alcohol on the brain. I designate the material as old because those of us who have followed Dr. Bennett's career recognize that he does not include the more recent studies on the effects of alcohol on the brain. Although this book's appeal is to a limited audience, many of us need to be reminded that along with its psychological and social ramifications, alcohol abuse definitely affects the brain and the nervous system.

MORRIS E. CHAFETZ, M.D.
Washington, D.C.

Theories of Group Processes, *edited by Cary L. Cooper. New York, N.Y., John Wiley & Sons, 1976, 272 pp., $9.95 (paper).*

This book is an academic study of T groups. It focuses on these groups in community organizations and business but does not say much about group psychotherapy. There are 10 chapters and 14 authors, and the goals and quality of the articles are inconsistent. The extensive bibliography after each chapter will acquaint the psychiatrist with some of the extensive literature by nonpsychiatrists.

This book is a must for the serious group therapy researcher and for serious students of group therapy. The practitioner and casual student of group therapy will find a few interesting and helpful ideas; they will find the book is worth a quick reading.

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Meditational Therapy, *edited by Swami Ajaya. Glenview, Ill., Himalayan International Institute of Yoga Science and Philosophy, 1977, 92 pp., $2.95 (paper).*

Homosexuality: A Symbolic Confusion, *by Ruth Tiffany Barnhouse. New York, N.Y., Seabury Press, 1977, 188 pp., $8.95.*

Juvenile Victimization: The Institutional Paradox, *by Clemens Bartollas, Stuart J. Miller, and Simon Dinitz. New York, N.Y., Sage Publications (Halsted Press, John Wiley & Sons, distributor), 1976, 316 pp., $17.50.*

Having It Your Way: The Strategy of Settling Everyday Conflicts, *by Robert Bell. New York, N.Y., W.W. Norton & Co., 1977, 189 pp., $8.95.*

Behavior Modification: An Introductory Textbook, *by Alan S. Bellak, Ph.D., and Michel Hersen, Ph.D. Baltimore, Md., Williams & Wilkins Co. (New York, N.Y., Oxford University Press, publisher), 1977, 361 pp., $14.95.*

The Guilty Book, *by Barry R. Berkey, M.D., and Velma A. Berkey; illustrated by Rita Malone. Minneapolis, Minn., T.S. Denison & Co., 1977, 44 pp., $4.95.*

Psychiatric Terror: How Soviet Psychiatry Is Used to Suppress Dissent, *by Sidney Bloch and Peter Reddaway. New York, N.Y., Basic Books, 1977, 497 pp., $12.95.*

Assessing Personality Through Tree Drawings, *by Karen Bolander. New York, N.Y., Basic Books, 1977, 412 pp., $20.00.*

Why Survive? Being Old in America, *by Robert N. Butler, M.D. New York, N.Y., Harper & Row, 1977, 496 pp., $5.95 (paper).*

A Structural Theory of the Emotions. Psychological Issues Monograph 40, *by Joseph De Rivera. New York, N.Y., International Universities Press, 1977, 169 pp., $12.50.*

Child Development: Analysis and Synthesis, *by Joseph H. Di Leo, M.D. New York, N.Y., Brunner/Mazel, 1977, 170 pp., $13.50.*

Alcoholism: Development, Consequences, and Interventions, *edited by Nada J. Estes, R.N., M.S., and M. Edith Heinemann, R.N., M.A. St. Louis, Mo., C.V. Mosby Co., 1977, 318 pp., $9.25 (paper).*

Child Abuse and Neglect Case Studies: 50 Case Histories Related to Child Abuse and Neglect, *by Abe R. Fosson, M.D., and H. Otto Kaak, M.D. Flushing, N.Y., Medical Examination Publishing Co., 1977, 192 pp., $10.00 (spiral bound).*

Alternatives to Psychiatric Hospitalization, *by Harry Gottesfeld, Ph.D. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributors), 1977, 128 pp., $11.95.*

The Art and Technique of Analytic Group Therapy, *by Martin Grotjahn, M.D. New York, N.Y., Jason Aronson, 1977, 276 pp., $20.00.*

Borderline Personality Disorders: The Concept, the Syndrome, the Patient, *edited by Peter Hartocollis, M.D., Ph.D. New York, N.Y., International Universities Press, 1977, 517 pp., $22.50.*

Maturity and Competence: A Transcultural View, *by Douglas H. Heath. New York, N.Y., Gardner Press (Halsted Press, John Wiley & Sons, distributor), 1977, 281 pp., no price listed.*

Psychotherapy in Chronic Ulcerative Colitis, *by Aaron Karush, M.D., George E. Daniels, M.D., Charles Flood, M.D., and John F. O'Connor, M.D. Philadelphia, Pa., W.B. Saunders Co., 1977, 140 pp., $13.75.*

Behind the Wall of Respect: Community Experiments in Heroin Addiction Control, *by Patrick H. Hughes, M.D. Chicago, Ill., University of Chicago Press, 1977, 151 pp., $10.95.*

Therapeutic Discourse: Psychotherapy as Conversation, *by William Labov and David Fanshel. New York, N.Y., Academic Press (Harcourt Brace Jovanovich), 1977, 377 pp., $15.00.*

Écrits: A Selection, *by Jacques Lacan; translated by Alan Sheridan. New York, N.Y., W.W. Norton & Co., 1977, 335 pp., $16.75.*

A Concise Encyclopaedia of Psychiatry, *edited by Denis Leigh, M.D., B.Sc., C.M.B. Pare, M.D., D.P.M., and John Marks, M.A., M.D. Baltimore, Md., University Park Press, 1977, 399 pp., $18.50.*

The Chase: Career of the Compulsive Gambler, *by Henry R. Leisieur. Garden City, N.Y., Anchor Books (Anchor Press/Doubleday), 1977, 285 pp., $2.95 (paper).*

The Dynamics of University Protest, *by Donald Light, Jr., and John Spiegel, with C.J. Lammers, R.S. Laufer, M. Levin, and J.L. Norr. Chicago, Ill., Nelson-Hall, 1977, 198 pp., $12.00.*

Renewal in Psychiatry: A Critical Rational Perspective, *edited by Theo C. Manschreck and Arthur M. Kleinman. Washington, D.C., Hemisphere Publishing Corp. (New York, N.Y., Halstead Press, John Wiley & Sons, distributor), 1977, 326 pp., $23.50.*

Ethical Issues in Sex Therapy and Research, *edited by William H. Masters, M.D., Virginia E. Johnson, and Robert C. Kolodny, M.D. Boston, Mass., Little, Brown and Co., 1977, 219 pp., $12.50.*

The Self: Psychological and Philosphical Issues, *edited by Theodore Mischel. Totowa, N.J., Rowman and Littlefield, 1977, 348 pp., $17.50.*

A Manual of Death Education and Simple Burial, 8th ed., *by Ernest Morgan. Burnsville, N.C., Celo Press, 1977, 64 pp., $2.00 (paper).*

Findings of Drug Abuse Research: An Annotated Bibliography of NIMH- and NIDA-Supported Extramural Grant Research, 1967–1974, Vols. 1 and 2. NIDA Research Monograph 1. *Rockville, Md., National Institute on Drug Abuse, 1975; 384 pp. (Vol. 1), 377 pp. (Vol. 2); $7.00 (Vol. 1), $5.05 (Vol. 2).*

Operational Definitions in Socio-Behavioral Drug Use Research 1975. NIDA Research Monograph 2, *edited by Jack Elinson and David Nurco. Rockville, Md., National Institute on Drug Abuse, 1975, 163 pp., $2.50.*

Aminergic Hypotheses of Behavior: Reality or Cliché? NIDA Research Monograph 6, *by Jay R. Williams, Ph.D. Rockville, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1975, 148 pp., $2.25.*

Narcotic Antagonists: The Search for Long-Acting Preparations. NIDA Research Monograph 4, *edited by Robert Willette, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1976, 45 pp., $1.10.*

Young Men and Drugs—A Nationwide Survey. NIDA Research Monograph 5, *by John A. O'Donnell, Ph.D., Harwin L. Voss, Ph.D., Richard R. Clayton, Ph.D., Gerald T. Slatin, Ph.D., and Robin G.W. Room, M.A. Rockville, Md., National Institute on Drug Abuse, 1976, 141 pp., $2.25.*

Effects of Labeling the "Drug-Abuser": An Inquiry. NIDA Research Monograph 6, *by Jay R. Williams, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1976, 39 pp., $1.05.*

Cannabinoid Assays in Humans. NIDA Research Monograph 7, *edited by Robert E. Willette, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1976, 119 pp., $1.95.*

Rx: 3x/Week LAAM. Alternative to Methadone. NIDA Research Monograph 8, *edited by Jack D. Blaine, M.D., and Pierre F. Renault, M.D. Rockville, Md., National Institute on Drug Abuse, 1976, 126 pp., $6.00.*

Narcotic Antagonists: Naltrexone. Progress Report. NIDA Research Monograph 9, *edited by Demetrios Julius, M.D., and Pierre Renault, M.D. Rockville, Md., National Institute on Drug Abuse, 1976, 178 pp., $2.55.*

The Epidemiology of Drug Abuse: Current Issues. NIDA Research Monograph 10, *edited by Louise G. Richards, Ph.D., and Louise B. Blevens. Rockville, Md., National Institute on Drug Abuse, 1977, 257 pp., $2.60.*

Drugs and Driving. NIDA Research Monograph 11, *edited by Robert E. Willette, Ph.D. Rockville, Md., National Institute on Drug Abuse, 1977, 135 pp., $1.70.*

Cocaine: 1977. NIDA Research Monograph 13, *edited by Robert C. Petersen, Ph.D., and Richard C. Stillman, M.D. Rockville, Md., National Institute on Drug Abuse, 1977, 210 pp., $3.00.*

Psychiatry: Pretest Self-Assessment and Review, *edited by J. Craig Nelson, M.D. Wallingford, Conn., Pretest Service, 1977, 196 pp., no price listed (paper).*

Psychiatric Diagnosis. The C.M. Hincks Memorial Lectures, *edited by Vivian M. Rakoff, M.A., M.B.B.S., Harvey C. Stancer, Ph.D., M.D., and Henry B. Kedward, M.A., M.D. New York, N.Y., Brunner/Mazel, 1977, 228 pp., $15.00.*

Patients and Their Hospitals, 3rd ed., *by Winifred Raphael, B.Sc. London, England, King Edward's Hospital Fund for London, 1977, 44 pp., £3.75 (paper).*

Psychiatric Hospitals Viewed by Their Patients, 2nd ed., *by Winifred Raphael, B.Sc. London, England, King Edward's Hospital Fund for London, 1977, 48 pp., £3.75 (paper).*

Pathway to Piaget: A Guide for Clinicians, Educators, and Developmentalists, *by Hugh Rosen. Cherry Hill, N.J., Postgraduate International, 1977, 300 pp., no price listed.*

The Neuropsychopathology of Written Language, *by Joseph H. Rosenthal, M.D. Chicago, Ill., Nelson-Hall, 1977, 157 pp., $12.95.*

Psychotherapy Versus Iatrogeny: A Confrontation for Physicians, *by Nikola Schipkowensky. Detroit, Mich., Wayne State University Press, 1977, 494 pp., $30.00.*

Psychoanalysis and Contemporary Science: An Annual of Integrative and Interdisciplinary Studies, Vol. 5, 1976, *edited by Theodore Shapiro. New York, N.Y., International Universities Press, 1977, 624 pp., $20.00.*

Basic Rehabilitation Techniques: A Self-Instructional Guide, *edited by Robert D. Sine, M.D., Shelly E. Liss, M.D., Robert E. Rousch, Ed.D., and J. David Holcomb, Ed.D. Germantown, Md., Aspen Systems Corp., 1977, 218 pp., $12.95 (paper).*

Partners in Play: A Step-by-Step Guide to Imaginative Play in Children, *by Dorothy G. Singer and Jerome L. Singer. New York, N.Y., Harper & Row, 1977, 197 pp., $10.95.*

Klemer's Counseling in Marital and Sexual Problems: A Clinician's Handbook, 2nd ed., *edited by Robert E. Stahmann, Ph.D., and William J. Hiebert, S.T.M. Baltimore, Md., Williams & Wilkins Co., 1977, 370 pp., $16.95 (paper).*

The Children's Cause, *by Gilbert Y. Steiner, with the assistance of Pauline H. Milius. Washington, D.C., Brookings Institution, 1976, 255 pp., $9.95; $3.95 (paper).*

A History of Aggression in Freud. Psychological Issues Monograph 39, *by Paul E. Stepansky. New York, N.Y., International Universities Press, 1977, 196 pp., $12.50.*

Psychoanalysis and Family Therapy: Selected Papers, *by Helm Stierlin, M.D., Ph.D. New York, N.Y., Jason Aronson, 1977, 342 pp., $15.00.*

Ethical Policy in Mental Health Care: The Goals of Psychiatric Intervention, *by Laurence R. Tancredi, M.D., J.D., and Andrew E. Slaby, M.D., M.P.H. New York, N.Y., PRODIST/ William Heinemann Medical Books, 1977, 153 pp., $10.00.*

Toward Intimacy: Family Planning and Sexuality Concerns of Physically Disabled Women, *by the Task Force on Concerns of Physically Disabled Women. Everett, Wash., Planned Parenthood of Snohomish County, 1977, 62 pp., $1.50 for any 4 booklets (paper).*

Within Reach: Providing Family Planning Services to Physically Disabled Women, *by the Task Force on Concerns of Physically Disabled Women. Everett, Wash., Planned Parenthood of Snohomish County, 1977, 48 pp., $1.50 for any 4 booklets (paper).*

Biology of Play. Clinics in Developmental Medicine 62, *edited by Barbara Tizard and David Harvey. London, England, William Heinemann Medical Books; Philadelphia, Pa. J.B. Lippincott Co., 1977, 208 pp., $19.75.*

Homosexuality Re-Examined, *by D.J. West. Minneapolis, Minn., University of Minnesota Press, 1977, 348 pp., $20.00.*

Alternate States of Consciousness, *edited by Norman E. Zinberg. New York, N.Y., Free Press (Macmillan Publishing Co.), 1977, 286 pp., $14.95.*

The *Journal* has also received a set of cassettes in a vinyl folder:
Current Perspectives in Psychosomatic Medicine, *edited by Robert D. Martin, M.D. New York, N.Y., BioMonitoring Applications, 1977, $55.00.*The set includes the following: **Introduction to Psychosomatic Medicine,** *by Robert D. Martin, M.D., $9.95;* **Autonomic Nervous System and Psychophysiology,** *by Richard A. Sternbach, Ph.D., $9.95;* **Psychiatric Aspects of Endocrine Disorders,** *by Lorrin M. Koran, M.D., $9.95;* **Life Stress Measurement and Illness: Diagnosis and Treatment,** *by R.H. Rahe, M.D., $9.95;* **Clinical Issues in Psychosomatic Medicine,** *by Joseph D. Sapira, M.D., $9.95;* and **Perspectives in Current Psychosomatic Medicine,** *by Z.J. Lipowski, M.D., $9.95.* The cassettes are accompanied by selected bibliographies for each topic.

The American Board of Psychiatry and Neurology

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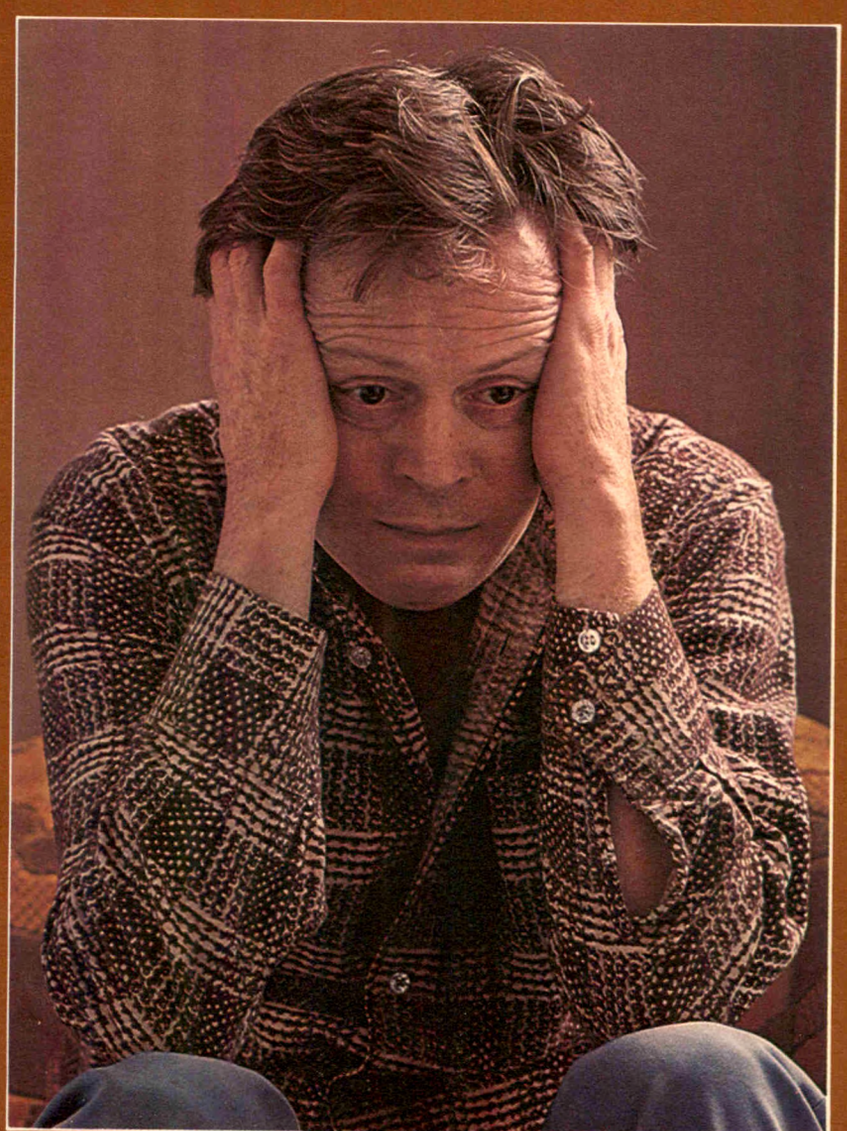
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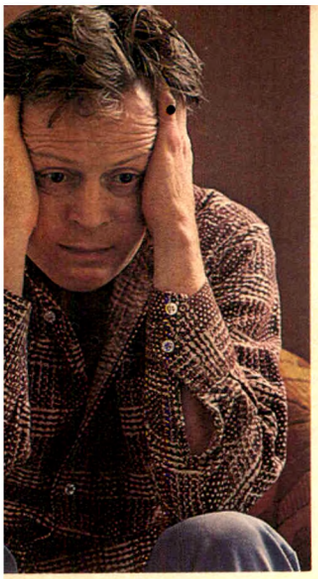
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For a brief summary of prescribing information, please see following page.



for moderate to severe anxiety or agitation and depression

DUAL-ACTION Triavil®

containing perphenazine and amitriptyline HC

Available:
TRIAVIL® 2-25: Each tablet contains
2 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 2-10: Each tablet contains
2 mg perphenazine and 10 mg amitriptyline HCl.
TRIAVIL® 4-25: Each tablet contains
4 mg perphenazine and 25 mg amitriptyline HCl.
TRIAVIL® 4-10: Each tablet contains
4 mg perphenazine and 10 mg amitriptyline HCl.

INITIAL THERAPY FOR MANY PATIENTS
TRIAVIL® 2-25 (or TRIAVIL® 4-25) t.i.d. or q.i.d.

FOR FLEXIBILITY IN ADJUSTING MAINTENANCE THERAPY
TRIAVIL® 2-10 (or TRIAVIL® 4-10)

CONTRAINDICATIONS: Central nervous system depression from drugs (barbiturates, alcohol, narcotics, analgesics, antihistamines); evidence of bone marrow depression; known hypersensitivity to phenothiazines or amitriptyline. Should not be given concomitantly with a monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred from such combinations. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with TRIAVIL. Therapy should then be initiated cautiously with gradual increase in dosage until optimum response is achieved. Not recommended for use during acute recovery phase following myocardial infarction.

WARNINGS: TRIAVIL should not be given concomitantly with guanethidine or similarly acting compounds since TRIAVIL may block the antihypertensive action of such compounds. Use cautiously in patients with history of urinary retention, angle-closure glaucoma, increased intraocular pressure, or convulsive disorders. Dosage of anticonvulsive agents may have to be increased. In patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely. Tricyclic antidepressants, including amitriptyline HCl, have been reported to produce arrhythmias, sinus tachycardia, and prolongation of conduction time, particularly in high doses. Myocardial infarction and stroke have been reported with tricyclic antidepressant drugs. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Not recommended in children or during pregnancy.

PRECAUTIONS: Suicide is a possibility in depressed patients and may remain until significant remission occurs. Such patients should not have access to large quantities of this drug.

Perphenazine: Should not be used indiscriminately. Use with caution in patients who have previously exhibited severe adverse reactions to other phenothiazines. Likelihood of some untoward actions is greater with high doses. Closely supervise with any dosage. The antiemetic effect of perphenazine may obscure signs of toxicity due to overdosage of other drugs or make more difficult the diagnosis of disorders such as brain tumor or intestinal obstruction. A significant, not otherwise explained, rise in body temperature may suggest individual intolerance to perphenazine, in which case discontinue.

If hypotension develops, epinephrine should not be employed, as its action is blocked and partially reversed by perphenazine. Phenothiazines may potentiate the action of central nervous system depressants (opiates, analgesics, antihistamines, barbiturates, alcohol) and atropine. In concurrent therapy with any of these, TRIAVIL should be given in reduced dosage. May also potentiate the action of heat and phosphorous insecticides.

Amitriptyline: In manic-depressive psychosis, depressed patients may experience a shift toward the manic phase if they are treated with an antidepressant. Patients with paranoid symptomatology may have an exaggeration of such symptoms. The tranquilizing effect of TRIAVIL seems to reduce the likelihood of this effect. When amitriptyline HCl is given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required. Paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs.

Caution is advised if patients receive large doses of ethchlorvynol concurrently. Transient delirium has been reported in patients who were treated with 1 g of ethchlorvynol and 75-150 mg of amitriptyline HCl.

Amitriptyline HCl may enhance the response to alcohol and the effects of barbiturates and other CNS depressants.

Concurrent administration of amitriptyline HCl and electroshock therapy may increase the hazards associated with such therapy. Such treatment should be limited to patients for whom it is essential. Discontinue several days before elective surgery if possible. Elevation and lowering of blood sugar levels have both been reported. Use with caution in patients with impaired liver function.

ADVERSE REACTIONS: Similar to those reported with either constituent alone.

Perphenazine: Extrapyramidal symptoms (opisthotonus, oculogyric hyperreflexia, dystonia, akathisia, acute dyskinesia, ataxia, parkinso have been reported and can usually be controlled by the concomitant effective antiparkinsonian drugs and/or by reduction in dosage, but times persist after discontinuation of the phenothiazine.

Tardive dyskinesia may appear in some patients on long-term thera may occur after drug therapy with phenothiazines and related agen been discontinued. The risk appears to be greater in elderly patients or dose therapy, especially females. Symptoms are persistent and in patients appear to be irreversible. The syndrome is characterized by mical involuntary movements of the tongue, face, mouth, or jaw. In tary movements of the extremities sometimes occur. There is no k treatment for tardive dyskinesia; antiparkinsonism agents usually do n viate the symptoms. It is advised that all antipsychotic agents be disc ued if the above symptoms appear. If treatment is reinstituted, or dos the particular drug increased, or another drug substituted, the syndrom be masked. Fine vermicular movements of the tongue may be an earl of the syndrome. The full-blown syndrome may not develop if medica stopped when lingual vermiculation appears.

Other side effects are skin disorders (photosensitivity, itching, ery urticaria, eczema, up to exfoliative dermatitis); other allergic rea (asthma, laryngeal edema, angioneurotic edema, anaphylactoid reac peripheral edema; reversed epinephrine effect; hyperglycemia; end disturbances (lactation, galactorrhea, gynecomastia, disturbances o strual cycle); altered cerebrospinal fluid proteins; paradoxical excit hypertension, hypotension, tachycardia, and ECG abnormalities (qui like effect); reactivation of psychotic processes; catatonic-like states nomic reactions, such as dry mouth or salivation, headache, an nausea, vomiting, constipation, obstipation, urinary frequency or tinence, blurred vision, nasal congestion, and a change in pulse rate; adverse reactions reported with various phenothiazine compounds, b with perphenazine, include grand mal convulsions, cerebral edema phagia, pigmentary retinopathy, photophobia, skin pigmentation, and of ejaculation.

The phenothiazine compounds have produced blood dyscrasias topenia, thrombocytopenic purpura, leukopenia, agranulocytosis, eosi lia); and liver damage (jaundice, biliary stasis).

Pigmentation of the cornea and lens has been reported to occur afte term administration of some phenothiazines. Although it has not reported in patients receiving TRIAVIL, the possibility that it might should be considered.

Hypnotic effects, lassitude, muscle weakness, and mild insomnia also been reported.

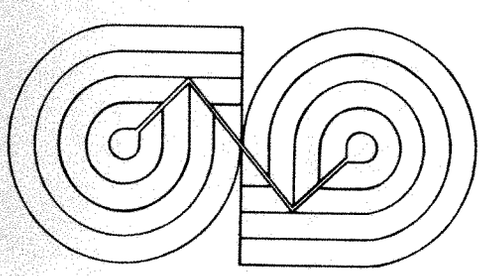
Amitriptyline: Note: Listing includes a few reactions not reported f drug, but which have occurred with other pharmacologically similar tri antidepressant drugs and must be considered when amitriptyline is ad tered. *Cardiovascular:* Hypotension; hypertension; tachycardia; palpi myocardial infarction; arrhythmias; heart block; stroke. *CNS and Neu cular:* Confusional states; disturbed concentration; disorientation; delu hallucinations; excitement; anxiety; restlessness; insomnia; nightm numbness, tingling, and paresthesias of the extremities; peripheral pathy; incoordination; ataxia; tremors; seizures; alteration in EEG pa extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH diuretic hormone) secretion. *Anticholinergic:* Dry mouth; blurred disturbance of accommodation; increased intraocular pressure; constip paralytic ileus; urinary retention; dilatation of urinary tract. *Allergic* rash; urticaria; photosensitization; edema of face and tongue. *Hemat* Bone marrow depression including agranulocytosis; leukopenia; eos lia; purpura; thrombocytopenia. *Gastrointestinal:* Nausea; epigastr tress; vomiting; anorexia; stomatitis; peculiar taste; diarrhea; p swelling; black tongue. Rarely hepatitis (including altered liver functi jaundice). *Endocrine:* Testicular swelling and gynecomastia in the breast enlargement and galactorrhea in the female; increased or dec libido; elevated or lowered blood sugar levels. *Other:* Dizziness, wea fatigue; headache; weight gain or loss; increased perspiration; u frequency; mydriasis; drowsiness; alopecia. *Withdrawal Symptoms:* cessation after prolonged administration may produce nausea, headach malaise. These are not indicative of addiction.

OVERDOSAGE: All patients suspected of having taken an over should be admitted to a hospital as soon as possible. Treatment is s matic and supportive. However, the intravenous administration of 1- of physostigmine salicylate is reported to reverse the symptoms of tr antidepressant poisoning. Because physostigmine is rapidly metaboliz dosage of physostigmine should be repeated as required particularly threatening signs such as arrhythmias, convulsions, and deep coma re persist after the initial dosage of physostigmine. On this basis, in overdosage with perphenazine-amitriptyline combinations, sympt treatment of central anticholinergic effects with physostigmine sal should be considered.

J 6TR06 R3 (DC6613212)

For more detailed information, consult your MSD Representative or see full Prescribing Information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486.

MSD



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Phillip H.* was an engineer with a problem... but now he's solving problems for industry

Patient name	Phillip H.*	**Sex:** Male	**Age:** 29
Marital status S. (M.) W. D.		**Occupation:**	Mechanical engineer

History: Studious, bright, only son; much older sister. Father an invalid; mother, a successful business woman. A "loner" with multiple psychosomatic illnesses, he started dating late and married first girl friend, several years older

Onset of illness: Upset at wife's pregnancy, expressed strong guilt feelings after her miscarriage; said people accused him of "murder." Unable to eat or sleep properly, felt he was "losing his mind." Unable to work, obtained leave of absence and was referred to psychiatrist

Diagnosis: Acute schizophrenia

Treatment: LOXITANE® Loxapine Succinate 25 mg b.i.d. increased to 50 mg t.i.d. over 2 weeks. Supportive psychotherapy three times a week

Therapeutic course: First week, eating and sleeping better. Guilt feelings expressed le often. Mild akathisia responded to appropriate antiparkinson medication

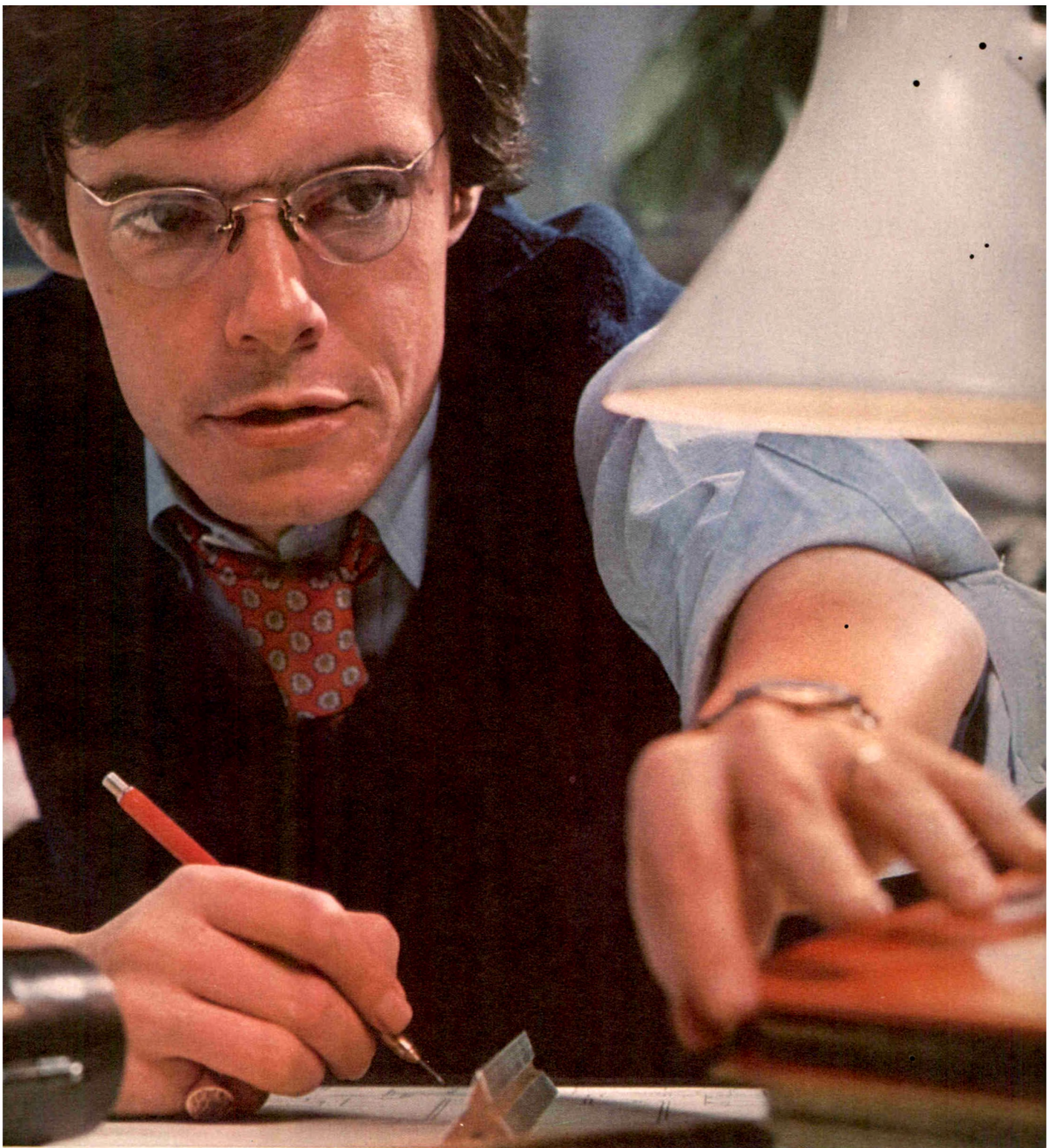
Second week, communicating better. Feared that "fatherhood w develop into rivalry," feared loss of wife's love

Third week, no delusions or hallucinations, relating better, bu to only one person at a time

Affect still blunted at one month, otherwise asymptomatic. LOXITANE decreased to 50 mg b.i.d.; antiparkinson medicati continued

Follow-up: 10 weeks; at work for one week, seems to be adjusting well. A parkinson medication discontinued; psychotherapy weekly LOXITANE 25 mg t.i.d.

*Not an actual patient. The collection of clinical d upon which this simulation is built illustrates the action of LOXITANE® Loxapine Su as reported in clinical studies.



He's adjusting well in his job, and with psychotherapy and medication, he also can be helped to feel more secure, less threatened in his relationships with others.

Loxitane®
LOXAPINE SUCCINATE
Lederle

For the symptoms that interrupt careers

See LOXITANE prescribing information on following page for indications, warnings and precautions and for more detailed information concerning side effects.

Loxitane®
LOXAPINE SUCCINATE
Lederle

For the symptoms that interrupt careers

Recommended Daily Dosage

Initial Dosage	MILD 10 mg b.i.d.	MODERATE 10 mg t.i.d. or q.i.d.	SEVERE 25 mg b.i.d.
First 7 to 10 Days	Increase dosage until psychotic symptoms are controlled Dosage should not exceed 250 mg/day Usual dosage during titration: 50 to 150 mg/day		
Maintenance Dosage	Adjust to lowest effective level. Usual maintenance dosage: 60 to 100 mg/day. Many patients are controlled with dosages as low as 20 to 60 mg/day.		



Brief Summary

LOXITANE® Loxapine Succinate Capsules
OR
LOXITANE® C Loxapine Hydrochloride Oral Concentrate

Description: A dibenzoxazepine compound, representing a new subclass of tricyclic antipsychotic agent, chemically distinct from the thioxanthenes, butyrophenones, and phenothiazines.

Indications: For the manifestations of schizophrenia. Loxapine has not been evaluated for the management of behavioral complications in mental retardation, and therefore cannot be recommended.

Contraindications: In comatose or severe, drug-induced depressed states (alcohol, barbiturates, narcotics, etc.); and in individuals with known hypersensitivity to the drug.

Warnings: In Pregnancy: Safe use during pregnancy or lactation has not been established; in pregnancy, nursing mothers, or women of child-bearing potential, weigh potential benefits against possible hazards to mother and child. No embryotoxicity or teratology was observed in studies in rats, rabbits or dogs, although with the exception of one rabbit study, the highest dosage was only two times the maximum recommended human dose and in some studies they were below this dose. Perinatal studies have shown renal papillary abnormalities in offspring of rats treated from mid-pregnancy with doses of 0.6 and 1.8 mg./kg., doses which approximate the usual human dose but which are considerably below the maximum recommended human dose. **In Children:** Studies have not been performed in children; therefore this drug is not for use below the age of 16.

May impair mental and/or physical abilities, especially during the first few days of therapy; warn ambulatory patients about activities requiring alertness (*e.g.*, operating vehicles or machinery), and about concomitant use of alcohol and other CNS depressants.

Precautions: Use with extreme caution in patients with history of convulsive disorders; seizures have been reported in epileptics receiving this drug at antipsychotic dose levels, and may occur even with maintenance of routine anticonvulsant drug therapy. Has an antiemetic effect in animals; this effect in man may mask signs of overdosage of toxic drugs and obscure conditions such as intestinal obstruction and brain tumor. Use with caution in patients with cardiovascular disease. Increased pulse rates in the majority of patients receiving antipsychotic doses and transient hypotension have been reported. In hypotension requiring vasopressor therapy, preferred drugs may be norepinephrine or angiotensin. Usual doses of epinephrine may be ineffective because of inhibition of its vasopressor effect by loxapine. Possibility of ocular toxicity from loxapine cannot be excluded at this time, therefore observe carefully for pigmentary retinopathy and lenticular pigmentation (observed in some patients receiving certain other antipsychotic drugs for prolonged periods). Because of possible anticholinergic action, use with caution in patients with glaucoma or a tendency to urinary retention, particularly with concomitant administration of anticholinergic-type antiparkinson medication.

Adverse Reactions: CNS: Adverse effects other than extrapyramidal, infrequent. Drowsiness, usually mild, may occur at beginning of therapy or when dosage is increased, usually subsiding with continued therapy. Incidence of sedation is less than that of certain aliphatic phenothiazines and slightly more than piperazine phenothiazines. Dizziness, faintness, staggering gait, muscle twitching, weakness, and confusional states have been reported. Extrapyramidal reactions during use of this drug have been reported frequently, often during the first few days of treatment. In most patients, these involve Parkinson-like symptoms such as tremor, rigidity, excessive salivation, masked facies, akathisia, usually not severe and controlled by dosage reduction or use of antiparkinson drugs in usual dosage. *Less frequent, but more severe:* Dystonias, including spasms of muscles of neck and face, tongue protrusion, oculogyric movement; dyskinesia in the form of choreo-athetoid movements. These sometimes require reduction or temporary withdrawal of drug dosage in addition to appropriate counteractive drugs. *Persistent Tardive Dyskinesia:* May appear in some patients on long-term therapy, or after therapy has been discontinued; the risk greater in the elderly, especially females, on high dosage. These symptoms, persistent and in some patients apparently irreversible, are characterized by rhythmical involuntary movement of tongue, face, mouth or jaw (*e.g.*, protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements, sometimes accompanied by involuntary movements of extremities). No known effective treatment; discontinue all antipsychotic agents if these symptoms appear. The necessity to reinstitute treatment or increase dosage, or switch to a different antipsychotic agent, may mask syndrome. If medication is stopped when fine vermicular movements of the tongue occur, the syndrome may not develop. *Cardiovascular:* Tachycardia, hypotension, hypertension, lightheadedness, syncope. A few cases of EKG changes similar to those seen with phenothiazines have been reported, not known to be related to loxapine use. *Skin:* Dermatitis, edema (puffiness of face), pruritus, seborrhea. Possible photosensitivity and/or phototoxicity; skin rashes of uncertain etiology seen in a few patients during hot summer months. *Anticholinergic:* Dry mouth, nasal congestion, constipation, blurred vision—more likely to occur with concomitant use of antiparkinson agents. *Other:* Nausea, vomiting, weight gain or loss, dyspnea, ptosis, hyperpyrexia, flushed facies, headache, paresthesia, polydipsia. Rarely, galactorrhea and menstrual irregularity of uncertain etiology.

Dosage and Administration: Administered orally, usually in 2 to 4 divided doses a day. Adjust dosage to patient's need relative to severity of symptoms and history of response to antipsychotic drugs. Recommended initial dosage is 10 mg. b.i.d.; in severely disturbed patients up to 50 mg. daily. Increase dosage during first 7 to 10 days until psychotic symptoms are controlled. Usual therapeutic and maintenance range is 60 mg.-100 mg. daily. More than 250 mg. daily is not recommended. Maintenance dosage should be at lowest level to control symptoms; many patients have been maintained on 20 mg. to 60 mg. daily.

LOXITANE C Oral Concentrate should be mixed with orange and grapefruit juice shortly before administration. Use only enclosed calibrated dropper.

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A Division of American Cyanamid Company
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Just Published . . .

The Rape Victim

by Elaine Hilberman, M.D.

Mythologies about rape are legion. However, in recent years behavioral scientists have accumulated a body of information about rape and about an unfolding series of reactions experienced by the victim after rape. The aim of this concise, tightly reasoned book is to summarize what is now known about the needs and experiences of the victim and her family, and to provide a framework in which the clinician can more knowledgeably supply assistance and support. This document, published by the American Psychiatric Association, is the result of a project of the Association's Committee on Women.

Pointing out that the profound impact of rape stress is best understood in the context of rape as "a crime against the person and not the hymen," Dr. Hilberman sees rape as the ultimate violation of the self short of homicide, as an act of violence and humiliation in which not only is the victim's very existence threatened, and her inner and most private space invaded, but her autonomy and control are totally demolished. Dr. Hilberman shows that if and when the victim reports the crime, she is confronted with a complex series of yet additional stresses growing out of her contact with the hospital, police, courts, media and community opinion. Additionally, her crisis differs from other crises in that her usual social support system is likely to be disrupted. Her immediate needs are for empathy and safety, and a sense of control over what is happening in her dealings with the several agencies. Dr. Hilberman shows that in the absence of sensitivity to these needs, the experience of reporting becomes another assault.

This book presents a professional, sobering, and balanced picture of the problems of the rape victim and details what the clinician must know—about local hospital policy, criminal justice procedure, rape statutes, and community attitudes and services—in order to be able to treat victims with an understanding of the larger context in which rape occurs.

ELAINE HILBERMAN, M.D., *is in the Department of Psychiatry at the University of North Carolina School of Medicine.*

Paperback edition—$5.00 each, may be ordered from the AMERICAN PSYCHIATRIC ASSOCIATION, Publication Sales, 1700 18th St., N.W., Washington, D.C. 20009.

Hardback edition—$7.95 may be ordered from Basic Books, Inc., 10 East 53rd Street, New York, New York 10022.

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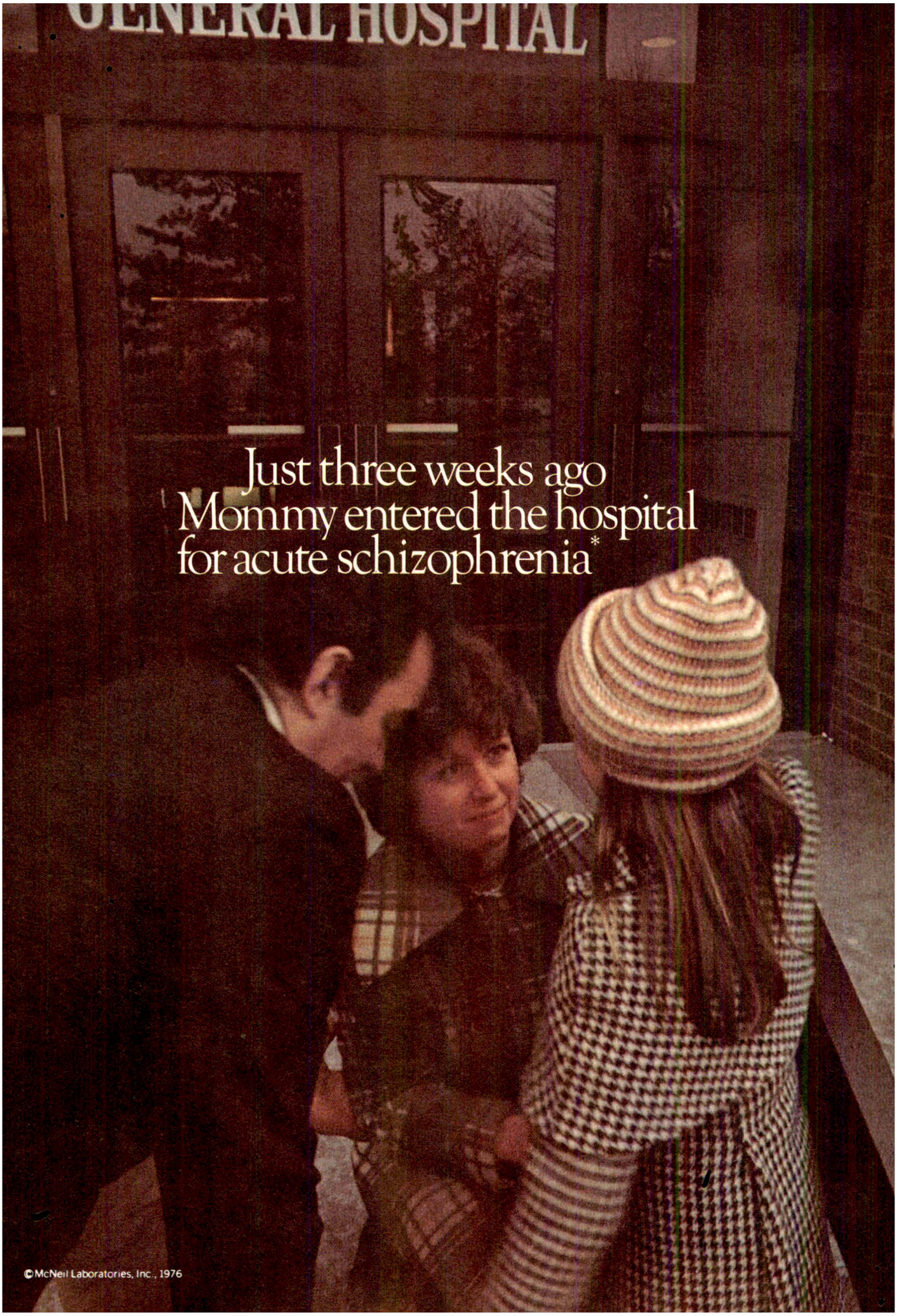
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Haldol®
(haloperidol)
tablets/concentrate/injection

For prompt control with minimal risk of adverse reactions

Highly effective in a wide range of psychotic symptoms,[1-10]
such as hallucinations, delusions, suspiciousness, hostility, mania, psychomotor agitation, etc., in both acute and chronic disorders.

Usually leaves patients more responsive to rehabilitation...[1,2]
better able to care for themselves and take part in therapy sessions.

Helps avoid long-term hospitalization.[1,3-5]
It is often possible to release the patient for treatment as an outpatient after a short confinement. HALDOL haloperidol controls his psychotic symptoms, usually leaving him sufficiently alert to carry on normal functions.

Permits aggressive titration to effective dosage levels,[3,6]
up to 100 mg/day orally, to achieve optimal response when patient is inadequately controlled at lower dosage...to help you promptly control and stabilize new patients.

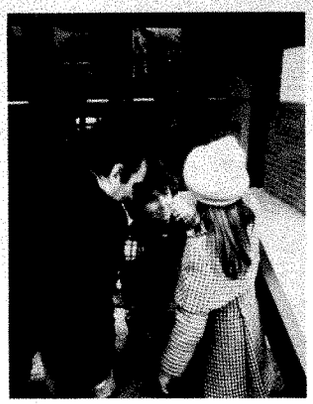
Common side effects easily controlled.[1,3,6-8]
Although extrapyramidal symptoms (EPS) have been reported frequently, they are usually dose-related and readily controlled with dose adjustment or antiparkinson drugs. EPS often diminish spontaneously with continued use of HALDOL haloperidol.

*Not an actual case history, this situation illustrates the action of HALDOL haloperidol as reported in various clinical studies (available on request).

Reduces risk of several treatment problems.[1,2,6,7,9]
Transient hypotension occurs rarely and severe orthostatic hypotension has not been reported. Marked sedation is rare, although some instances of drowsiness have been reported. In addition, with chronic use, it is unlikely to cause hepatic damage, serious hematologic reactions, photosensitivity reactions and skin rashes...and has minimal effect on renal function.

Please turn page for information relating to Indications, Contraindications, Warnings, Precautions and Adverse Reactions.

Important: Full directions for use should be read before HALDOL haloperidol is administered or prescribed.



Haldol®
(haloperidol)
tablets/concentrate/injection

For prompt control of psychotic symptoms with minimal risk of adverse reactions

A Dosage Form for Every Need:

5 tablet strengths for convenience in individualizing dosage: ½ mg., 1 mg., 2 mg., 5 mg. and 10 mg.

A tasteless, odorless, colorless liquid concentrate for better patient acceptability: 2 mg. per ml.

A rapid-acting injection for psychiatric emergencies: 5 mg. per ml., with 1.8 mg. methylparaben and 0.2 mg. propylparaben per ml., and lactic acid for pH adjustment to 3.4±0.2.

Contraindications: Severe depression, coma, CNS depression due to centrally-acting depressants, Parkinson's disease, hypersensitivity to the drug.

Warnings: ***Usage in Pregnancy:*** Safe use in pregnancy or in women likely to become pregnant has not been established; use only if benefit clearly justifies potential hazards. Infants should not be nursed during drug treatment.

Usage in Children: Safety and effectiveness not established; not recommended in pediatric age group.

Combined Use with Lithium: Patients receiving lithium plus haloperidol should be monitored closely for early evidence of neurological toxicity.

General: Bronchopneumonia, sometimes fatal, has followed use of major tranquilizers, including haloperidol. Prompt remedial therapy should be instituted if dehydration, hemoconcentration or reduced pulmonary ventilation occurs, especially in the elderly. Decreased serum cholesterol and/or cutaneous and ocular changes have been reported with chemically-related drugs, although not with haloperidol. Mental and/or physical abilities required for hazardous tasks or driving may be impaired. Alcohol should be avoided due to possible additive effects and hypotension.

Precautions: Administer cautiously to patients: (1) with severe cardiovascular disorders, due to the possibility of transient hypotension and/or precipitation of anginal pain (if a vasopressor is required, epinephrine should not be used since HALDOL haloperidol may block its vasopressor activity and paradoxical further lowering of blood pressure may occur); (2) receiving anticonvulsant medication since HALDOL haloperidol may lower the convulsive threshold; (3) with known allergies or a history of allergic reactions to drugs; (4) receiving anticoagulants. Concomitant antiparkinson medication, if required, may have to be continued after HALDOL haloperidol is discontinued because of different excretion rates; if both are discontinued simultaneously, extrapyramidal symptoms may occur. Intraocular pressure may increase when anticholinergic drugs, including antiparkinson drugs, are administered concomitantly with HALDOL haloperidol. When HALDOL haloperidol is used for mania in cyclic disorders, there may be a rapid mood swing to depression. Severe neurotoxicity may occur in patients with thyrotoxicosis receiving antipsychotic medication, including HALDOL haloperidol.

Adverse Reactions: ***CNS Effects:*** *Extrapyramidal Reactions:* Neuromuscular (extrapyramidal) reactions have been reported frequently, often during the first few days of treatment. Generally they involved Parkinson-like symptoms which were usually mild to moderately severe and usually reversible. Other types of neuromuscular reactions (motor restlessness, dystonia, akathisia, hyperreflexia, opisthotonos, oculogyric crises) have been reported far less frequently, but were often more severe. Severe extrapyramidal reactions have been reported at relatively low doses. Generally, extrapyramidal symptoms are dose-related since they occur at relatively high doses and disappear or become less severe when the dose is reduced. Antiparkinson drugs may be required. Persistent extrapyramidal reactions have been reported and the drug may have to be discontinued in such cases. *Withdrawal Emergent Neurological Signs:* Abrupt discontinuation of short-term antipsychotic therapy is generally uneventful. However, some patients on maintenance treatment experience transient dyskinetic signs after abrupt withdrawal. In certain cases these are indistinguishable from "Persistent Tardive Dyskinesia" except for duration. It is unknown whether gradual withdrawal will reduce the occurrence of these signs, but until further evidence is available haloperidol should be gradually withdrawn. *Persistent Tardive Dyskinesia:* Although rarely reported with HALDOL haloperidol, tardive dyskinesia may appear during or after long-term therapy. The risk appears to be greater in elderly patients on high-dose therapy, especially females. Symptoms are persistent and sometimes appear irreversible; there is no known effective treatment and all antipsychotic agents should be discontinued. The syndrome may be masked by reinstitution of drug, increasing dosage, or switching to a different antipsychotic agent. *Other CNS Effects:* Insomnia, restlessness, anxiety, euphoria, agitation, drowsiness, depression, lethargy, headache, confusion, vertigo, grand mal seizures, and exacerbation of psychotic symptoms. ***Cardiovascular Effects:*** Tachycardia and hypotension. ***Hematologic Effects:*** Reports of mild, usually transient leukopenia and leukocytosis, minimal decreases in red blood cell counts, anemia, or a tendency toward lymphomonocytosis; agranulocytosis rarely reported and only in association with other medication. ***Liver Effects:*** Impaired liver function and/or jaundice reported. ***Dermatologic Reactions:*** Maculopapular and acneiform reactions, isolated cases of photosensitivity, loss of hair. ***Endocrine Disorders:*** Lactation, breast engorgement, mastalgia, menstrual irregularities, gynecomastia, impotence, increased libido, hyperglycemia and hypoglycemia. ***Gastrointestinal Effects:*** Anorexia, constipation, diarrhea, hypersalivation, dyspepsia, nausea and vomiting. ***Autonomic Reactions*** Dry mouth, blurred vision, urinary retention and diaphoresis. ***Respiratory Effects:*** Laryngospasm, bronchospasm and increased depth of respiration.

The injectable form is intended only for acutely agitated psychotic patients with moderately severe to very severe symptoms.

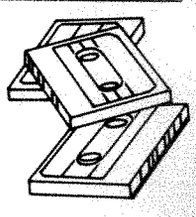
IMPORTANT: Full directions for use should be read before HALDOL haloperidol is administered or prescribed. HALDOL tablets are manufactured by McNeil Laboratories Co., Dorado, Puerto Rico 00646. 5/77

References:

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Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

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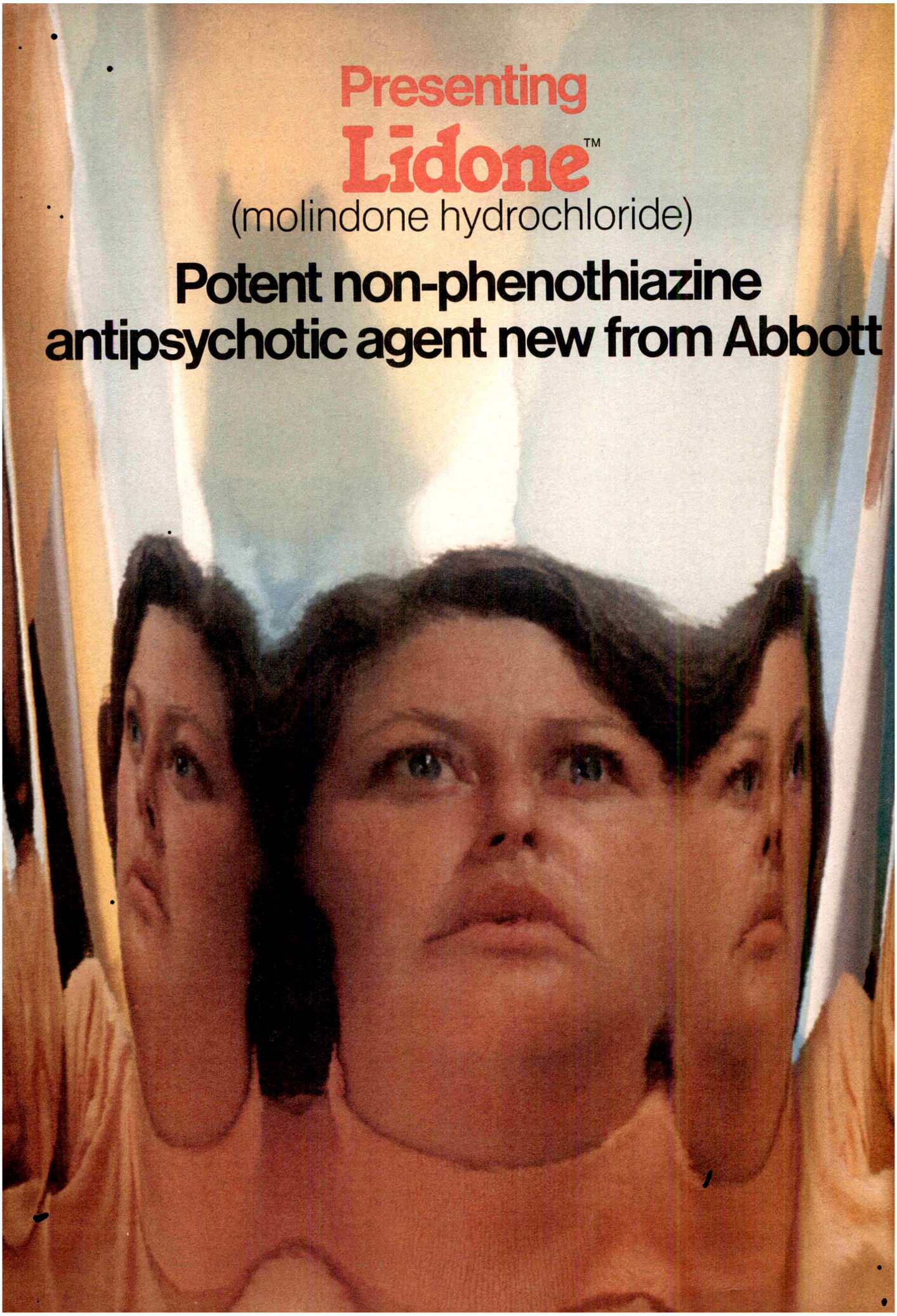
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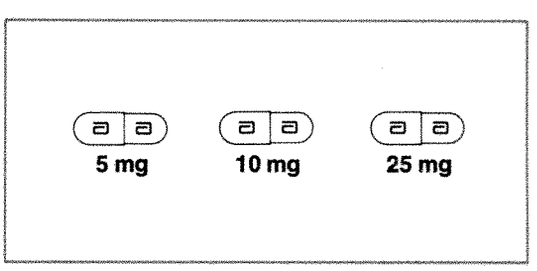
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Lidone is classed among the "alerting" or less-sedating group of major tranquilizers. If drowsiness does occur during initial therapy, it is usually transient. Patients on continuing Lidone therapy tend to stay alert; hence they can be more responsive to rehabilitation measures.

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Brief summary of
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Lidone™ (molindone hydrochloride)

DESCRIPTION — LIDONE (molindone hydrochloride) is a dihydroindolone compound which is not structurally related to the phenothiazines, the butyrophenones or the thioxanthenes.

LIDONE is 3-ethyl-6, 7-dihydro-2-methyl-5-(morpholinomethyl) indol-4(5H)-one hydrochloride. It is a white crystalline powder, freely soluble in water and alcohol and has a molecular weight of 312.67.

ACTIONS — LIDONE (molindone hydrochloride) has a pharmacological profile in laboratory animals which predominantly resembles that of major tranquilizers causing reduction of spontaneous locomotion and aggressiveness, suppression of a conditional response and antagonism of the bizarre stereotyped behavior and hyperactivity induced by amphetamines. In addition, LIDONE antagonizes the depression caused by the tranquilizing agent tetrabenazine.

In human clinical studies tranquilization is achieved in the absence of muscle relaxing or incoordinating effects. Based on EEG studies, LIDONE exerts its effect on the ascending reticular activating system.

Human metabolic studies show LIDONE (molindone hydrochloride) to be rapidly absorbed and metabolized when given orally. Unmetabolized drug reached a peak blood level at 1.5 hours. Pharmacological effect from a single oral dose persists for 24 to 36 hours. There are 36 recognized metabolites with less than 2% to 3% unmetabolized LIDONE being excreted in urine and feces.

INDICATIONS — LIDONE (molindone hydrochloride) is indicated in the management of the manifestations of schizophrenia.

CONTRAINDICATIONS — LIDONE (molindone hydrochloride) is contraindicated in severe central nervous system depression (alcohol, barbiturates, narcotics, etc.) or comatose states, and in patients with known hypersensitivity to the drug.

WARNINGS

Usage in Pregnancy: Studies in pregnant patients have not been carried out. Reproduction studies have been performed in the following animals:

Pregnant Rats oral dose —
20 mg/kg/day — 2 weeks, no adverse effect
40 mg/kg/day — 2 weeks, no adverse effect

Pregnant Mice oral dose —
20 mg/kg/day — 9 days, slight increase resorptions
40 mg/kg/day — 9 days, slight increase resorptions

Pregnant Rabbits oral dose —
5 mg/kg/day — 12 days, no adverse effect
10 mg/kg/day — 12 days, no adverse effect
20 mg/kg/day — 12 days, no adverse effect

Animal reproductive studies have not demonstrated a teratogenic potential. The anticipated benefits must be weighed against the unknown risks to the fetus if used in pregnant patients.

Nursing Mothers: Data are not available on the content of LIDONE (molindone hydrochloride) in the milk of nursing mothers.

Usage in Children: Use of LIDONE (molindone hydrochloride) in children below the age of twelve years is not recommended because safe and effective conditions for its usage have not been established.

PRECAUTIONS — Some patients receiving LIDONE (molindone hydrochloride) may note drowsiness initially and they should be advised against activities requiring mental alertness until their response to the drug has been established.

Increased activity has been noted in patients receiving LIDONE. Caution should be exercised where increased activity may be harmful.

LIDONE does not lower the seizure threshold in experimental animals to the degree noted with more sedating antipsychotic drugs. However, convulsive seizures have been reported in a few instances.

LIDONE has an antiemetic effect in animals. A similar effect may occur in humans and may obscure signs of intestinal obstruction or brain tumor.

ADVERSE REACTIONS

CNS Effects — The most frequently occurring effect is initial drowsiness that generally subsides with continued usage of the drug or lowering of the dose.

Noted less frequently were depression, hyperactivity and euphoria.

Neurological Extrapyramidal Reactions — Extrapyramidal reactions noted below may occur in susceptible individuals and are usually reversible with appropriate management.

Akathisia — Motor restlessness may occur early.

Parkinson Syndrome — Akinesia, characterized by rigidity, immobility and reduction of voluntary movements and tremor, have been observed. Occurrence is less frequent than akathisia.

Dystonic Syndrome — Prolonged abnormal contractions of muscle groups occur infrequently. These symptoms may be managed by the addition of a synthetic antiparkinson agent (other than L-dopa), small doses of sedative drugs, and/or reduction in dosage.

Autonomic Nervous System — Occasionally blurring of vision, tachycardia, nausea, dry mouth and salivation have been reported. Urinary retention and constipation may occur particularly if anticholinergic drugs are used to treat extrapyramidal symptoms.

Hematological — There have been rare reports of leucopenia and leucocytosis. If such reactions occur, treatment with LIDONE may continue if clinical symptoms are absent. Alterations of blood glucose, liver function tests, B.U.N., and red blood cells have not been considered clinically significant.

Metabolic and Endocrine Effects — Alteration of thyroid function has not been significant. Amenorrhea has been reported infrequently. Resumption of menses in previously amenorrheic women has been reported. Initially heavy menses may occur. Lactation associated with LIDONE therapy has not been reported. Increase in libido has been noted in some patients. Impotence has not been reported. Although both weight gain and weight loss have been in the direction of normal or ideal weight, excessive weight gain has not occurred with LIDONE.

Cardiovascular — Rare, transient, non-specific T wave changes have been reported on E.K.G. Association with a clinical syndrome has not been established. Rarely has significant hypotension been reported.

Ophthalmological — Lens opacities and pigmentary retinopathy have not been reported where patients have received LIDONE (molindone hydrochloride). In some patients, phenothiazine induced lenticular opacities have resolved following discontinuation of the phenothiazine while continuing therapy with LIDONE.

Skin — Early, non-specific skin rash, probably of allergic origin, has occasionally been reported. Skin pigmentation has not been seen with LIDONE usage alone.

LIDONE (molindone hydrochloride) has certain pharmacological similarities to other antipsychotic agents. Because adverse reactions are often extensions of the pharmacological activity of a drug, all of the known pharmacological effects associated with other antipsychotic drugs should be kept in mind when LIDONE is used. Upon abrupt withdrawal after prolonged high dosage an abstinence syndrome has not been noted.

Tardive Dyskinesia — Although rarely reported with LIDONE (molindone hydrochloride) symptoms were reversible upon discontinuation of therapy.

Tardive dyskinesia associated with other agents has appeared in some patients on long-term therapy and has also appeared after drug therapy has been discontinued. The risk appears to greater in elderly patients on high-dose thera especially females. The symptoms are persiste and in some patients appear to be irreversible. T syndrome is characterized by rhythmical involu tary movements of the tongue, face, mouth or j (e.g., protrusion of tongue, puffing of chee puckering of mouth, chewing movements). The may be involuntary movements of extremities.

There is no known effective treatment of tardi dyskinesia; antiparkinsonism agents usually not alleviate the symptoms of this syndrome. It suggested that all antipsychotic agents be disc tinued if these symptoms appear. Should it be n essary to reinstitute treatment, or increase t dosage of the agent, or switch to a different an psychotic agent, the syndrome may be masked. has been reported that fine vermicular movemer of the tongue may be an early sign of the syndro and if the medication is stopped at that time t syndrome may not develop.

DOSAGE AND ADMINISTRATION — Initial a maintenance doses of LIDONE (molindone hyd chloride) should be individualized, and t minimal effective dose should be employed. Elde and debilitated patients should be started on low dosage.

DOSAGE SCHEDULE, BASED ON SEVERITY OF SYMPTOMATOLOGY

1. Mild — 5 mg three or four times a day; an crease to 15 mg three or four times a day may required.
2. Moderate — 10 mg three or four times a day; increase to 25 mg three or four times a day m be required.
3. Severe — daily dosage as high as 225 mg may required.

DRUG INTERACTIONS — Potentiation of dru administered concurrently with LIDONE (mol done hydrochloride) has not been reported. Ad tionally, animal studies have not shown increas toxicity when LIDONE is given concurrently w representative members of three classes of dru (i.e., barbiturates, chloral hydrate and antipark son drugs).

MANAGEMENT OF OVERDOSAGE — Symp matic, supportive therapy should be the rule.

Gastric lavage is indicated for the reduction absorption of LIDONE (molindone hydrochlori which is freely soluble in water.

Since the adsorption of LIDONE (molindone drochloride) by activated charcoal has not be determined, the use of this antidote must be c sidered of theoretical value.

Emesis in a comatose patient is contraindicat Additionally, while the emetic effect apomorphine is blocked by LIDONE in animals, t blocking effect has not been determined humans.

A significant increase in the rate of removal unmetabolized LIDONE from the body by for diuresis, peritoneal or renal dialysis would not expected. (Only 2% of a single ingested dose LIDONE is excreted unmetabolized in the urin However, poor response of the patient may jus use of these procedures.

While the use of laxatives or enemas might based on general principles, the amount of metabolized LIDONE in feces is less than 1%. trapyramidal symptoms have responded to the of diphenhydramine (Benadryl*) and the s thetic anticholinergic antiparkinson agents (Artane*, Cogentin*, Akineton*).

HOW SUPPLIED — LIDONE (molindone drochloride) capsules are supplied in bottles of in the following dosage strengths and colors:

5 mg (NDC 0074-5542-13) blue and cream
10 mg (NDC 0074-5543-13) red and cream
25 mg (NDC 0074-5544-13) brown and cream.

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For Brief Summary please see following page.

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● PAMELOR® helps relieve the sleep problems that accompany depression: difficulty in falling asleep, restless sleep, and early-morning awakening. An improved pattern of sleep begins to appear in some patients within the first week of therapy.

● Overall improvement is often observed by the end of the second week. Maximum improvement with Pamelor®, as with other antidepressants, may require a longer period of therapy, particularly in severe depressive illnesses.

PAMELOR® allows patients to remain calm yet alert

● Helps improve depressed mood with a low level of stimulatory effect.

● PAMELOR® also has a mild calming action to help relieve the anxiety and tension that are often symptoms of depression. Because PAMELOR® rarely causes excessive drowsiness, patients are more alert and better able to function. Nevertheless, they should be cautioned about driving or operating machinery.

Indications: For relief of depressive symptoms. Endogenous depressions are more likely to be alleviated than others.

Contraindications: Hypersensitivity. Should not be given concomitantly with MAO inhibitors or within 2 weeks of the use of this drug since hyperpyretic crises, severe convulsions, and fatalities have occurred when similar tricyclic antidepressants were used in such combinations. Cross-sensitivity with other dibenzazepines is a possibility. Contraindicated during acute recovery period after myocardial infarction.

Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: The pharmacologic similarities among the tricyclic antidepressant drugs require that each of the following reactions be considered when nortriptyline is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though these are not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

Dosage and Administration: *Usual adult dose*—25 mg. three or four times daily; dosage should begin at a low level and increase as required. *Elderly and Adolescent:* 30 to 50 mg. per day, in divided doses. Doses above 100 mg. per day and use in children are not recommended. If a patient develops minor side effects, the dosage should be reduced. The drug should be discontinued promptly if adverse effects of a serious nature or allergic manifestations occur.

How Supplied: Capsules 10 mg. and 25 mg.; solution 10 mg./5 cc.

For more detailed information see full prescribing information.

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a valuable link to the resources of the American Psychiatric Association

for institutions and agencies concerned with the care of the mentally disabled

Enrollment in the Hospital & Community Psychiatry Service brings multiple copies of *Hospital & Community Psychiatry* into member agencies every month, keeping staff up to date on developments and issues in the mental health field, offering new ideas and fresh perspectives, and serving as a useful resource in staff development and training programs.

Hospital & Community Psychiatry is just one of the benefits of membership in the H&CP Service. Others include a film library containing more than a hundred films specially chosen for their usefulness in staff development and community education programs; supplementary mailings of important books, reports, articles, or other material of special interest to administrators or clinicians; reduced registration fees at the annual fall Institute on Hospital & Community Psychiatry; and, on request, information and consultation from the professional staff of the American Psychiatric Association. The H&CP Service also sponsors the annual Achievement Awards competition, which gives special recognition to outstanding programs for the mentally ill and mentally retarded.

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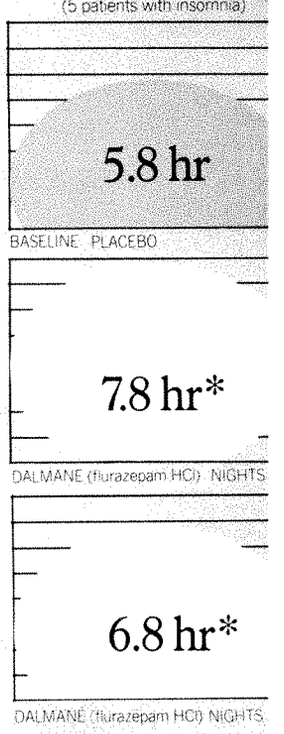
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WASHINGTON, D.C. 20009

From the Sleep Research Laborator

Dalmane® (flurazepam HC proved the most effective hypnotic over 14 nights...[1]



For those patients who need it, Dalmane (flurazepam HCl) alone provides continued effectiveness[2,3]

Since insomnia is often transient and intermittent, the prolonged administration of a hypnotic is generally not necessary or recommended. But for those patients whose insomnia is a chronic problem, the continued effectiveness of Dalmane is a decided benefit. Even at the end of a 28-night medication period, patients with chronic insomnia were falling asleep faster, spending less time awake during the night, and sleeping substantially longer on Dalmane 30 mg *h.s.* than on baseline placebo nights (average results). This effectiveness was proved[2] and confirmed[3] in two independent sleep research studies using a new 47-night protocol. When similarly evaluated, pentobarbital was ineffective at the end of two weeks.[2]

14 consecutive nights:
3 in sleep laboratory, 8 at home,
3 in sleep laboratory



..and now, the only one proved effective over 28 nights.[2,3]

n previous studies,[1] hloral hydrate and glutethimide ost most or all effectiveness

ı fact, continued effectiveness for both inducing and maintain-ıg sleep over 14 consecutive nights[1] has long been an exclusive enefit of Dalmane. No other available sleep agent, including ıloral hydrate or glutethimide,[1] has been able to demonstrate ıch effectiveness in the sleep research laboratory.

.he safety record of)almane (flurazepam HCl) is well stablished

almane is relatively safe and well tolerated. Prolonged dministration is not generally necessary, but if used repeatedly, eriodic blood counts and liver and kidney function tests ıould be performed.

Dalmane®
(flurazepam HCl)

relatively safe...
no need
to increase
dosage

ROCHE

28 consecutive nights:
3 in sleep laboratory, 7 at home,
4 in sleep laboratory, 10 at home,
4 in sleep laboratory[3]

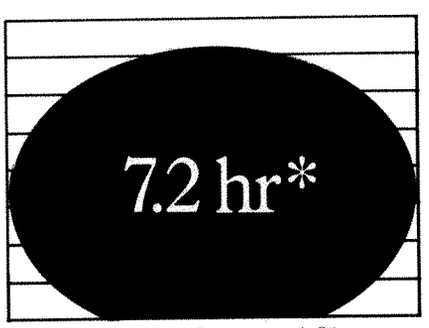
Please see following page for a summary of product information.

1,5,7,14,28 nights... as needed, the effectiveness of **Dalmane**® one *h.s.* (flurazepam HCl) C IV meets the challenge of insomnia

One 30-mg capsule *h.s.*—usual adult dosage (15 mg may suffice in some patients).
One 15-mg capsule *h.s.*—initial dosage for elderly or debilitated patients.

- **Patients fall asleep rapidly, sleep longer on a single *h.s.* dose**
- **Nighttime awakenings and time spent awake are reduced**
- **Effectiveness maintained without dosage increase from night to night**
- **Patients generally awaken refreshed; morning "hangover" is infrequent**

TOTAL SLEEP TIME (OVERALL MEAN)[3]
(5 patients with insomnia)



Dalmane (flurazepam HCl)
nights 1-3, 12-14, 26-28

*Significantly increased over baseline placebo ($p < .05$).

REFERENCES:

1. Kales A, *et al*: *Arch Gen Psychiatry* 23:226-232, Sep 1970
2. Kales A, *et al*: *Clin Pharmacol Ther* 18:356-363, Sep 1975
3. Dement WC, *et al*: Long-term effectiveness of flurazepam 30 mg *h.s.* on chronic insomniacs. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, Jun 30-Jul 4, 1975

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).
***Usage in Pregnancy:* Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane (flurazepam HCl), a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

ROCHE

ROCHE LABORATORIES
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

Coverage and Utilization of Care for Mental Conditions Under Health Insurance —Various Studies, 1973–74

By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

This book reports six studies that add to the growing body of data demonstrating the feasibility of covering mental illness under health insurance. Although in some ways it may be considered a supplement to APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (Reed, Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

- Utilization of mental benefits under the Blue Cross and Blue Shield plan for federal employees, documenting that a "plateau" in the use of these benefits was reached in 1973–74.
- Comparison of benefits for mental and other illnesses under selected employee health benefit plans.
- Coverage of mental illness under collective bargaining agreements of selected unions.
- Utilization of care for mental conditions under the Canadian health insurance program, which gives the same coverage for mental as for other conditions.
- Updating of information on Blue Cross benefits for hospital care of mental illness.
- Data from selected Blue Cross and Blue Shield plans on coverage and utilization of mental conditions, with emphasis on major medical coverage.

80pp. Paperbound

Single copy, $4.00; 10–49 copies; $3.80 each; 50 or more copies, $3.20 each.

SPECIAL OFFER: One copy of this book (regular price, $4.00) and one copy of **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (regular price, $6.50) for $8.50.

Publications Services Division
American Psychiatric Association
1700 18th St., N.W., Washington, D.C. 20009

Please send me _____ copies of **COVERAGE AND UTILIZATION OF CARE FOR MENTAL CONDITIONS UNDER HEALTH INSURANCE—VARIOUS STUDIES, 1973–74**. Single copy, $4.00; 10–49 copies, $3.80 each; 50 or more copies, $3.20 each.

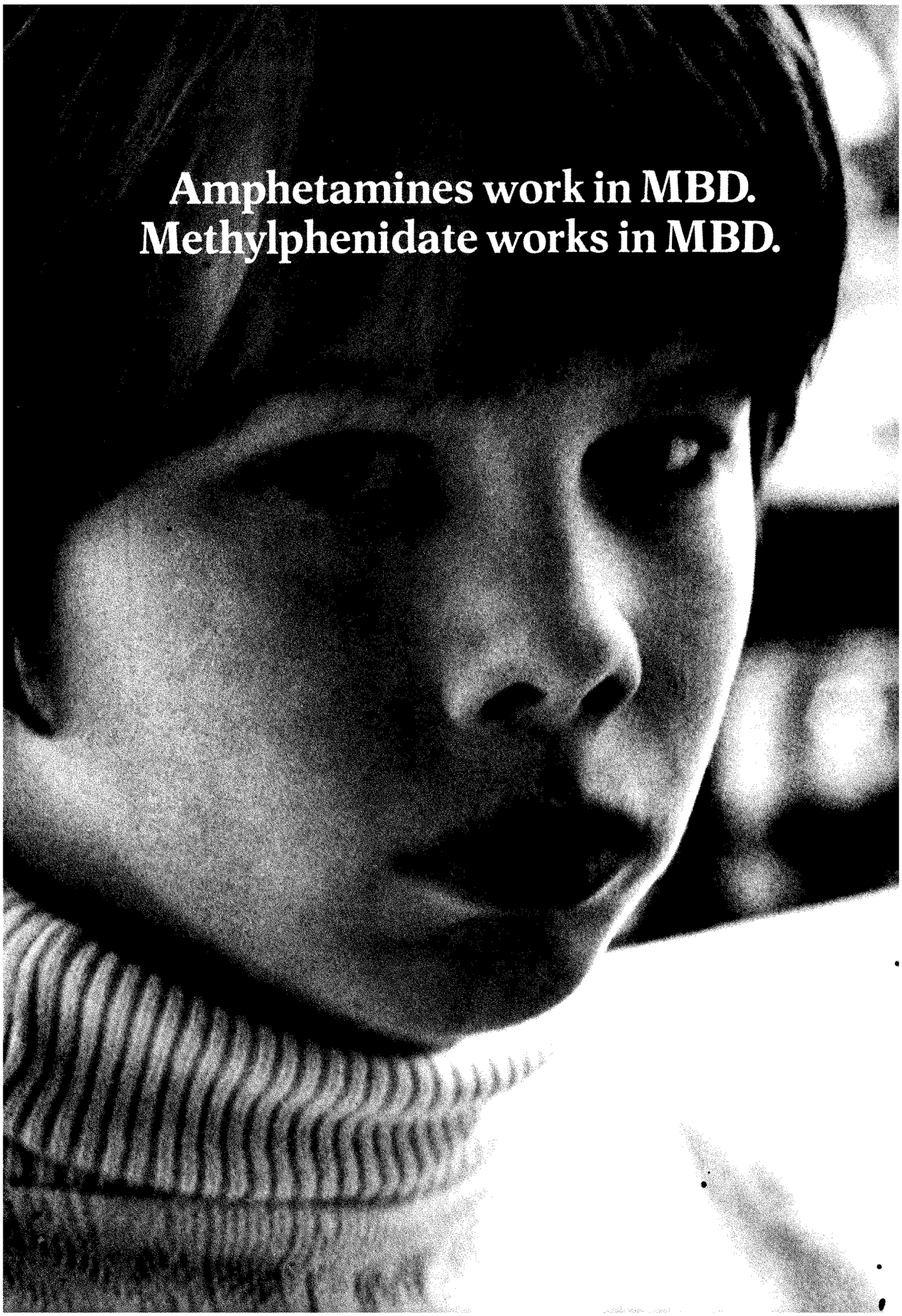
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Amphetamines work in MBD.
Methylphenidate works in MBD.

Then why Cylert® for MBD?

(pemoline)

Because Cylert offers a lot:

- Once-a-day dosage at home
- Elimination of mid-day school dose and need for school personnel supervision
- Avoids ups and downs of drug action brought about by multiple daily dosage
- Control of medication by the parent
- A chewable dosage form
- Less physician paper work (Cylert is in schedule IV)
- Safety and efficacy proven in extensive clinical studies*

*Copy of the Cylert Monograph available to Physicians on written request.

Dosage and administration

Cylert is administered as a single oral dose each morning.

The recommended starting dose is 37.5 mg/day. This daily dose should be gradually increased by 18.75 mg at one week intervals until the desired clinical response is obtained. The effective daily dose for most patients will range from 56.25 to 75 mg. The maximum recommended daily dose of pemoline is 112.5 mg.

Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally (once or twice a year) to determine if behavioral symptoms are sufficient to require continued therapy.

When not to use medication

Cylert should not be used for (and will not be effective in) simple cases of overactivity in school age children.

Neither should it be used in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

The physician should rely on a complete history of the child and a thorough description of symptoms from both parents and teacher before postulating a diagnosis of MBD.

ABBOTT

Please see next page for Prescribing Information.

7083202 R1

Cylert and Cylert Chewable Tablets

(pemoline)

Prescribing Information

Description: Cylert (pemoline) is a central nervous system stimulant. Pemoline is structurally dissimilar to the amphetamines and methylphenidate.

It is an oxazolidine compound and is chemically identified as 2-amino-5-phenyl-2-oxazolin-4-one.

Pemoline is a white, tasteless, odorless powder, relatively insoluble (less than 1 mg./ml.) in water, chloroform, ether, acetone, and benzene; its solubility in 95% ethyl alcohol is 2.2 mg./ml.

Cylert (pemoline) is supplied as tablets for oral administration.

Clinical Pharmacology: Cylert (pemoline) has a pharmacological activity similar to that of other known central nervous system stimulants; however, it has minimal sympathomimetic effects. Although studies indicate that pemoline may act in animals through dopaminergic mechanisms, the exact mechanism and site of action of the drug in man is not known.

The serum half-life of pemoline is approximately 12 hours. Peak serum levels of the drug occur within 2 to 4 hours after ingestion of a single dose. Multiple dose studies in adults at several dose levels indicate that steady state is reached in approximately 2 to 3 days.

Metabolites of pemoline include pemoline conjugate, pemoline dione, mandelic acid, and unidentified polar compounds. Cylert is excreted primarily by the kidneys; approximately 75% of an oral dose is recovered in the urine within 24 hours. Approximately 43% of pemoline is excreted unchanged.

Cylert (pemoline) has a gradual onset of action in children with minimal brain dysfunction. Using the recommended schedule of dosage titration, significant clinical benefit may not be evident until the third or fourth week of drug administration.

Indications: *MINIMAL BRAIN DYSFUNCTION IN CHILDREN*—as adjunctive therapy to other remedial measures (psychological, educational, social).

Special Diagnostic Considerations: The cause of minimal brain dysfunction (MBD) is unknown. Diagnosis of MBD involves the use of medical, psychological, educational, and social tools, since no single diagnostic test is adequate.

MBD is characterized by chronic moderate to severe hyperactivity, short attention span, distractibility, emotional lability, and impulsivity. Nonlocalizing (soft) neurological signs, learning disability, and abnormal EEG may or may not be present. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. In the primary therapy of MBD, appropriate educational placement is essential and psychosocial intervention is generally necessary. When these measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis.

Contraindications: Cylert (pemoline) is contraindicated in patients with known hypersensitivity or idiosyncrasy to the drug. (See ADVERSE REACTIONS.)

Warnings: Cylert is not recommended for children less than 6 years of age since its safety and efficacy in this age group have not been established.

Sufficient data on the safety and the efficacy of the long-term use of Cylert in children with minimal brain dysfunction are not yet available.

A temporary suppression of the predicted growth rate (weight and/or height gain) has been reported for children receiving long-term stimulant therapy. A definite causal relationship between stimulant drugs and this finding has not been established.

Precautions: Liver function tests should be performed periodically during therapy with Cylert. The drug should be discontinued if abnormalities are revealed and confirmed by follow-up tests. (See ADVERSE REACTIONS regarding reports of abnormal liver function tests and jaundice.)

Cylert should be administered with caution to patients with significantly impaired hepatic or renal function.

The interaction of Cylert with other drugs has not been studied in humans. Patients who are receiving Cylert concurrently with other drugs, especially drugs with CNS activity, should be monitored carefully.

Cylert failed to demonstrate a potential for self-administration in primates. However, the pharmacologic similarity of pemoline to other psychostimulants with known dependence liability suggests that psychological and/or physical dependence might also occur with Cylert. There have been isolated reports of transient psychotic symptoms occurring in adults following the long-term misuse of excessive oral doses of pemoline. Cylert should be given with caution to emotionally unstable patients who may increase the dosage on their own initiative.

Usage during Pregnancy and Lactation: The safety of Cylert (pemoline) for use during pregnancy and lactation has not been established.

Fertility, reproduction, and teratology studies were conducted in laboratory animals. Pemoline, in doses of 18.75 or 37.5 mg./kg./day, had no effect on the fertility of male or female rats. The drug, when given to pregnant rats (from gestation day 15 through weaning) and to rabbits (from gestation days 6-18) at these same dosage levels, produced no teratogenic or embryotoxic effects, and had no effect on the viability of the young at birth. However, increased incidences of stillbirths and cannabilization were observed when pemoline was given to rats at these dosage levels, beginning 14 days prior to conception.

Adverse Reactions: Insomnia is the most frequently reported side effect of Cylert; it usually occurs early in therapy, prior to an optimum therapeutic response. In the majority of cases it is transient in nature or responds to a reduction in dosage.

Anorexia with weight loss may occur during the first weeks of therapy. In the majority of cases it is transient in nature; weight gain usually resumes within three to six months.

Stomach ache, skin rashes, increased irritability, mild depression, nausea, dizziness, headache, drowsiness, and hallucinations have been reported.

Elevations of SGOT, SGPT, and serum LDH have occurred in patients taking Cylert, usually after several months of therapy. These effects appear to be reversible upon withdrawal of the drug, and are thought to be manifestations of a delayed hypersensitivity reaction. There have also been a few reports of jaundice occurring in patients taking Cylert; a causal relationship between the drug and this clinical finding has not been established.

There have been reports of dyskinetic movements of the lips, face, and extremities occurring with the use of Cylert. Convulsive seizures have also been reported. A definite causal relationship between Cylert and these reactions has not been established.

Mild adverse reactions appearing early during the course of treatment with Cylert often remit with continuing therapy. If adverse reactions are of a significant or protracted nature, dosage should be reduced or the drug discontinued.

Overdosage: Signs and symptoms of acute Cylert overdosage may include agitation, restlessness, hallucinations, tachycardia. The treatment for an acute overdosage of pemoline is essentially the same as that for an overdosage of any CNS stimulant. Management is primarily symptomatic and may include induction of emesis or gastric lavage, sedation, and other appropriate supportive measures.

Results of preliminary studies in dogs indicate that extracorporeal hemodialysis may be useful in the management of Cylert overdosage; forced diuresis and peritoneal dialysis appear to be of little value.

Dosage and Administration: Cylert (pemoline) is administered as a single oral dose each morning.

The recommended starting dose is 37.5 mg./day. This daily dose should be gradually increased by 18.75 mg. at one week intervals until the desired clinical response is obtained. The effective daily dose for most patients will range from 56.25 to 75 mg. The maximum recommended daily dose of pemoline is 112.5 mg.

Clinical improvement with Cylert is gradual. Using the recommended schedule of dosage titration, significant benefit may not be evident until the third or fourth week of drug administration. Drug administration should be interrupted occasionally (once or twice a year) to determine if behavioral symptoms are sufficient to require continued therapy.

How Supplied: Cylert (pemoline) is supplied as monogrammed, grooved tablets in three dosage strengths:

18.75 mg. tablets (yellow-colored) in bottles of 100 (NDC 0074-6025-13)

37.5 mg. tablets (orange-colored) in bottles of 100 (NDC 0074-6057-13)

75 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-6073-13)

Cylert Chewable is supplied as monogrammed, grooved tablets in one dosage strength:

37.5 mg. tablets (orange-colored) in bottles of 100 (NDC 0074-6088-13)

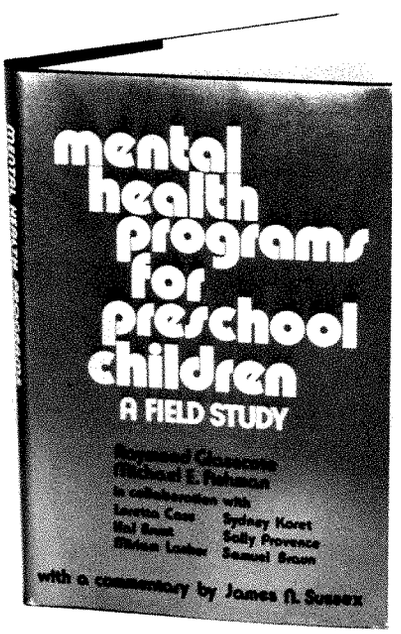
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A Timely New Book from The Joint Information Service

This latest Joint Information Service field study provides a concise, comprehensive overview of the complex matter of the very young American child and his mental health, and then describes in intimate detail seven unique programs for pre-school children with problems—from North Hollywood's Dubnoff Center, emphasizing education as its primary therapeutic modality, to Baltimore's Martin Luther King, Jr. Parent-Child Center, serving two housing projects, served by Johns Hopkins, and largely governed by its own people. All different in treatment approach and philosophy, the programs described are uniform in their dedication to innovation in improving service to the emotionally disturbed very young child, and, equally, to the emotional well being of all young children.

This thoughtful and perceptive description of what has been created by some of the best informed, most experienced and thoughtful specialists in mental health services for very young children should be welcome to all involved in the mental health of children and a casebook for those dealing with child and family mental health.



An articulate exposition of the immense problems and encouraging accomplishments in a particularly difficult area of endeavor . . . . Beyond their concern with the manifestly ill child, the authors intriguingly explore what may be needed to enhance the mental good health of all young children . . . . A significant contribution.

James N. Sussex, M.D.
President, American Association of Psychiatric Services for Children

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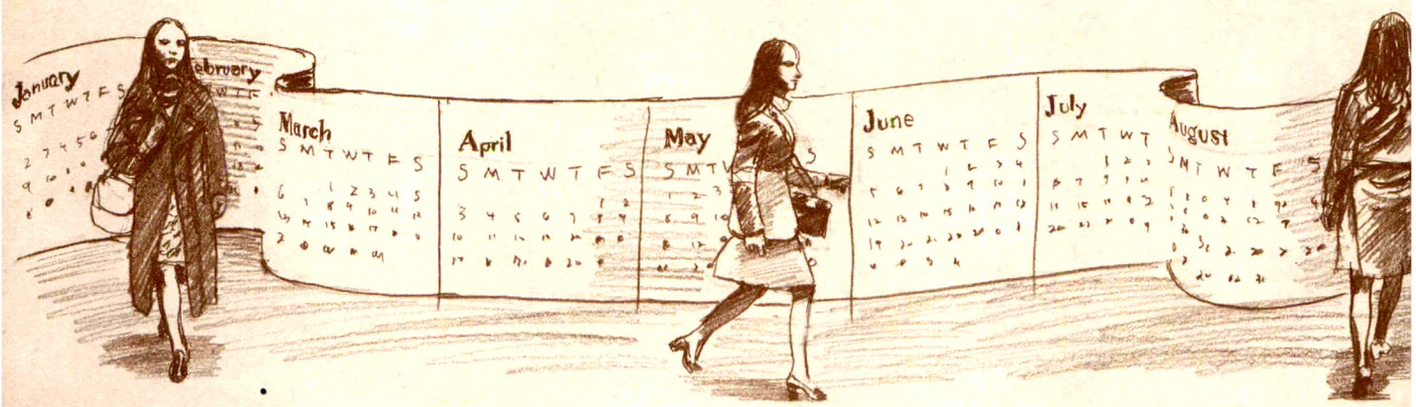
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Why Endep® makes
with amitriptyline HC



Scored tablets h.s. enhance compliance and tolerability

Once you select the appropriate maintenance dose, you can prescribe a single tablet of Endep h.s. secure in the knowledge that you can adjust the dose by a half tablet without requiring the patient to obtain a new Rx. The simplicity of this h.s. schedule not only encourages compliance, but lessens the likelihood of anticholinergic and sedative side effects during daytime.

Scored tablets h.s. reduce confusion

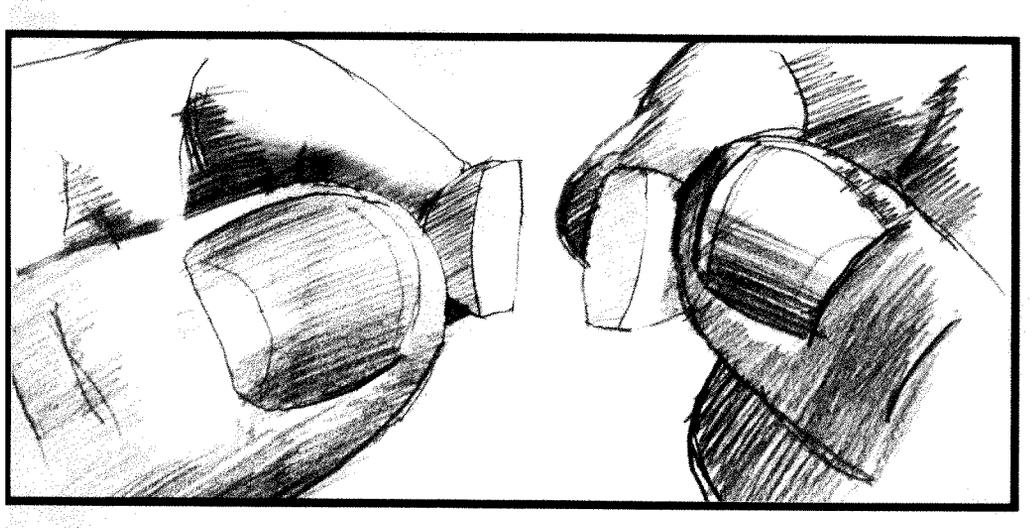
Bedtime dosage means that the patient has only one dose of Endep to remember, thus minimizing possible confusion with other medications taken concomitantly. The built-in flexibility of the scored tablet permits half-strength dosage adjustments without confusing the patient with a new color or size of medication.

ROCHE

ong-term maintenance nore practical:

Scored tablets h.s. promote economy

The economy of Endep (amitriptyline HCl) is particularly evident during long-term maintenance therapy, when your patient is purchasing refills of the single strength you have prescribed. The h.s. schedule enables you to prescribe a single strength in place of a more expensive multiple-dose prescription. The scored tablet permits you to adjust the regimen without putting the patient through the expense of a new prescription fee, without wasting tablets and without necessitating the purchase of more costly multiple-dose refills.



Endep®

amitriptyline HCl / Roche

The half-strength advantage

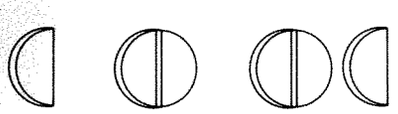
Before prescribing, please see following page for a summary of product information.

Practicality is a fundamental fact about Endep®

amitriptyline HCl/Roche

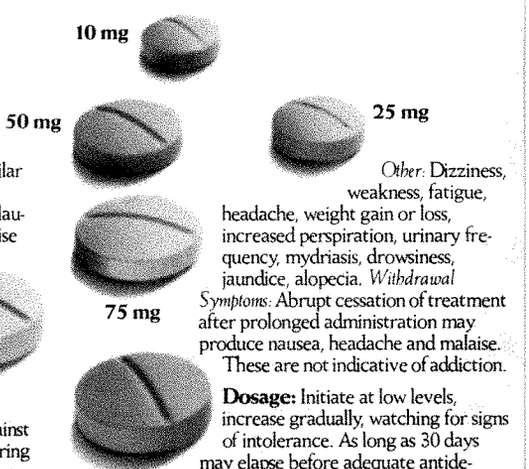
Every tablet of Endep (amitriptyline HCl) is scored for precise, convenient adjustment of dosage.

Half-tablet adjustment of dosage does not require a new Rx.



Single daily *h.s.* dosage promotes compliance and economy.

Compliance with practical regimen promotes optimal response.



Warnings: May block action of guanethidine or similar antihypertensives. Use with caution in patients with history of seizures, urinary retention, angle-closure glaucoma, increased intraocular pressure. Closely supervise cardiovascular patients, hyperthyroid patients and those receiving thyroid medications. (Arrhythmias, sinus tachycardia and prolongation of conduction time reported with use of tricyclic antidepressants, including amitriptyline HCl, especially in high doses. Myocardial infarction and stroke reported with use of this class of drugs.) May impair alertness; warn against hazardous occupations or driving a motor vehicle during therapy. Weigh possible benefits against hazards during pregnancy, the nursing period and in women of childbearing potential. Not recommended in children under 12.

Precautions: May exaggerate symptoms in schizophrenic and paranoid patients, or shift manic-depressives to manic stage; reduce dose or administer major tranquilizer concomitantly. Close supervision and careful dose adjustments required when given with anticholinergic or sympathomimetic agents. Exercise care in patients receiving large doses of ethchlorvynol; transient delirium reported with concomitant administration. May enhance effects of alcohol, barbiturates and other CNS depressants. Because of the possibility of suicide in depressed patients, do not permit easy access to large drug quantities in these patients. Because it may increase hazards of electroshock therapy, limit concomitant use to essential treatment. If possible, discontinue drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: This list includes a few adverse reactions not reported with this specific drug but requiring consideration because of similarities of tricyclic antidepressants. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue. *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction.

Dosage: Initiate at low levels, increase gradually, watching for signs of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier.

Initial Adult Dosage: Outpatients—25 mg *t.i.d.*; may be increased to 150 mg/day. Add increased drug to afternoon and/or bedtime doses. Alternate—50 to 100 mg *h.s.*, gradually increasing *h.s.* dose up to 150 mg/day. Hospitalized Patients—Up to 100 mg/day; increase gradually to 200 mg if necessary. A few patients may require 300 mg/day.

Adolescent and Elderly Patients: In general, 10 mg *t.i.d.* with 20 mg *h.s.* may be satisfactory for those who do not tolerate higher doses.

Maintenance Dosage: With symptomatic improvement, reduce dosage to lowest amount that gives relief, usually 25 mg *b.i.d.* to *q.i.d.*, or 10 mg *q.i.d.* Continue maintenance therapy 3 months or longer to avoid relapse.

Overdosage: Immediately hospitalize patient suspected of having taken an overdose. Treatment is symptomatic and supportive. IV administration of 1 to 3 mg physostigmine salicylate reported to reverse the symptoms of amitriptyline poisoning. See complete product information for manifestations and treatment.

Supplied: 10-mg, 25-mg, 50-mg, 75-mg, 100-mg and 150-mg scored tablets—bottles of 100 and 500; Tel-E-Dose® packages of 100; Prescription Paks of 60 (10 mg, 25 mg and 50 mg) or 30 (75 mg, 100 mg and 150 mg), available singly and in trays of 10.

Before prescribing, please consult complete product information, a summary of which follows:

Indications: To relieve symptoms of depression (notably endogenous depression) and depression accompanied by anxiety.

Contraindications: Known hypersensitivity. Do not use with monoamine oxidase (MAO) inhibitors or within at least 14 days following discontinuation of MAO inhibitors since hyperpyretic crises, severe convulsions and deaths have occurred with concomitant use; then initiate cautiously, gradually increasing dosage until optimal response is achieved. Use not recommended during acute recovery phase after myocardial infarction.

ROCHE

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"I'm just a shadow of what I used to be."

That is what depressed individuals may feel is the substance of their being. There is no pleasure, no joy – nothing grows – and in the cold shadow of depression their activities are inhibited, while initiative may be eroded or destroyed. The tragedy is that they can see that others are able to live on the brighter side but they, themselves, cannot reach it on their own.

Your experience in treating depression, and Tofranil-**PM** can help light the way.

Tofranil-**PM**®
imipramine **pamoate**

Geigy

Unsurpassed effectiveness among tricyclics in relieving symptoms of anxious depression.

Before prescribing Tofranil-PM, please review a summary of the prescribing information on the back of this page.

Tofranil-PM®
imipramine pamoate

Geigy

As anxiety, agitation, sleep disturbances, and other depressive symptoms are relieved, mood and motivation may be markedly improved.

Patients are usually alert and capable of functioning at more normal levels of behavior.

Tofranil-PM encourages patient compliance because one capsule lasts from bedtime to bedtime.

Good results are usually seen at the starting dose of one 75-mg capsule h.s.

For many patients, dosage can be safely increased to 150 mg daily.

As with all tricyclics, sedation may occur. Please caution patients against driving or operating dangerous machinery.

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)
(B) 98-146-840-A(9/75) 667120

For complete details, including dosage and administration, please refer to the full prescribing information.

GEIGY Pharmaceuticals
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Tranxene® CV CAPSULES SINGLE DOSE TABLETS

CLORAZEPATE DIPOTASSIUM 4306CB **Brief Summary**

ACTIONS—Pharmacologically, TRANXENE (clorazepate dipotassium) has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine.

INDICATIONS—TRANXENE is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of TRANXENE for long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS—TRANXENE is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS—TRANXENE is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on TRANXENE should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since TRANXENE has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, TRANXENE is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:

Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:

An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:

TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS—In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on TRANXENE for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS—The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION—

For symptomatic relief of anxiety:

TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For symptomatic relief of acute alcohol withdrawal:

Recommended schedule: 1st 24 hours, 30 mg TRANXENE initially, followed by 30 to 60 mg in divided doses; 2nd 24 hours, 45 to 90 mg in divided doses; 3rd 24 hours, 22.5 to 45 mg in divided doses; 4th day, 15 to 30 mg in divided doses. Thereafter gradually reduce to 7.5 to 15 mg daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS—If TRANXENE is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that TRANXENE prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If TRANXENE (clorazepate dipotassium) is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE—Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of TRANXENE as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed, since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of TRANXENE overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to TRANXENE.

CLINICAL PHARMACOLOGY—Studies in healthy men have shown that TRANXENE has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption–Excretion:

After oral administration of TRANXENE, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-TRANXENE, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED—TRANXENE (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).

7.5 mg. capsules (gray with maroon cap) in bottles of 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3418-11).

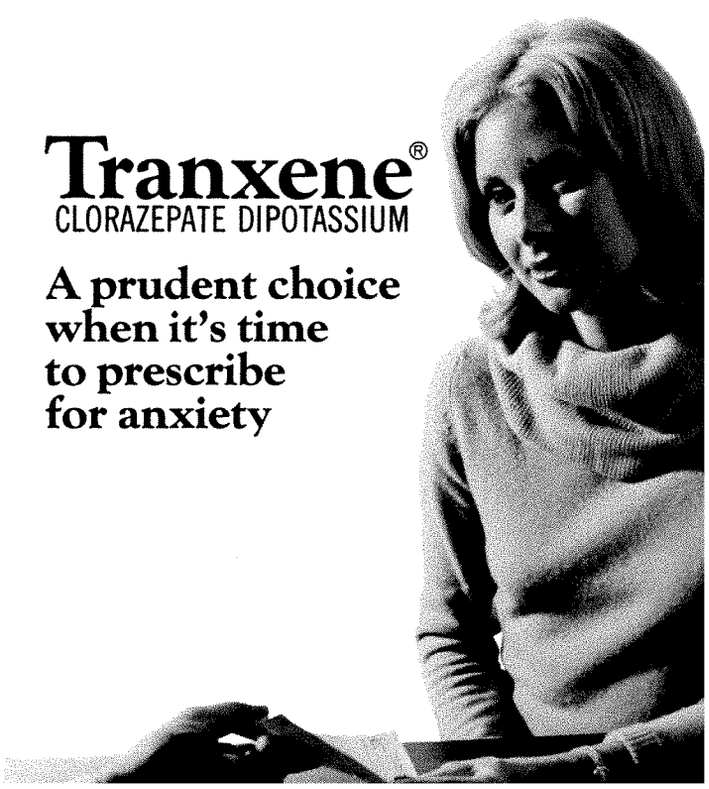
15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3419-11).

TRANXENE-SD single dose tablets are supplied in two dosage strengths:

TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 100 (NDC 0074-2699-13).

7043191/R



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A different tranquilizer. Times change. Today's patient has changed. And the physician's most prudent choice of therapy for anxious patients may also change. Thus if you are now thinking about changing your choice of minor tranquilizers, consider Tranxene.

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Tranxene® 4306CB C IV

CLORAZEPATE DIPOTASSIUM

Maybe it's time?

ABBOTT

7043191



Please see overleaf for brief summary of prescribing information.

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27378

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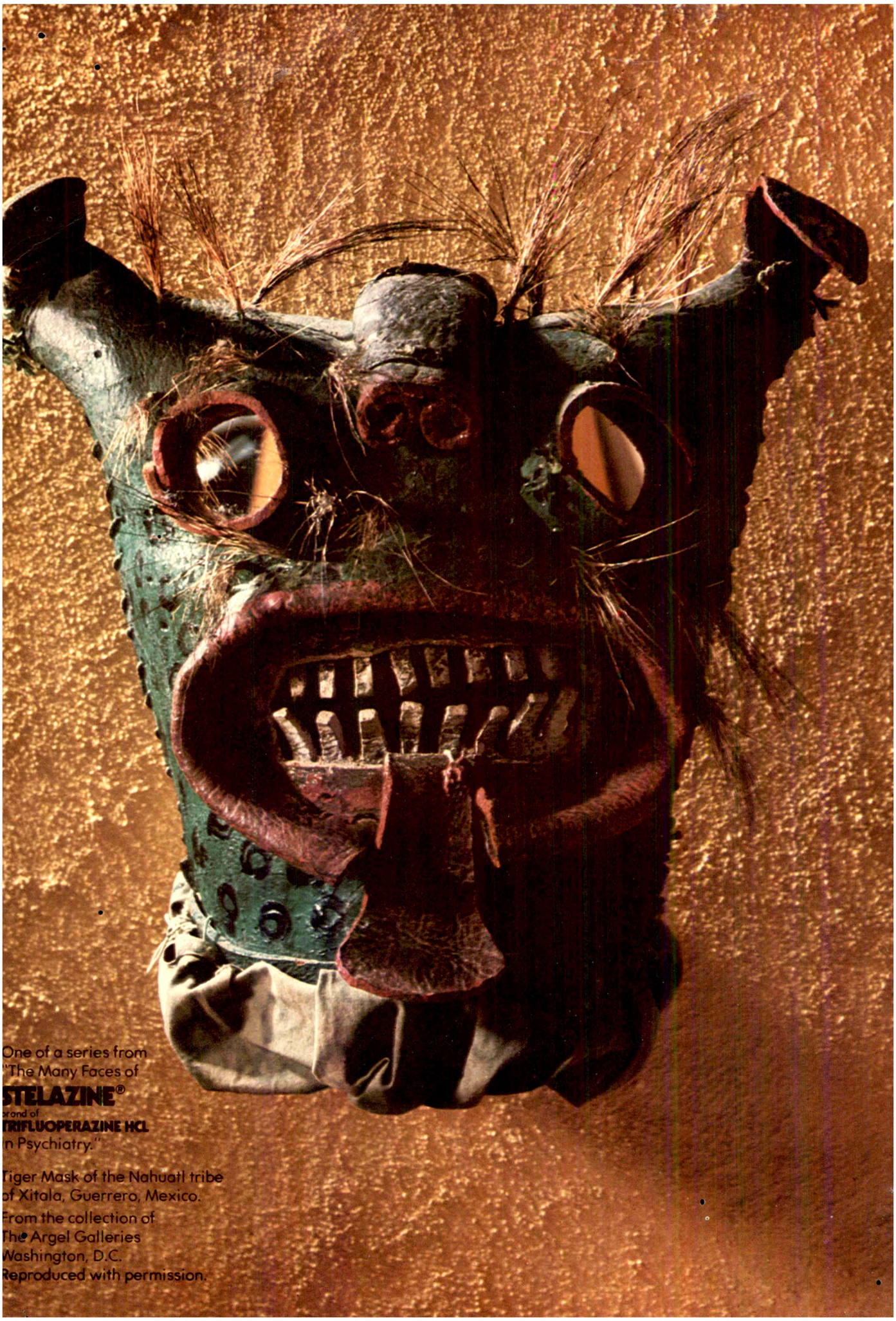
In this issue

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of Xitala, Guerrero, Mexico.
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REMOVE THE MASK OF SCHIZOPHRENIC SYMPTOMS

'STELAZINE' PROVIDES EFFECTIVE CONTROL OF HALLUCINATIONS, DELUSIONS, ANXIETY, AGITATION AND OTHER SCHIZOPHRENIC SYMPTOMS.

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- EFFECTIVELY CONTROLS PSYCHOTIC SYMPTOMS
- SELDOM CAUSES EXCESSIVE SEDATION
- CONVENIENT B.I.D. DOSAGE

Before prescribing, see complete prescribing information in SK&F literature or *PDR*. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of the manifestations of psychotic disorders.

Possibly effective: To control excessive anxiety, tension and agitation as seen in neuroses or associated with somatic conditions.

'Stelazine' has not been shown effective in the management of behavioral complications in patients with mental retardation.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose or greatly depressed states due to C.N.S. depressants; blood dyscrasias; bone marrow depression; liver damage.

Warnings: Caution patients about activities requiring alertness (e.g., operating vehicles or machinery), especially during the first few days' therapy.

Use in pregnancy only when necessary for patient's welfare.

Precautions: Use cautiously in angina. Avoid high doses and parenteral administration when cardiovascular system is impaired. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Additive effect is possible with other C.N.S. depressants. Prolonged administration of high doses may result in cumulative effects with severe C.N.S. or vasomotor symptoms. If retinal changes occur, discontinue drug. Agranulocytosis, thrombocytopenia, pancytopenia, anemia, cholestatic jaundice, liver damage have been reported.

Adverse Reactions: Drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, lactation, blurred vision. Neuromuscular (extrapyramidal) reactions: motor restlessness, dystonias, pseudoparkinsonism, persistent tardive dyskinesia.

Other adverse reactions reported with Stelazine (trifluoperazine HCl, SK&F) or other phenothiazines: Some adverse effects are more frequent or intense in specific disorders (e.g., mitral insufficiency or pheochromocytoma).

Grand mal convulsions; altered cerebrospinal fluid proteins; cerebral edema; prolongation and intensification of the action of C.N.S. depressants, atropine, heat, and organophosphorus insecticides; nasal congestion, headache, nausea, constipation, obstipation, adynamic ileus, inhibition of ejaculation; reactivation of psychotic processes, catatonic-like states; hypotension (sometimes fatal); cardiac arrest; leukopenia, eosinophilia, pancytopenia, agranulocytosis, thrombocytopenic purpura; jaundice, biliary stasis; menstrual irregularities, galactorrhea, gynecomastia, false positive pregnancy tests; photosensitivity, itching, erythema, urticaria, eczema up to exfoliative dermatitis; asthma, laryngeal edema, angioneurotic edema, anaphylactoid reactions; peripheral edema; reversed epinephrine effect; hyperpyrexia; a systemic lupus erythematosus-like syndrome; pigmentary retinopathy; with prolonged administration of substantial doses, skin pigmentation, epithelial keratopathy, and lenticular and corneal deposits. EKG changes have been reported, but relationship to myocardial damage is not confirmed. Discontinue long-term, high-dose therapy gradually. NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 1 mg., 2 mg., 5 mg. and 10 mg., in bottles of 100; in Single Unit Packages of 100 (intended for institutional use only); Injection, 2 mg./ml.; and Concentrate (intended for institutional use only), 10 mg./ml.

SK&F CO.
a SmithKline company

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3. Blackwell B, Marley E, Price J, et al: Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. Br J Psychiatry 113:349–365, 1967
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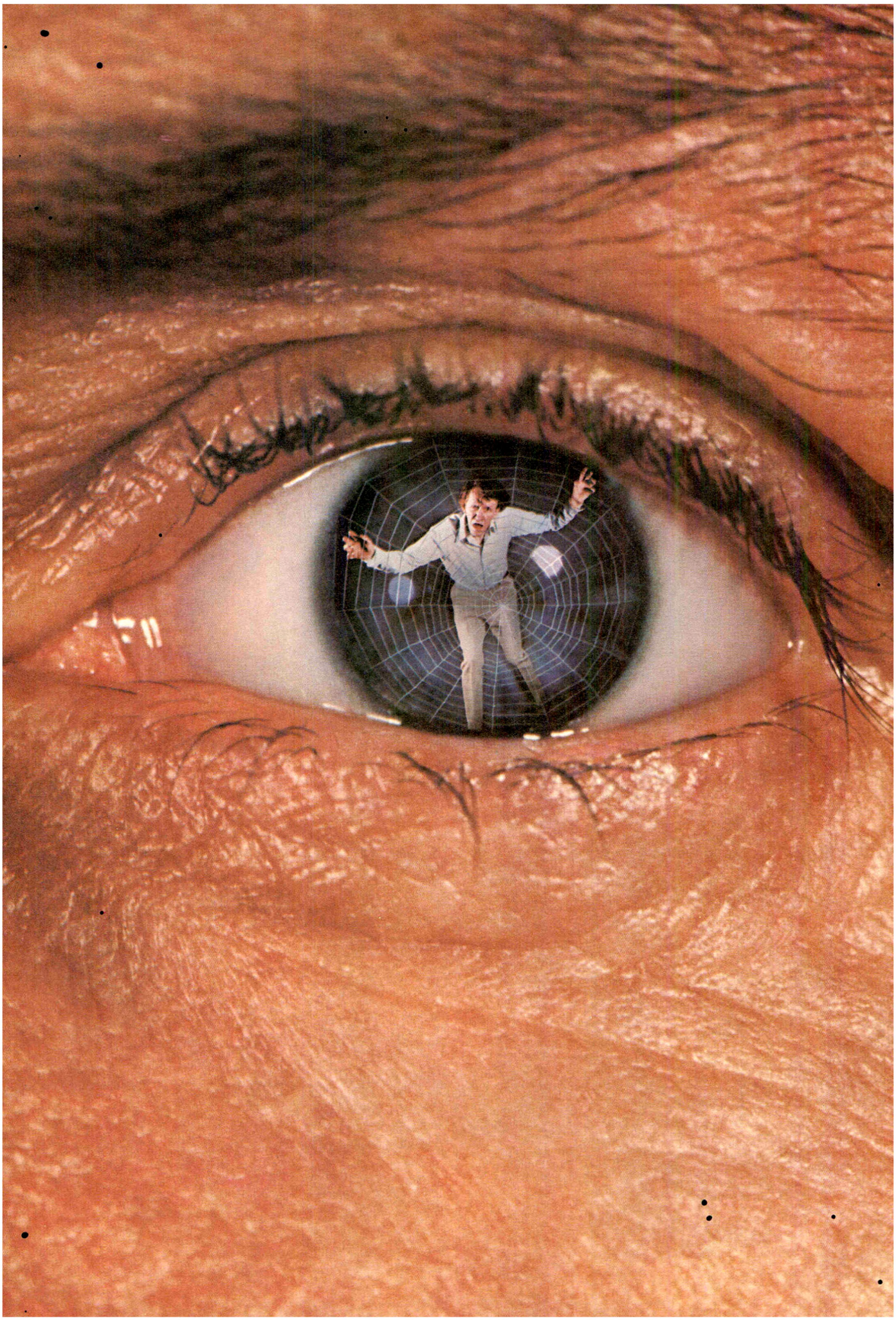
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Actions. Navane is a psychotropic agent of the thioxanthene series. Navane possesses certain chemical and pharmacological similarities to the piperazine phenothiazines and differences from the aliphatic group of phenothiazines. Navane's mode of action has not been clearly established.

Indications. Navane is effective in the management of manifestations of psychotic disorders.

Contraindications. Navane is contraindicated in patients with circulatory collapse, comatose states, central nervous system depression due to any cause, and blood dyscrasias. Navane is contraindicated in individuals who have shown hypersensitivity to the drug. It is not known whether there is a cross-sensitivity between the thioxanthenes and the phenothiazine derivatives, but this possibility should be considered.

Warnings. *Usage in Pregnancy* – Safe use of Navane during pregnancy has not been established. Therefore, this drug should be given to pregnant patients only when, in the judgment of the physician, the expected benefits from the treatment exceed the possible risks to mother and fetus. Animal reproduction studies and clinical experience to date have not demonstrated any teratogenic effects.

In the animal reproduction studies with Navane, there was some decrease in conception rate and litter size, and an increase in resorption rate in rats and rabbits, changes which have been similarly reported with other psychotropic agents. After repeated oral administration to rats (5 to 15 mg/kg/day), rabbits (3 to 50 mg/kg/day), and monkeys (1 to 3 mg/kg/day) before and during gestation, no teratogenic effects were seen. (See Precautions.)

Usage in Children–The use of Navane in children under 12 years of age is not recommended because safety and efficacy in the pediatric age group have not been established.

As is true with many CNS drugs, Navane may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as driving a car or operating machinery, especially during the first few days of therapy. Therefore, the patient should be cautioned accordingly.

As in the case of other CNS-acting drugs, patients receiving Navane should be cautioned about the possible additive effects (which may include hypotension) with CNS depressants and with alcohol.

Precautions. An antiemetic effect was observed in animal studies with Navane; since this effect may also occur in man, it is possible that Navane may mask signs of overdosage of toxic drugs and may obscure conditions such as intestinal obstruction and brain tumor.

In consideration of the known capability of Navane and certain other psychotropic drugs to precipitate convulsions, extreme caution should be used in patients with a history of convulsive disorders or those in a state of alcohol withdrawal since it may lower the convulsive threshold. Although Navane potentiates the actions of the barbiturates, the dosage of the anticonvulsant therapy should not be reduced when Navane is administered concurrently.

Caution as well as careful adjustment of the dosage is indicated when Navane is used in conjunction with other CNS depressants other than anticonvulsant drugs.

Though exhibiting rather weak anticholinergic properties, Navane should be used with caution in patients who are known or suspected to have glaucoma, or who might be exposed to extreme heat, or who are receiving atropine or related drugs.

Use with caution in patients with cardiovascular disease.

Also, careful observation should be made for pigmentary retinopathy, and lenticular pigmentation (fine lenticular pigmentation has been noted in a small number of patients treated with Navane for prolonged periods). Blood dyscrasias (agranulocytosis, pancytopenia, thrombocytopenic purpura), and liver damage (jaundice, biliary stasis) have been reported with related drugs.

Undue exposure to sunlight should be avoided. Photosensitive reactions have been reported in patients on Navane.

Intramuscular Administration – As with all intramuscular preparations, Navane Intramuscular should be injected well within the body of a relatively large muscle. The preferred sites are the upper outer quadrant of the buttock (i.e., gluteus maximus) and the mid-lateral thigh.

The deltoid area should be used only if well developed, such as in certain adults and older children, and then only with caution to avoid radial nerve injury. Intramuscular injections should not be made into the lower and mid-thirds of the upper arm. As with all intramuscular injections, aspiration is necessary to help avoid inadvertent injection into a blood vessel.

Adverse Reactions. *Note:* Not all of the following adverse reactions have been reported with Navane (thiothixene). However, since Navane has certain chemical and pharmacologic similarities to the phenothiazines, all of the known side effects and toxicity associated with phenothiazine therapy should be borne in mind when Navane is used.

Cardiovascular effects: Tachycardia, hypotension, lightheadedness, and syncope. In the event hypotension occurs, epinephrine should not be used as a pressor agent since a paradoxical further lowering of blood pressure may result. Nonspecific EKG changes have been observed in some patients receiving Navane. These changes are usually reversible and frequently disappear on continued Navane therapy. The incidence of these changes is lower than that observed with some phenothiazines. The clinical significance of these changes is not known.

CNS effects: Drowsiness, usually mild, may occur although it usually subsides with continuation of Navane therapy. The incidence of sedation appears similar to that of the piperazine group of phenothiazines, but less than that of certain aliphatic phenothiazines. Restlessness, agitation and insomnia have been noted with Navane. Seizures and paradoxical exacerbation of psychotic symptoms have occurred with Navane infrequently.

Hyperreflexia has been reported in infants delivered from mothers having received structurally related drugs.

In addition, phenothiazine derivatives have been associated with cerebral edema and cerebrospinal fluid abnormalities.

Extrapyramidal symptoms, such as pseudoparkinsonism, akathisia, and dystonia have been reported. Management of these extrapyramidal symptoms depends upon the type and severity. Rapid relief of acute symptoms may require the use of an injectable antiparkinson agent. More slowly emerging symptoms may be managed by reducing the dosage of Navane and/or administering an oral antiparkinson agent.

Persistent Tardive Dyskinesia: As with all antipsychotic agents tardive dyskinesia may appear in some patients on long term therapy or may occur after drug therapy has been discontinued. The risk seems to be greater in elderly patients on high-dose therapy, especially females. The symptoms are persistent and in some patients appear to be irreversible. The syndrome is characterized by rhythmical involuntary movements of the tongue, face, mouth or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements). Sometimes these may be accompanied by involuntary movements of extremities.

There is no known effective treatment for tardive dyskinesia: antiparkinsonism agents usually do not alleviate the symptoms of this syndrome. It is suggested that all antipsychotic agents be discontinued if these symptoms appear.

Should it be necessary to reinstitute treatment, or increase the dosage of the agent, or switch to a different antipsychotic agent, the syndrome may be masked.

It has been reported that fine vermicular movements of the tongue may be an early sign of the syndrome and if the medication is stopped at that time, the syndrome may not develop.

Hepatic effects: Elevations of serum transaminase and alkaline phosphatase, usually transient, have been infrequently observed in some patients. No clinically confirmed cases of jaundice attributable to Navane have been reported.

Hematologic effects: As is true with certain other psychotropic drugs, leukopenia and leucocytosis, which are usually transient, can occur occasionally with Navane. Other antipsychotic drugs have been associated with agranulocytosis, eosinophilia, hemolytic anemia, thrombocytopenia and pancytopenia.

Allergic reactions: Rash, pruritus, urticaria, photosensitivity and rare cases of anaphylaxis have been reported with Navane. Undue exposure to sunlight should be avoided. Although not experienced with Navane, exfoliative dermatitis and contact dermatitis (in nursing personnel) have been reported with certain phenothiazines.

Endocrine disorders: Lactation, moderate breast enlargement and amenorrhea have occurred in a small percentage of females receiving Navane. If persistent, this may necessitate a reduction in dosage or the discontinuation of therapy. Phenothiazines have been associated with false positive pregnancy tests, gynecomastia, hypoglycemia, hyperglycemia, and glycosuria.

Autonomic effects: Dry mouth, blurred vision, nasal congestion, constipation, increased sweating, increased salivation, and impotence have occurred infrequently with Navane therapy. Phenothiazines have been associated with miosis, mydriasis, and adynamic ileus.

Other adverse reactions: Hyperpyrexia, anorexia, nausea, vomiting, diarrhea, increase in appetite and weight, weakness or fatigue, polydipsia and peripheral edema.

Although not reported with Navane, evidence indicates there is a relationship between phenothiazine therapy and the occurrence of a systemic lupus erythematosus-like syndrome.

NOTE: Sudden deaths have occasionally been reported in patients who have received certain phenothiazine derivatives. In some cases the cause of death was apparently cardiac arrest or asphyxia due to failure of the cough reflex. In others, the cause could not be determined nor could it be established that death was due to phenothiazine administration.

Dosage and Administration. Dosage of Navane should be individually adjusted depending on the chronicity and severity of the condition. In general, small doses should be used initially and gradually increased to the optimal effective level, based on patient response.

Some patients have been successfully maintained on once-a-day Navane therapy.

Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

Navane Intramuscular Solution–For Intramuscular Use Only. Where more rapid control and treatment of acute behavior is desirable, the intramuscular form of Navane may be indicated. It is also of benefit where the very nature of the patient's symptomatology, whether acute or chronic, renders oral administration impractical or even impossible.

For treatment of acute symptomatology or in patients unable or unwilling to take oral medication, the usual dose is 4 mg of Navane Intramuscular administered 2 to 4 times daily. Dosage may be increased or decreased depending on response. Most patients are controlled on a total daily dosage of 16 to 20 mg. The maximum recommended dosage is 30 mg/day. An oral form should supplant the injectable form as soon as possible. It may be necessary to adjust the dosage when changing from the intramuscular to oral dosage forms. Dosage recommendations for Navane Capsules and Concentrate appear in the following paragraphs.

Navane Capsules; Navane Concentrate–In milder conditions, an initial dose of 2 mg three times daily. If indicated, a subsequent increase to 15 mg/day total daily dose is often effective.

In more severe conditions, an initial dose of 5 mg twice daily.

The usual optimal dose is 20 to 30 mg daily. If indicated, an increase to 60 mg/day total daily dose is often effective. Exceeding a total daily dose of 60 mg rarely increases the beneficial response.

Overdosage. Manifestations include muscular twitching, drowsiness, and dizziness. Symptoms of gross overdosage may include CNS depression, rigidity, weakness, torticollis, tremor, salivation, dysphagia, hypotension, disturbances of gait, or coma.

Treatment: Essentially symptomatic and supportive. For Navane oral, early gastric lavage is helpful. For Navane oral and Intramuscular, keep patient under careful observation and maintain an open airway, since involvement of the extrapyramidal system may produce dysphagia and respiratory difficulty in severe overdosage. If hypotension occurs, the standard measures for managing circulatory shock should be used (I.V. fluids and/or vasoconstrictors).

If a vasoconstrictor is needed, levarterenol and phenylephrine are the most suitable drugs. Other pressor agents, including epinephrine, are not recommended, since phenothiazine derivatives may reverse the usual pressor elevating action of these agents and cause further lowering of blood pressure.

If CNS depression is present, recommended stimulants include amphetamine, dextroamphetamine, or caffeine and sodium benzoate. Picrotoxin or pentylenetetrazol should be avoided. Extrapyramidal symptoms may be treated with antiparkinson drugs.

There are no data on the use of peritoneal or hemodialysis, but they are known to be of little value in phenothiazine intoxication.

How Supplied. Navane (thiothixene) is available as capsules containing 1 mg, 2 mg, 5 mg, and 10 mg, of thiothixene in bottles of 100 and 1,000. Navane is also available as capsules containing 20 mg of thiothixene in bottles of 100 and 500.

Navane (thiothixene hydrochloride) Concentrate is available in 120 ml (4 oz.) bottles with an accompanying dropper calibrated at 2 mg, 4 mg, 5 mg, 6 mg, 8 mg, and 10 mg. Each ml contains thiothixene hydrochloride equivalent to 5 mg of thiothixene. Contains alcohol, U.S.P. 7.0% v/v (small loss unavoidable).

Navane (thiothixene hydrochloride) Intramuscular solution is available in a 2 ml amber glass vial in packages of 10. Each ml contains thiothixene hydrochloride equivalent to 2 mg of thiothixene, dextrose 5% w/v, benzyl alcohol 0.9% w/v, and propyl gallate 0.02% w/v.

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Indications: Relief of anxiety and tension occurring alone or accompanying various disease states.

Contraindications: Patients with known hypersensitivity to the drug.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. As with all CNS-acting drugs, caution patients against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving). Though physical and psychological dependence have rarely been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage; withdrawal symptoms (including convulsions), following discontinuation of the drug and similar to those seen with barbiturates, have been reported.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: In the elderly and debilitated, and in children over six, limit to smallest effective dosage (initially 10 mg or less per day) to preclude ataxia or oversedation, increasing gradually as needed and tolerated. Not recommended in children under six. Though generally not recommended, if combination therapy with other psychotropics seems indicated, carefully consider individual pharmacologic effects, particularly in use of potentiating drugs such as MAO inhibitors and phenothiazines. Observe usual precautions in presence of impaired renal or hepatic function. Paradoxical reactions (*e.g.*, excitement, stimulation and acute rage) have been reported in psychiatric patients and hyperactive aggressive children. Employ usual precautions in treatment of anxiety states with evidence of impending depression; suicidal tendencies may be present and protective measures necessary. Variable effects on blood coagulation have been reported very rarely in patients receiving the drug and oral anticoagulants; causal relationship has not been established clinically.

Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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Contraindications: Hypersensitivity. Should not be given concomitantly with MAO inhibitors or within 2 weeks of the use of this drug since hyperpyretic crises, severe convulsions, and fatalities have occurred when similar tricyclic antidepressants were used in such combinations. Cross-sensitivity with other dibenzazepines is a possibility. Contraindicated during acute recovery period after myocardial infarction.

Warnings: Use with caution in patients with cardiovascular disease because of tendency to produce sinus tachycardia and prolong conduction time. Myocardial infarction, arrhythmia, and strokes have occurred. May block antihypertensive action of guanethidine and similar agents. Because of anticholinergic activity, use cautiously in patients with glaucoma or a history of urinary retention. Patients with a history of seizures should be followed closely because the drug is known to lower the convulsive threshold. Great care is required for hyperthyroid patients and those taking thyroid medication because of possible development of cardiac arrhythmia. Caution patients about possibility of impaired mental and/or physical ability to operate a motor vehicle or dangerous machinery. Response to alcoholic beverages may be exaggerated and may lead to suicidal attempts. Safe use during pregnancy, lactation, and women of childbearing potential has not been established and the drug should not be given unless clinical situation warrants potential risk. Not recommended for use in children.

Precautions: Psychotic symptoms may be exacerbated in schizophrenic patients. Increased anxiety and agitation may occur in overactive or agitated patients. Manic-depressive patients may experience shift to manic phase. Hostility may be aroused. Concomitant administration of reserpine may produce a "stimulating" effect. Watch for possible epileptiform seizures during treatment. Use cautiously with anticholinergic or sympathomimetic drugs. Concurrent electroconvulsive therapy may increase hazards associated with nortriptyline HCl. When possible, discontinue drug several days prior to surgery. Potentially suicidal patients require supervision and protective measures during therapy. Prescriptions should be limited to the least possible quantity. Both elevation and lowering of blood sugar levels have been reported.

Adverse Reactions: Note: The pharmacologic similarities among the tricyclic antidepressant drugs require that each of the following reactions be considered when nortriptyline is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia, panic, nightmares; hypomania; exacerbation of psychosis.

Neurologic: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alteration in EEG patterns; tinnitus.

Anticholinergic: Dry mouth and rarely, associated sublingual adenitis; blurred vision, disturbance of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue), drug fever, cross-sensitivity with other tricyclic drugs.

Hematologic: Bone-marrow depression, including agranulocytosis; eosinophilia; purpura; thrombocytopenia.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea, peculiar taste, stomatitis, abdominal cramps, black-tongue.

Endocrine: Gynecomastia in the male, breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency, nocturia; drowsiness, dizziness, weakness, fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though these are not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache, and malaise.

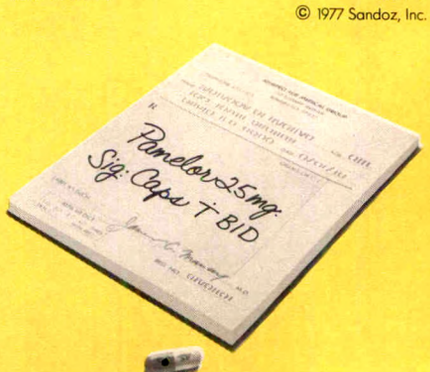
Dosage and Administration: *Usual adult dose*—25 mg. three or four times daily; dosage should begin at a low level and increase as required. *Elderly and Adolescent:* 30 to 50 mg. per day, in divided doses. Doses above 100 mg. per day and use in children are not recommended. If a patient develops minor side effects, the dosage should be reduced. The drug should be discontinued promptly if adverse effects of a serious nature or allergic manifestations occur.

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4 in sleep laboratory, 10 at home,
4 in sleep laboratory[3]

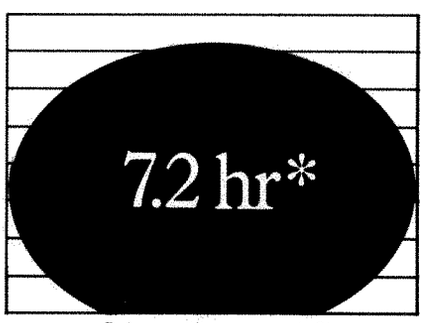
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1,5,7,14,28 nights... as needed, the effectiveness of Dalmane® one *h.s.* (flurazepam HCl) C IV meets the challenge of insomnia

One 30-mg capsule *h.s.*—usual adult dosage (15 mg may suffice in some patients).
One 15-mg capsule *h.s.*—initial dosage for elderly or debilitated patients.

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- **Patients generally awaken refreshed; morning "hangover" is infrequent**

TOTAL SLEEP TIME (OVERALL MEAN)[3]
(5 patients with insomnia)



Dalmane (flurazepam HCl)
nights 1-3, 12-14, 26-28

*Significantly increased over baseline placebo ($p < .05$).

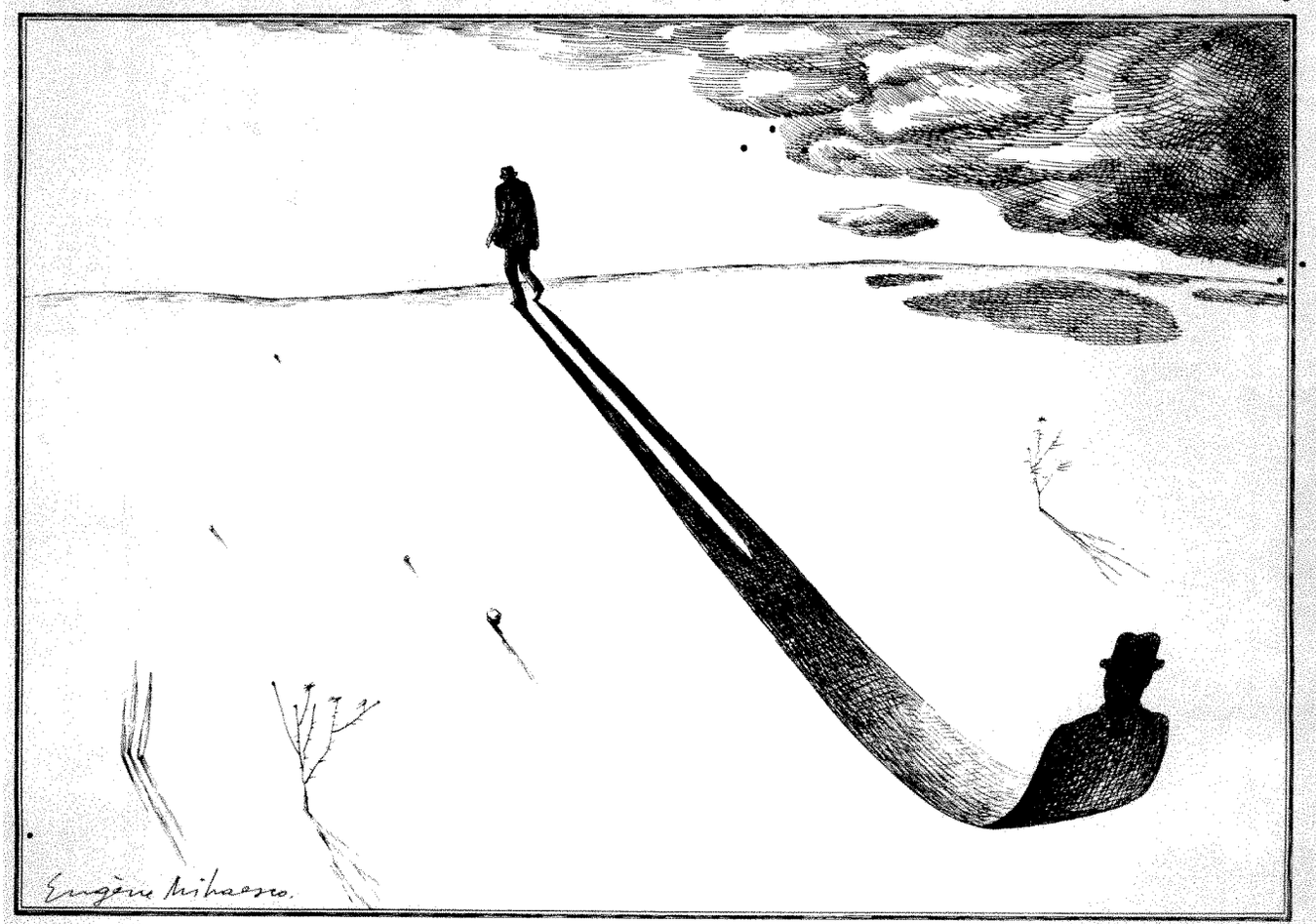
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2. Kales A, *et al*: *Clin Pharmacol Ther* *18*:356-363, Sep 1975
3. Dement WC, *et al*: Long-term effectiveness of flurazepam 30 mg *h.s.* on chronic insomniacs. Presented at the 15th annual meeting of the Association for Psychophysiological Study of Sleep, Edinburgh, Scotland, Jun 30-Jul 4, 1975

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).
***Usage in Pregnancy:* Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane (flurazepam HCl), a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

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"I'm just a shadow of what I used to be."

That is what depressed individuals may feel is the substance of their being. There is no pleasure, no joy – nothing grows – and in the cold shadow of depression their activities are inhibited, while initiative may be eroded or destroyed. The tragedy is that they can see that others are able to live on the brighter side but they, themselves, cannot reach it on their own.

Your experience in treating depression, and Tofranil-**PM** can help light the way.

Tofranil-**PM**®
imipramine **pamoate**

Geigy

Unsurpassed effectiveness among tricyclics in relieving symptoms of anxious depression.

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Tofranil-PM®
imipramine **pamoate**

Geigy

As anxiety, agitation, sleep disturbances, and other depressive symptoms are relieved, mood and motivation may be markedly improved.

Patients are usually alert and capable of functioning at more normal levels of behavior.

Tofranil-PM encourages patient compliance because one capsule lasts from bedtime to bedtime.

Good results are usually seen at the starting dose of one 75-mg capsule h.s.

For many patients, dosage can be safely increased to 150 mg daily.

As with all tricyclics, sedation may occur. Please caution patients against driving or operating dangerous machinery.

Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg of imipramine hydrochloride.

Tofranil-PM®
brand of imipramine pamoate

Indications: For the relief of symptoms of depression. Endogenous depression is more likely to be alleviated than other depressive states.

Contraindications: The concomitant use of monoamine oxidase inhibiting compounds is contraindicated. Hyperpyretic crises or severe convulsive seizures may occur in patients receiving such combinations. The potentiation of adverse effects can be serious, or even fatal. When it is desired to substitute Tofranil-PM, brand of imipramine pamoate, in patients receiving a monoamine oxidase inhibitor, as long an interval should elapse as the clinical situation will allow, with a minimum of 14 days. Initial dosage should be low and increases should be gradual and cautiously prescribed. The drug is contraindicated during the acute recovery period after a myocardial infarction. Patients with a known hypersensitivity to this compound should not be given the drug. The possibility of cross-sensitivity to other dibenzazepine compounds should be kept in mind.

Warnings: *Usage in Pregnancy:* Safe use of imipramine during pregnancy and lactation has not been established; therefore, in administering the drug to pregnant patients, nursing mothers, or women of childbearing potential, the potential benefits must be weighed against the possible hazards. Animal reproduction studies have yielded inconclusive results. There have been clinical reports of congenital malformation associated with the use of this drug, but a causal relationship has not been confirmed.

Extreme caution should be used when this drug is given to:

—patients with cardiovascular disease because of the possibility of conduction defects, arrhythmias, myocardial infarction, strokes and tachycardia;
—patients with increased intraocular pressure, history of urinary retention, or history of narrow-angle glaucoma because of the drug's anticholinergic properties;
—hyperthyroid patients or those on thyroid medication because of the possibility of cardiovascular toxicity;
—patients with a history of seizure disorder because this drug has been shown to lower the seizure threshold;
—patients receiving guanethidine or similar agents since imipramine may block the pharmacologic effects of these drugs.

Since imipramine may impair the mental and/or physical abilities required for the performance of potentially hazardous tasks such as operating an automobile or machinery, the patient should be cautioned accordingly.

Usage in Children: Tofranil-PM, brand of imipramine pamoate, should not be used in children of any age because of the increased potential for acute overdosage due to the high unit potency (75 mg., 100 mg., 125 mg. and 150 mg.). Each capsule contains imipramine pamoate equivalent to 75 mg., 100 mg., 125 mg. or 150 mg. imipramine hydrochloride.

Precautions: It should be kept in mind that the possibility of suicide in seriously depressed patients is inherent in the illness and may persist until significant remission occurs. Such patients should be carefully supervised during the early phase of treatment with Tofranil-PM, brand of imipramine pamoate, and may require hospitalization. Prescriptions should be written for the smallest amount feasible.

Hypomanic or manic episodes may occur, particularly in patients with cyclic disorders. Such reactions may necessitate discontinuation of the drug. If needed, Tofranil-PM, brand of imipramine pamoate, may be resumed in lower dosage when these episodes are relieved. Administration of a tranquilizer may be useful in controlling such episodes.

Prior to elective surgery, imipramine should be discontinued for as long as the clinical situation will allow.

An activation of the psychosis may occasionally be observed in schizophrenic patients and may require reduction of dosage and the addition of a phenothiazine.

In occasional susceptible patients or in those receiving anticholinergic drugs (including antiparkinsonism agents) in addition, the atropine-like effects may become more pronounced (e.g., paralytic ileus). Close supervision and careful adjustment of dosage is required when this drug is administered concomitantly with anticholinergic or sympathomimetic drugs.

Avoid the use of preparations, such as decongestants and local anesthetics, which contain any sympathomimetic amine (e.g., adrenalin, noradrenalin), since it has been reported that tricyclic antidepressants can potentiate the effects of catecholamines.

Patients should be warned that the concomitant use of alcoholic beverages may be associated with exaggerated effects.

Both elevation and lowering of blood sugar levels have been reported.

Concurrent administration of imipramine with electroshock therapy may increase the hazards; such treatment should be limited to those patients for whom it is essential, since there is limited clinical experience.

Adverse Reactions: Note: Although the listing which follows includes a few adverse reactions which have not been reported with this specific drug, the pharmacological similarities among the tricyclic antidepressant drugs require that each of the reactions be considered when imipramine is administered.

Cardiovascular: Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke, falls.

Psychiatric: Confusional states (especially in the elderly) with hallucinations, disorientation, delusions; anxiety, restlessness, agitation; insomnia and nightmares; hypomania; exacerbation of psychosis.

Neurological: Numbness, tingling, paresthesias of extremities; incoordination, ataxia, tremors; peripheral neuropathy; extrapyramidal symptoms; seizures, alterations in EEG patterns; tinnitus.

Anticholinergic: Dry mouth, and, rarely, associated sublingual adenitis; blurred vision, disturbances of accommodation, mydriasis; constipation, paralytic ileus; urinary retention, delayed micturition, dilation of the urinary tract.

Allergic: Skin rash, petechiae, urticaria, itching, photosensitization (avoid excessive exposure to sunlight); edema (general or of face and tongue); drug fever; cross-sensitivity with desipramine.

Hematologic: Bone marrow depression including agranulocytosis; eosinophilia; purpura; thrombocytopenia. Leukocyte and differential counts should be performed in any patient who develops fever and sore throat during therapy; the drug should be discontinued if there is evidence of pathological neutrophil depression.

Gastrointestinal: Nausea and vomiting, anorexia, epigastric distress, diarrhea; peculiar taste, stomatitis, abdominal cramps, black tongue.

Endocrine: Gynecomastia in the male; breast enlargement and galactorrhea in the female; increased or decreased libido, impotence; testicular swelling; elevation or depression of blood sugar levels.

Other: Jaundice (simulating obstructive); altered liver function; weight gain or loss; perspiration; flushing; urinary frequency; drowsiness, dizziness, weakness and fatigue; headache; parotid swelling; alopecia.

Withdrawal Symptoms: Though not indicative of addiction, abrupt cessation of treatment after prolonged therapy may produce nausea, headache and malaise.

Dosage and Administration: In adult outpatients, therapy should be initiated on a once-a-day basis with 75 mg./day. This may be increased to 150 mg./day which is the dose level which usually obtains optimum response. If necessary, dosage may be increased to 200 mg./day. Dosage should be modified as necessary by clinical response and any evidence of intolerance. Daily dosage may be given at bedtime, or in some patients in divided daily doses.

Hospitalized patients should be started on a once-a-day basis with 100-150 mg./day and may be increased to 200 mg./day. Dosage should be increased to 250-300 mg./day if there is no response after two weeks.

Following remission, maintenance medication may be required for a longer period of time at the lowest dose that will maintain remission. The usual adult maintenance dosage is 75-150 mg./day on a once-a-day basis, preferably at bedtime.

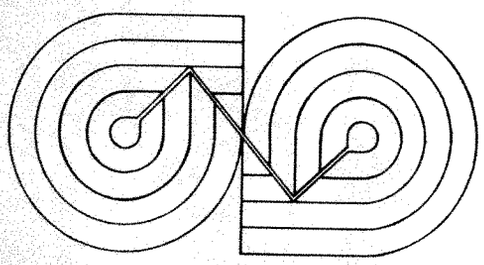
In adolescent and geriatric patients, capsules of Tofranil-PM, brand of imipramine pamoate, may be used when total daily dosage is established at 75 mg. or higher. It is generally unnecessary to exceed 100 mg./day in these patients. This dosage may be given once a day at bedtime or, if needed, in divided daily doses.

How Supplied: Tofranil-PM, brand of imipramine pamoate: Capsules of 75, 100, 125 and 150 mg. (Each capsule contains imipramine pamoate equivalent to 75, 100, 125 or 150 mg. of imipramine hydrochloride.)
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For complete details, including dosage and administration, please refer to the full prescribing information.

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"Now Andy's classmates laug



vith him, not at him"



Today, direct coaching in appropriate social interaction, role playing, and encounter techniques are being used to help hyperactive children overcome disruptive social behavior patterns that often cause them to become objects of ridicule to their peers.

Ritalin, when indicated, can also play a useful role in this respect, as improving social interaction in the MBD child often involves reducing hyperactivity. Indeed, as one investigator[1] has reported, with Ritalin therapy, the MBD child can be less aggressive, less impulsive, and better able to interact with his peers. Other studies also report improved social behavior of MBD children on Ritalin.[2,3]

A drug of choice when treatment of MBD is indicated,[4,5] controlled studies and clinical experience have demonstrated the ability of Ritalin to effectively reduce hyperactivity,[1,2] distractibility,[1,3] and disorganized behavior[1,3,6-8] in the MBD child.

And these symptomatic improvements achieved with Ritalin often result in the child being more responsive to the nonpharmacological measures of the MBD management team.[8]

Although side effects – insomnia and appetite loss – are occasionally seen with Ritalin, they are less frequent or severe than with dextroamphetamine.[4,10]

Therapy with Ritalin should be undertaken only after a medical diagnosis of MBD has been made. Dosage should be periodically interrupted. Often these interruptions reveal some "stabilization" in the child's behavior even without medication. In some MBD children they permit a reduction in dosage and eventual discontinuance of drug therapy.

Ritalin...Only when medication is indicated

Ritalin® (methylphenidate) C II

An effective member of the MBD management team



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(methylphenidate)
Only when medication is indicated

Ritalin® hydrochloride
(methylphenidate hydrochloride)
TABLETS

INDICATION

Minimal Brain Dysfunction in Children—as adjunctive therapy to other remedial measures (psychological, educational, social)

Special Diagnostic Considerations

Specific etiology of Minimal Brain Dysfunction (MBD) is unknown, and there is no single diagnostic test. Adequate diagnosis requires the use not only of medical but of special psychological, educational, and social resources.

Characteristics commonly reported include: chronic history of short attention span, distractibility, emotional lability, impulsivity, and moderate to severe hyperactivity; minor neurological signs and abnormal EEG. Learning may or may not be impaired. The diagnosis of MBD must be based upon a complete history and evaluation of the child and not solely on the presence of one or more of these characteristics.

Drug treatment is not indicated for all children with MBD. Stimulants are not intended for use in the child who exhibits symptoms secondary to environmental factors and/or primary psychiatric disorders, including psychosis. Appropriate educational placement is essential and psychosocial intervention is generally necessary. When remedial measures alone are insufficient, the decision to prescribe stimulant medication will depend upon the physician's assessment of the chronicity and severity of the child's symptoms.

CONTRAINDICATIONS

Marked anxiety, tension, and agitation, since Ritalin may aggravate these symptoms. Also contraindicated in patients known to be hypersensitive to the drug and in patients with glaucoma.

WARNINGS

Ritalin should not be used in children under six years, since safety and efficacy in this age group have not been established.

Sufficient data on safety and efficacy of long-term use of Ritalin in children with minimal brain dysfunction are not yet available. Although a causal relationship has not been established, suppression of growth (*ie*, weight gain and/or height) has been reported with long-term use of stimulants in children. Therefore, children requiring long-term therapy should be carefully monitored.

Ritalin should not be used for severe depression of either exogenous or endogenous origin or for the prevention of normal fatigue states.

Ritalin may lower the convulsive threshold in patients with or without prior seizures; with or without prior EEG abnormalities, even in absence of seizures. Safe concomitant use of anticonvulsants and Ritalin has not been established. If seizures occur, Ritalin should be discontinued.

Use cautiously in patients with hypertension. Blood pressure should be monitored at appropriate intervals in all patients taking Ritalin, especially those with hypertension.

Symptoms of visual disturbances have been encountered in rare cases. Difficulties with accommodation and blurring of vision have been reported.

Drug Interactions

Ritalin may decrease the hypotensive effect of guanethidine. Use cautiously with pressor agents and MAO inhibitors. Ritalin may inhibit the metabolism of coumarin anticoagulants, anticonvulsants (phenobarbital, diphenylhydantoin, primidone), phenylbutazone, and tricyclic antidepressants (imipramine, desipramine). Downward dosage adjustments of these drugs may be required when given concomitantly with Ritalin.

Usage in Pregnancy

Adequate animal reproduction studies to establish safe use of Ritalin during pregnancy have not been conducted. Therefore, until more information is available, Ritalin should not be prescribed for women of childbearing age unless, in the opinion of the physician, the potential benefits outweigh the possible risks.

Drug Dependence

Ritalin should be given cautiously to emotionally unstable patients, such as those with a history of drug dependence or alcoholism, because such patients may increase dosage on their own initiative.

Chronically abusive use can lead to marked tolerance and psychic dependence with varying degrees of abnormal behavior. Frank psychotic episodes can occur, especially with parenteral abuse. Careful supervision is required during drug withdrawal, since severe depression as well as the effects of chronic overactivity can be unmasked. Long-term follow-up may be required because of the patient's basic personality disturbances.

PRECAUTIONS

Patients with an element of agitation may react adversely; discontinue therapy if necessary.

Periodic CBC, differential, and platelet counts are advised during prolonged therapy.

ADVERSE REACTIONS

Nervousness and insomnia are the most common adverse reactions but are usually controlled by reducing dosage and omitting the drug in the afternoon or evening. Other reactions include: hypersensitivity (including skin rash, urticaria, fever, arthralgia, exfoliative dermatitis, erythema multiforme with histopathological findings of necrotizing vasculitis, and thrombocytopenic purpura); anorexia; nausea; dizziness; palpitations; headache; dyskinesia; drowsiness; blood pressure and pulse changes, both up and down; tachycardia; angina; cardiac arrhythmia; abdominal pain; weight loss during prolonged therapy. Toxic psychosis has been reported. Although a definite causal relationship has not been established, the following have been reported in patients taking this drug: leukopenia and/or anemia; a few instances of scalp hair loss.

In children, loss of appetite, abdominal pain, weight loss during prolonged therapy, insomnia, and tachycardia may occur more frequently; however, any of the other adverse reactions listed above may also occur.

DOSAGE AND ADMINISTRATION

Children with Minimal Brain Dysfunction (6 years and over).

Start with small doses (*eg*, 5 mg before breakfast and lunch) with gradual increments of 5 to 10 mg weekly. Daily dosage above 60 mg is not recommended. If improvement is not observed after appropriate dosage adjustment over a one-month period, the drug should be discontinued.

If paradoxical aggravation of symptoms or other adverse effects occur, reduce dosage, or, if necessary, discontinue the drug.

Ritalin should be periodically discontinued to assess the child's condition. Improvement may be sustained when the drug is either temporarily or permanently discontinued.

Drug treatment should not and need not be indefinite and usually may be discontinued after puberty.

HOW SUPPLIED

Tablets, 20 mg (peach, scored); bottles of 100 and 1000.

Tablets, 10 mg (pale green, scored); bottles of 100, 500, 1000 and Accu-Pak® blister units of 100.

Tablets, 5 mg (pale yellow); bottles of 100, 500, and 1000.

Consult complete product literature before prescribing.

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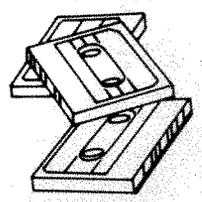
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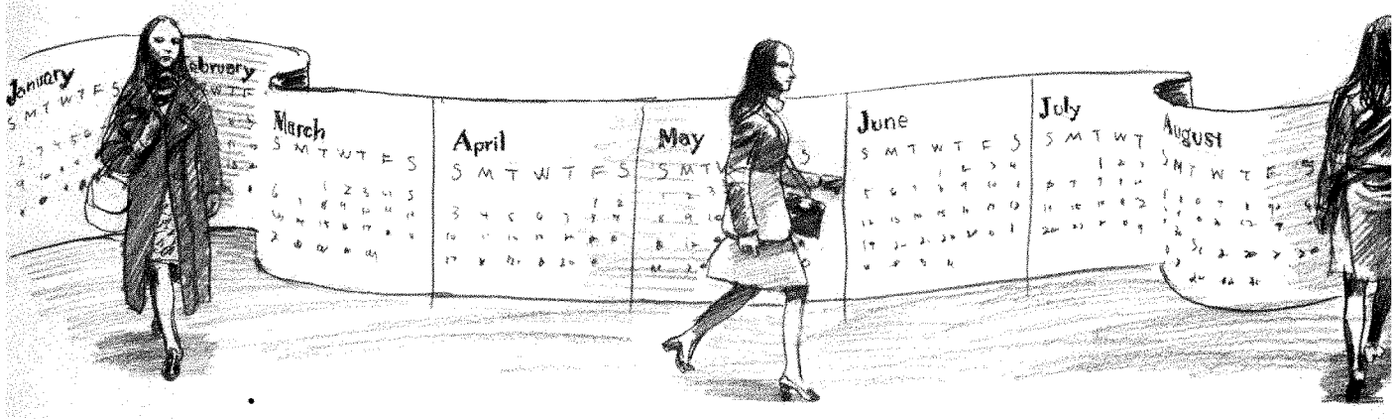
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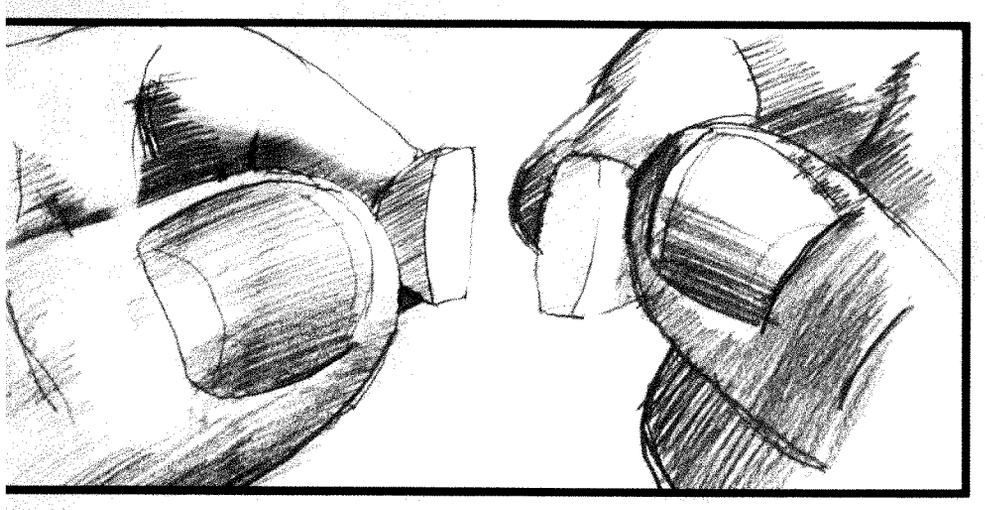
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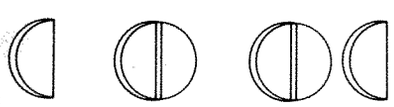
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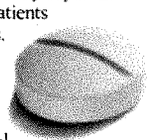
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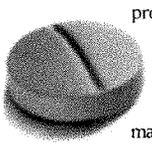
50 mg



25 mg



75 mg



150 mg

Other: Dizziness weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, jaundice, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache and malaise. These are not indicative of addiction

Dosage: Initiate at low levels, increase gradually, watching for sign of intolerance. As long as 30 days may elapse before adequate antidepressant effect develops; sedative effect may be noted earlier.

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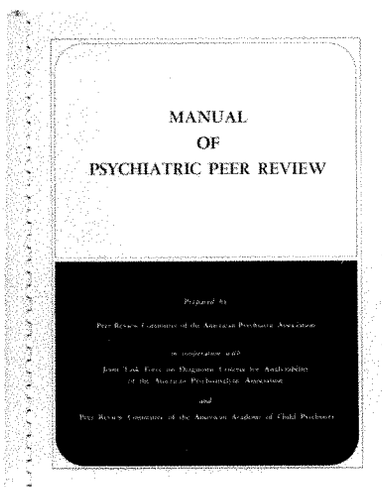
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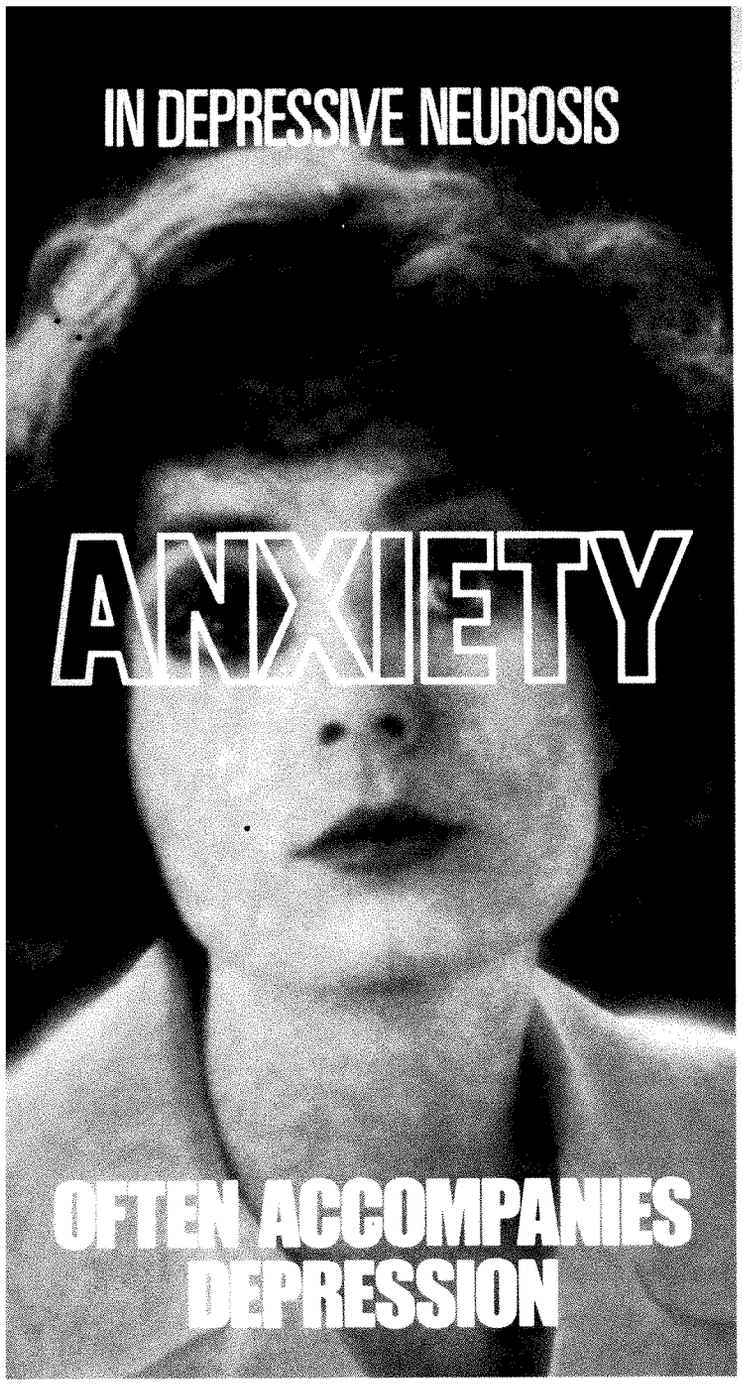
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Precautions: There have been infrequent reports of leukopenia and/or agranulocytosis and convulsive seizures. In epileptic patients, anticonvulsant medication should also be maintained. Pigmentary retinopathy, observed primarily in patients receiving larger than recommended doses, is characterized by diminution of visual acuity, brownish coloring of vision, and impairment of night vision; the possibility of its occurrence may be reduced by remaining within recommended dosage limits. Administer cautiously to patients participating in activities requiring complete mental alertness (e.g., driving), and increase dosage gradually. Orthostatic hypotension is more common in females than in males. Do not use epinephrine in treating drug-induced hypotension since phenothiazines may induce a reversed epinephrine effect on occasion. Daily doses in excess of 300 mg should be used only in severe neuropsychiatric conditions.

Adverse Reactions: *Central Nervous System*—Drowsiness, especially with large doses, early in treatment; infrequently, pseudoparkinsonism and other extrapyramidal symptoms; rarely, nocturnal confusion, hyperactivity, lethargy, psychotic reactions, restlessness, and headache. *Autonomic Nervous System*—Dryness of mouth, blurred vision, constipation, nausea, vomiting, diarrhea, nasal stuffiness, and pallor. *Endocrine System*—Galactorrhea, breast engorgement, amenorrhea, inhibition of ejaculation, and peripheral edema. *Skin*—Dermatitis and skin eruptions of the urticarial type, photosensitivity. *Cardiovascular System*—ECG changes (see *Cardiovascular Effects* below). *Other*—Rare cases described as parotid swelling.

It should be noted that efficacy, indications and untoward effects have varied with the different phenothiazines. It has been reported that old age lowers the tolerance for phenothiazines; the most common neurologic side effects are parkinsonism and akathisia, and the risk of agranulocytosis and leukopenia increases. The following reactions have occurred with phenothiazines and should be considered whenever one of these drugs is used. *Autonomic Reactions*—Miosis, obstipation, anorexia, paralytic ileus. *Cutaneous Reactions*—Erythema, exfoliative dermatitis, contact dermatitis. *Blood Dyscrasias*—Agranulocytosis, leukopenia, eosinophilia, thrombocytopenia, anemia, aplastic anemia, pancytopenia. *Allergic Reactions*—Fever, laryngeal edema, angioneurotic edema, asthma. *Hepatotoxicity*—Jaundice, biliary stasis. *Cardiovascular Effects*—Changes in terminal portion of electrocardiogram, including prolongation of Q-T interval, lowering and inversion of T-wave, and appearance of a wave tentatively identified as a bifid T or a U wave have been observed with phenothiazines, including Mellaril (thioridazine); these appear to be reversible and due to altered repolarization, not myocardial damage. While there is no evidence of a causal relationship between these changes and significant disturbance of cardiac rhythm, several sudden and unexpected deaths apparently due to cardiac arrest have occurred in patients showing characteristic electrocardiographic changes while taking the drug. While proposed, periodic electrocardiograms are not regarded as predictive. Hypotension, rarely resulting in cardiac arrest. *Extrapyramidal Symptoms*—Akathisia, agitation, motor restlessness, dystonic reactions, trismus, torticollis, opisthotonus, oculogyric crises, tremor, muscular rigidity, and akinesia. *Persistent Tardive Dyskinesia*—Persistent and sometimes irreversible tardive dyskinesia, characterized by rhythmical involuntary movements of the tongue, face, mouth, or jaw (e.g., protrusion of tongue, puffing of cheeks, puckering of mouth, chewing movements) and sometimes of extremities may occur on long-term therapy or after discontinuation of therapy, the risk being greater in elderly patients on high-dose therapy, especially females; if symptoms appear, discontinue all antipsychotic agents. Syndrome may be masked if treatment is reinstituted, dosage is increased, or antipsychotic agent is switched. Fine vermicular movements of tongue may be an early sign, and syndrome may not develop if medication is stopped at that time. *Endocrine Disturbances*—Menstrual irregularities, altered libido, gynecomastia, lactation, weight gain, edema, false positive pregnancy tests. *Urinary Disturbances*—Retention, incontinence. *Others*—Hyperpyrexia; behavioral effects suggestive of a paradoxical reaction, including excitement, bizarre dreams, aggravation of psychoses, and toxic confusional states; following long-term treatment, a peculiar skin-eye syndrome marked by progressive pigmentation of skin or conjunctiva and/or accompanied by discoloration of exposed sclera and cornea; stellate or irregular opacities of anterior lens and cornea; systemic lupus erythematosus-like syndrome.

Dosage: Dosage must be individualized according to the degree of mental and emotional disturbance, and the smallest effective dosage should be determined for each patient. In adults with depressive neurosis the usual starting dosage is 25 mg t.i.d. and the dosage ranges from 10 mg b.i.d. to q.i.d. in milder cases to 50 mg t.i.d. or q.i.d. for more severely disturbed patients; the total daily dose ranges from 20 mg to a maximum of 200 mg.

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THE AMERICAN JOURNAL OF PSYCHIATRY

The Brain as a Dream State Generator: An Activation-Synthesis Hypothesis of the Dream Process

BY J. ALLAN HOBSON, M.D., AND ROBERT W. MCCARLEY, M.D.

Recent research in the neurobiology of dreaming sleep provides new evidence for possible structural and functional substrates of formal aspects of the dream process. The data suggest that dreaming sleep is physiologically determined and shaped by a brain stem neuronal mechanism that can be modeled physiologically and mathematically. Formal features of the generator processes with strong implications for dream theory include periodicity and automaticity of forebrain activation, suggesting a preprogrammed neural basis for dream mentation in sleep; intense and sporadic activation of brain stem sensorimotor circuits including reticular, oculomotor, and vestibular neurons, possibly determining spatiotemporal aspects of dream imagery; and shifts in transmitter ratios, possibly accounting for dream amnesia. The authors suggest that the automatically activated forebrain synthesizes the dream by comparing information generated in specific brain stem circuits with information stored in memory.

SINCE THE TURN of the century, dream theory has been dominated by the psychoanalytic hypothesis that dreaming is a reactive process designed to protect consciousness and sleep from the disruptive effect of unconscious wishes that are released in sleep (1). Thus dreaming has been viewed as a psychodynamically determined state, and the distinctive formal features of dream content have been interpreted as manifestations of a defensive transformation of the unconscious wishes found unacceptable to consciousness by a hypothetical censor. A critical tenet of this wish fulfillment-disguise theory is that the transformation of the unconscious wish by the censor disguises or degrades the ideational information in forming the dream imagery. We were surprised to discover the origins of the major tenets of psychoanalytic dream theory in the neurophysiology of 1890 and have specified the transformations made by Freud in an earlier, related article (2). In detailing the neurophysiological origins of psychoanalytic dream theory, the concept of mind-body isomorphism, denoting similarity of form between psychological and physiological events, was seen as an explicit premise of Freud's thought.

Sharing Freud's conviction that mind-body isomorphism is a valid approach, we will now review modern neurophysiological evidence that we believe permits and necessitates important revisions in psychoanalytic dream theory. The activation-synthesis hypothesis that we will begin to develop in this paper asserts that many formal aspects of the dream experience may be the obligatory and relatively undistorted psychological concomitant of the regularly recurring and physiologically determined brain state called "dreaming sleep." It ascribes particular formal features of the dream experience to the particular organizational features of the brain during that state of sleep. More specifically, the theory details the mechanisms by which the brain becomes periodically activated during sleep and specifies the means by which both sensory input and motor output are simultaneously blocked, so as to account for the maintenance of sleep in the face of strong central activation of the brain. The occurrence and character of dreaming are seen as both determined and shaped by these physiological processes.

Based on the text of the Sandoz Lecture presented by Dr. Hobson at the University of Edinburgh, April 23, 1975.

Dr. Hobson is Associate Professor of Psychiatry and Dr. McCarley is Assistant Professor of Psychiatry, Harvard Medical School, Boston, Mass. Dr. Hobson is also Director and Dr. McCarley is Co-Director, Laboratory of Neurophysiology, Massachusetts Mental Health Center, 74 Fenwood Rd., Boston, Mass. 02115.

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The authors wish to express their appreciation to Drs. John Nemiah and John Nelson for their helpful comments on the manuscript.

The most important tenet of the activation-synthesis hypothesis is that during dreaming the activated brain generates its own information by a pontine brain stem neuronal mechanism, which will be described in detail. We hypothesize that this internally generated sensorimotor information, which is partially random and partially specific, is then compared with stored sensorimotor data in the synthesis of dream content. The functional significance of the brain activation and the synthesis of endogenous information in dreaming sleep is not known, but we suggest that state-dependent learning is at least as likely a result of dreaming as is tension reduction or sleep maintenance.

While we believe that the two processes emphasized in this paper—activation and synthesis—are major and important advances in dream theory, we wish to state explicitly and comment on some of the things that our theory does not attempt to do. The activation-synthesis hypothesis does not exclude possible defensive distortions of the value-free sensorimotor dream stimuli, but it does deny the primacy of any such process in attempting to explain *formal* aspects of dream content or the fundamental impetus to dreaming itself. The idea that dreams reveal wishes is also beyond the direct reach of our new theory, but some specific alternatives to this interpretation of several classic dream situations can be advanced.

The new theory cannot yet account for the emotional aspects of the dream experience, but we assume that they are produced by the activation of brain regions subserving affect in parallel with the activation of the better known sensorimotor pathways. Finally, the new theory does not deny meaning to dreams, but it does suggest 1) a more direct route to their acquisition than anamnesis via free association, since dream origins are in basic physiological processes and not in disguised wishes, 2) a less complex approach to their interpretation than conversion from manifest to latent content, since unusual aspects of dreams are not seen as disguises but as results of the way the brain and mind function during sleep, and 3) a broader view of their use in therapy than that provided by the transference frame of reference, since dreams are not to be interpreted as the product of disguised unconscious (transference) wishes. Dreams offer a royal road to the mind and brain in a behavioral state, with different operating rules and principles than during waking and with the possibility of clinically useful insights from the product of these differences. These points are discussed in the last section of this paper and elsewhere (3).

WHAT IS A DREAM?

A dream may be defined as a mental experience, occurring in sleep, which is characterized by hallucinoid imagery, predominantly visual and often vivid; by bizarre elements due to such spatiotemporal distortions as condensation, discontinuity, and acceleration; and by a delusional acceptance of these phenomena as "real" at the time that they occur. Strong emotion may or may not be associated with these distinctive formal properties of the dream, and subsequent recall of these mental events is almost invariably poor unless an immediate arousal from sleep occurs.

TABLE 1
Electrographic Criteria for Behavioral State Determination

State	Electromyogram	EEG	Electrooculogram
Waking	+	Low voltage, fast	+
Sleep			
Synchronized	+	High voltage, slow	—
Desynchronized	—	Low voltage, fast	+

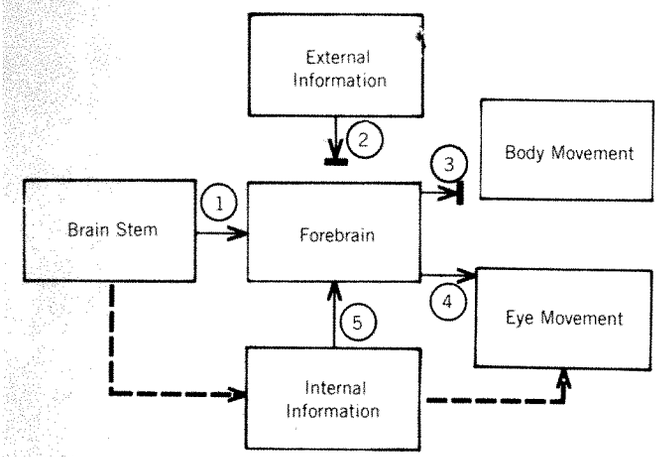
That this technical jargon describes a universal human experience seems certain, since the five key points in this definition are easily elicited from both naïve and sophisticated individuals when they are asked to characterize their dreams. We leave aside the question of whether other less vivid and nonperceptual forms of mental activity during sleep should also be called "dreams" and confine ourselves here to the psychophysiology of the hallucinoid type of dream. In doing so, we not only simplify the immediate task at hand but may also gain insight into the mechanisms underlying the most florid symptoms of psychopathology. We mean, of course, the hallucinations and delusions of the psychotic experience, which have so often invited comparison with the dream as we have defined it here.

WHAT IS THE STATE OF THE BRAIN DURING DREAMING SLEEP?

The physiological substrate of the dream experience is the CNS in one of its three principal operating states: waking (W), synchronized sleep (S), and desynchronized sleep (D). These states can be reliably and objectively differentiated by recording the EEG, the electromyogram (EMG), and the electrooculogram (see table 1). Hallucinoid dreaming in man occurs predominantly during the periodically recurrent phase of sleep characterized by EEG desynchronization, EMG suppression, and REMs (4). We call this kind of sleep "D" (meaning desynchronized, but also conveniently denoting dreaming).

In the systems analysis terms used in figure 1, this D brain state is characterized by the following "sensorimotor" properties: activation of the brain; relative exclusion of external input; generation of some internal input, which the activated forebrain then processes as information; and blocking of motor output, except for the oculomotor pathway. In this model the substrate of emotion is considered to be a part of the fore-

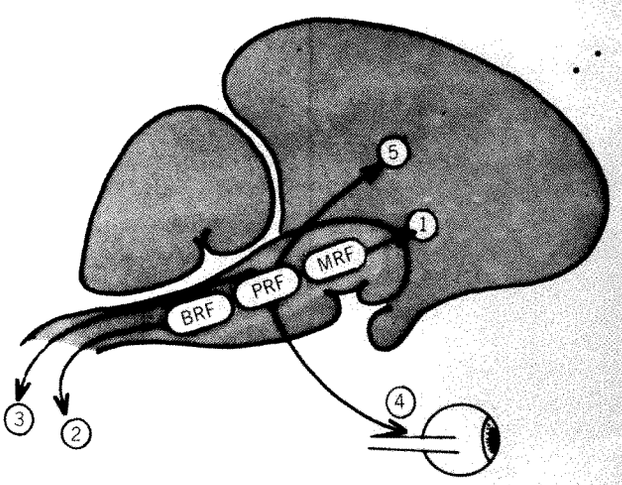
FIGURE 1
Systems Model of Dream State Generation



Processes Accounted for:

1. Activation of forebrain
2. Blockade of exteroceptive input
3. Blockade of motor output
4. Oculomotor activation
5. Provision of forebrain with internally generated information

FIGURE 2
Physiological Model of Dream State Generation Using the Sagittal Section of the Cat Brain and Showing the Bulbar (BRF), Pontine (PRF), and Midbrain (MRF) Divisions of the Reticular Formation



Processes Accounted for:

1. Activation of forebrain
2. Blockade of exteroceptive input
3. Blockade of motor output
4. Oculomotor activation
5. Provision of forebrain with internally generated information

brain; it will not be further distinguished here because we have no specific physiological evidence as to how this part of the system might work in any brain state. Memory is not shown but is considered to be a differentiated function of the brain that operates during the D state, such that output from long-term storage is facilitated but input to long-term storage is blocked. A highly specific hypothesis about dream amnesia has previously been derived (5) from the same evidence that we will now review in our attempt to account for the general sensorimotor aspects of the dream process.

ELECTROPHYSIOLOGY OF THE BRAIN DURING THE DREAM STATE

The three major electrographic features of the D state are of obvious relevance to our attempt to answer the following three questions about the organization of the brain in the dream state.

How is the forebrain activated in the D state? Since EEG desynchronization also characterizes waking, similar mechanisms of "activation" may be involved in both instances. Physiological evidence suggests that this is so: the reticular formation of the anterior brain stem is at least as active in D sleep as it is in the waking state (see figure 2).

How is motor output blocked in the D state? Physiological evidence clearly shows that the profound EMG suppression of D sleep is a consequence of the direct inhibition of spinal cord motoneurons (6). As a consequence, any organized motor patterns that might be generated during the intense brain activation of D sleep cannot be expressed.

That organized movement patterns are in fact generated, but not expressed, in normal D sleep is dramatically demonstrated by cats with lesions of the anterodorsal pontine brain stem (7). The animals show all of the major manifestations of D sleep except the atonia; instead of the fine twitches of the digits and the limb jerks that are normally present in D, these cats display complex motor behaviors including repetitive paw movements and well-coordinated attack and defense sequences that have no apparent relationship to the environment.

How is sensory imagery generated in the D state? In waking, a corollary discharge of the oculomotor system has been shown to suppress visual transmission during saccadic eye movements, possibly contributing to the stability of the visual field during that state (8). The same mechanisms might underlie the hallucinoid dream imagery by inhibiting and exciting neurons of the lateral geniculate body (9) and the visual cortex (10) during D sleep, when retinal input is reduced and unformed.

The possibility that oculomotor impulses trigger visual imagery is particularly intriguing in view of the demonstrated quantitative correlation between eye

movement intensity and dream intensity (11). More specific correlations have also been reported to relate eye movement direction to orientation of the hallucinated gaze in dreams (12). This finding has been interpreted as indicative of "scanning" the visual field—implying cortical control of the eye movements in dreaming sleep. An alternative, although not exclusive, hypothesis is that the oculomotor activity is generated at the brain stem level and that the cortex is then provided with feed-forward information about the eye movements. According to this view, we are not so much scanning dream imagery with our D sleep eye movements as we are synthesizing the visual imagery appropriate to them. We will return to the implications of this intriguing possibility in discussing the generation of eye movements in dreaming sleep, but we wish to stress here the general significance of this clue to the identity of an "internal information generator" operating at the brain stem level in the dreaming sleep state.

The eye-movement-related inhibition of sensory relays (13), as well as the possible occlusion of exogenous inputs by internally generated excitation, may also contribute to the maintenance of sleep in the face of strong central activation of the brain. In this sense the dream process is seen as having a sleep maintenance mechanism built into its physiological substrate rather than a sleep guardian function operating at the psychological level.

A firm general conclusion can be reached at this point: the desynchronized phase of sleep is the physiological substrate of hallucinoid dreaming, as defined. This conclusion is of profound significance to psychophysiology, since we can now reliably and objectively characterize and measure many aspects of the brain when it is in the dream state. For example, one feature that emerges from the psychophysiological study of dreaming and one that was not at all evident from introspective, psychoanalytically oriented research, is that the brain enters the dream state at regular intervals during sleep and stays in that state for appreciable and predictable lengths of time. One clear implication of this finding is that dreaming is an automatically preprogrammed brain event and not a response to exogenous (day residue) or endogenous (visceral) stimuli. A second implication is that the dream state generator mechanism is periodic, that is, the dream state generator is a neurobiological clock (14). Since the length of the sleep cycle and, by inference, the frequency of dreaming, is a function of body size within and across mammalian species (15), the system controlling the length of the period must have a structural substrate. Thus we must account for size-related periodicity with our model of the dream state generator.

AN ANIMAL MODEL OF THE BRAIN DURING THE DREAM STATE

We said that the length of the sleep cycle varies "across species." Does that mean that nonhuman animals dream? Unfortunately we cannot know, but we are willing to assert that if they do so, it is when their brains are in the D sleep state. Because we have no direct evidence of any significant difference between the brain state of man and the brain state of other mammals in D sleep, we therefore feel justified in asserting that the brain state of our experimental animal, the cat, constitutes a reasonable subject for our study of the brain as a dream process generator, whether or not cats dream. This assertion seems justified since we are restricting our attention here to formal aspects of the dream experience; our experimental model need not dream or even possess "consciousness" to be useful as a source of physiological information. If we accept this argument and use the definition of dreaming offered above, then the presence of D sleep in cats (16) offers nothing less than an animal model in which to study the neurophysiological basis of a hallucinoid mental process in man. Such a model is as important in experimental psychiatry as it is rare. Let us now turn to the biological data upon which our sketches of the brain as a dream state generator are based.

LOCALIZATION OF THE POWER SUPPLY OR TRIGGER ZONE OF THE DREAM STATE GENERATOR

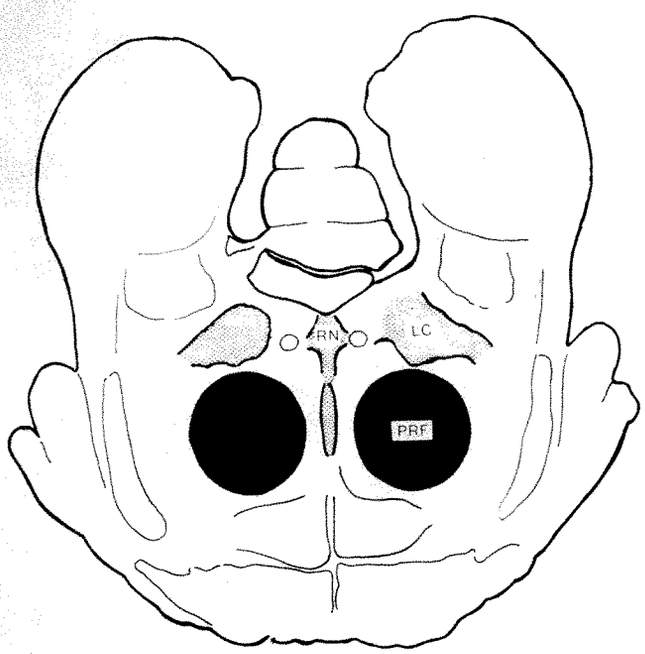
Lesion, stimulation, and recording studies pioneered by Jouvet (17) have strongly implicated the pontine brain stem as critical to the generation of the desynchronized sleep phase (see figure 3 for a summary of the neuroanatomy of this region). Important findings supporting this hypothesis include the following.

Large lesions of the pontine reticular formation prevent the occurrence of desynchronized sleep for several weeks in cats (17). This suggests that the pontine reticular formation may be the site of an executive or triggering mechanism for desynchronized sleep. Prepontine transections and forebrain ablation have no effect upon periodicity or duration of the skeletal, muscular, and oculomotor manifestations of D sleep (17). The data indicate that the trigger, the power supply, and the clock are pontine.

The pontine brain stem is thus implicated as the site of both the trigger and the clock. The periodicity of the D sleep clock in poikilothermic pontine cats lengthens as temperature declines, indicating orthodox metabolic mediation of the cycle, in contrast to the temperature independence of circadian rhythms. If we assume that the physiological substrate of consciousness is in the forebrain, these facts completely eliminate any possible contribution of ideas (or their neural substrate) to the primary driving force of the dream process.

Small lesions of the dorsal pontine brain stem, in the region of the locus coeruleus (LC), may eliminate the atonia but no other aspects of desynchronized sleep (7). This suggests that inhibition of muscle tone

FIGURE 3
The Anatomy of the Pontine Brain Stem*



*On this frontal section of the cat brain stem, the cells that are selectively activated are in the paramedian reticular formation (PRF) (giganto cellular tegmental field), while the cells that are selectively inactivated lie more dorsally (in the region of the locus coeruleus [LC]) and medially (in the region of the raphe nuclei [RN]). Compare this with figure 5, which summarizes the neurophysiology and shows the anatomy in a sagittal section.

is somehow dependent upon the integrity of the LC. The elaborate motor behavior that characterizes the D sleep of cats with LC lesions has been described as "pseudo-hallucinatory" (7). Whether or not one accepts the sensory implications of that designation, the importance of motor inhibition in quelling the effects of central excitation during the dream state is clear.

This finding has an important bearing on mechanisms of dream paralysis and suggests that in the classic chase dream, the dreamer who has trouble fleeing from a pursuer is as much accurately reading the activated state of his motor pattern generator and the paralyzed state of his spinal neurons as he is "wishing" to be caught. This dream experience is so universal and the feeling of constrained motor action so impressive as to make its physiological basis in the descending inhibition of motoneurons seem to us inescapable. Conversely, this reasonable and adequate explanation of the paradox of the chase dream makes its interpretation as wish fulfillment less compelling. Other implications of the D sleep activation of various motor system pattern generators for movements and dream plots have been discussed elsewhere (3).

The vestibular system, as classically established, integrates head position and movement with eye position and posture. Pompeiano and Morrison (18) showed that lesions of the vestibular nuclei interfered with the bursts of REM but not with the isolated eye movements of D. This finding suggested that the vestibular system contributed to the elaboration and rhythmicity of the eye movements but that the eye movement generator was extravestibular. Magherini and associates (19) found that systemic injections of the anticholinesterase agent physostigmine produced rhythmic eye movements in decerebrate cats, suggesting that the eye movement generator may be cholinergic. Thus the central, automatic activation during sleep of the vestibular system may provide a substrate for endogenously generated, specific information about body position and movement. Flying dreams may thus be a logical, direct, and unsymbolic way of synthesizing information generated endogenously by the vestibular system in D sleep. In view of this reasonable and direct explanation, it seems gratuitous to "interpret" the sensual flying dream as sexual.

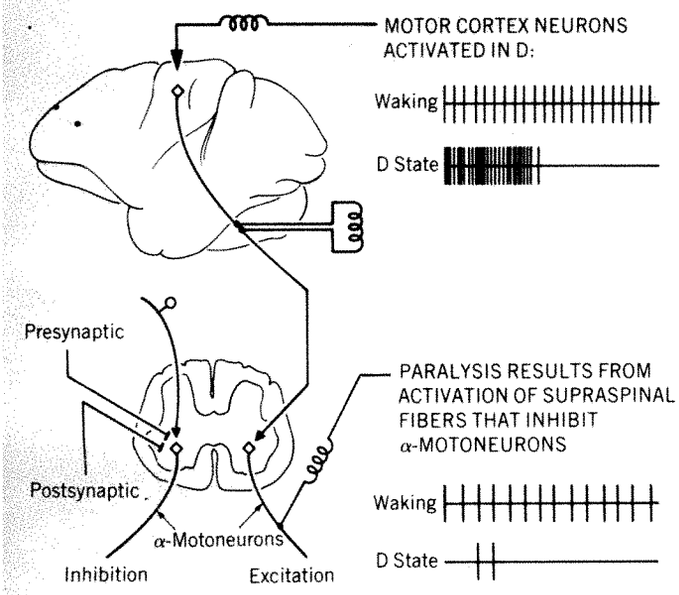
In accord with the isomorphism principle, the degree of neuronal activation in brain systems should parallel the frequency and intensity of dreams to these systems (3), and the predominance of visual sensorimotor activity in both brain and mind supports this notion. Symbol formation and the often bizarre juxtaposition of sensations in the dream may be a reflection of the heightened degree of simultaneous activation of multiple sensory channels in dreaming as compared with waking (3).

Long-term electrical stimulation of the pontine brain stem results in the earlier appearance of sleep episodes and in increases in the absolute amounts of desynchronized sleep, but it does not affect the periodicity of its occurrence (20). By implication, the delivery of electrical energy accomplishes what most psychological and behavioral treatments fail to achieve: an increase in the duration of dreaming sleep. Testing the assumption that the generator neurons are cholinoceptive, our laboratory team has recently established that injection of the cholinergic agent carbachol into the pontine reticular formation produces prolonged enhancement of D-like sleep behavior (21). In man the parenteral injection of the anticholinesterase agent physostigmine potentiates D sleep, and the pharmacologically induced episodes are associated with hallucinoid dreaming (22). The time of occurrence and duration of dreams may thus be chemically determined.

In summary, these results support the hypothesis that the pontine brain stem is the generator zone for the D sleep state. The trigger mechanism for the whole system, including the eye movement generator, may be cholinoceptive and the executive zones are probably in the reticular formation. The LC is involved, possibly in a permissive or reciprocal way, and is especially important in mediating spinal reflex inhibition. Together, these two regions may constitute the clock. We will have more to say about the hypothesis of reciprocal interaction between them later in this paper.

Although the brain stem mechanisms mediating atonia remain obscure, it is clear from the work of Pompeiano (6) that both monosynaptic and polysynaptic spinal reflexes are tonically inhibited during D sleep (see figure 4). In addition, during the bursts of REM,

FIGURE 4
Mechanisms of Sleep Paralysis*



*The upper part of the figure illustrates the intense activation in D sleep of antidromically identified pyramidal tract neurons of the motor cortex. Note the relatively regular discharge in waking (W) and the clustering of discharges in D sleep in these models of 3-second epochs of microelectrode recordings (vertical lines indicate discharges). The lower portion of the figure shows the inhibitory events of D at the spinal cord level that largely prevent alpha motoneuron discharge and consequent muscle excitation, despite the activation of excitatory (arrow) pyramidal tract fibers. Both presynaptic and postsynaptic inhibition (bars) are present in D (sketched on the left side of the cord section). Absence of this inhibition in W allows alpha motoneuron discharge in response to excitation from pyramidal tract fibers (17).

there is a descending presynaptic inhibition of the most rapidly conducting (group 1*a*) spinal afferent endings. Both presynaptic and postsynaptic inhibition appear to be of brain stem origin. Phasic presynaptic inhibition has also been shown to occur in sensory relays elsewhere in the brain during D sleep (6). Thus motor output is tonically damped throughout D and sensory input is phasically damped in concert with the REM bursts. In other words, we are not only paralyzed during our dreams, but the degree to which we are paralyzed fluctuates in concert with the intensity of the internally generated information and the degree to which we suppress exogenous input.

On the basis of this evidence, the systems terminology used earlier (see figure 1) can be tentatively translated into the anatomical and physiological terms of figure 2; and the activation-synthesis hypothesis of dreaming can be stated as follows: during D sleep, a cholinergic mechanism in the reticular formation of the pontine brain stem is periodically activated. The consequences of this activation are as follows:

1. The forebrain is tonically activated, probably via the midbrain reticular formation that is also responsible for its activation during waking. Thus the forebrain is made ready to process information.

2. The spinal reflexes are tonically inhibited, possibly via the bulbar reticular formation and LC; thus motor outflow is blocked despite high levels of activity in the brain, including the motor cortex.

3. The oculomotor and vestibular systems are phasically activated by the pontine reticular formation so as to produce eye movements. This circuitry, in its entirety, is an internal information source or generator that provides the forebrain with spatially specific but temporally disorganized information about eye velocity, relative position, and direction of movement. Information may similarly be derived from the brain stem generators of patterned motor activity.

4. At the same time that internal information feedback is being generated by the activation of various motor systems, exteroceptive input to sensory systems is phasically blocked. This may intensify the relative impact of the endogenous inputs to the brain, accounting for the intensity of dream imagery and preventing sleep disruption by the externally generated excitation.

This working sketch of the dream state generator, based on the classical localizing methods of experimental neurology, is intriguing but unsatisfying in that it fails to specify the mechanisms by which the pontine generator is turned on, kept active for a time, and then shut off. Further, it does not say anything about the mechanism of periodicity. To provide details about the anatomy and physiology of the periodic trigger mechanisms of the generator process, we will now turn our attention to the neuronal level of analysis. In doing so, we also come full circle in our reaffirmation of isomorphism since it was the neuron that Freud recognized as the physical unit of the nervous system on which he based his dream theory (2).

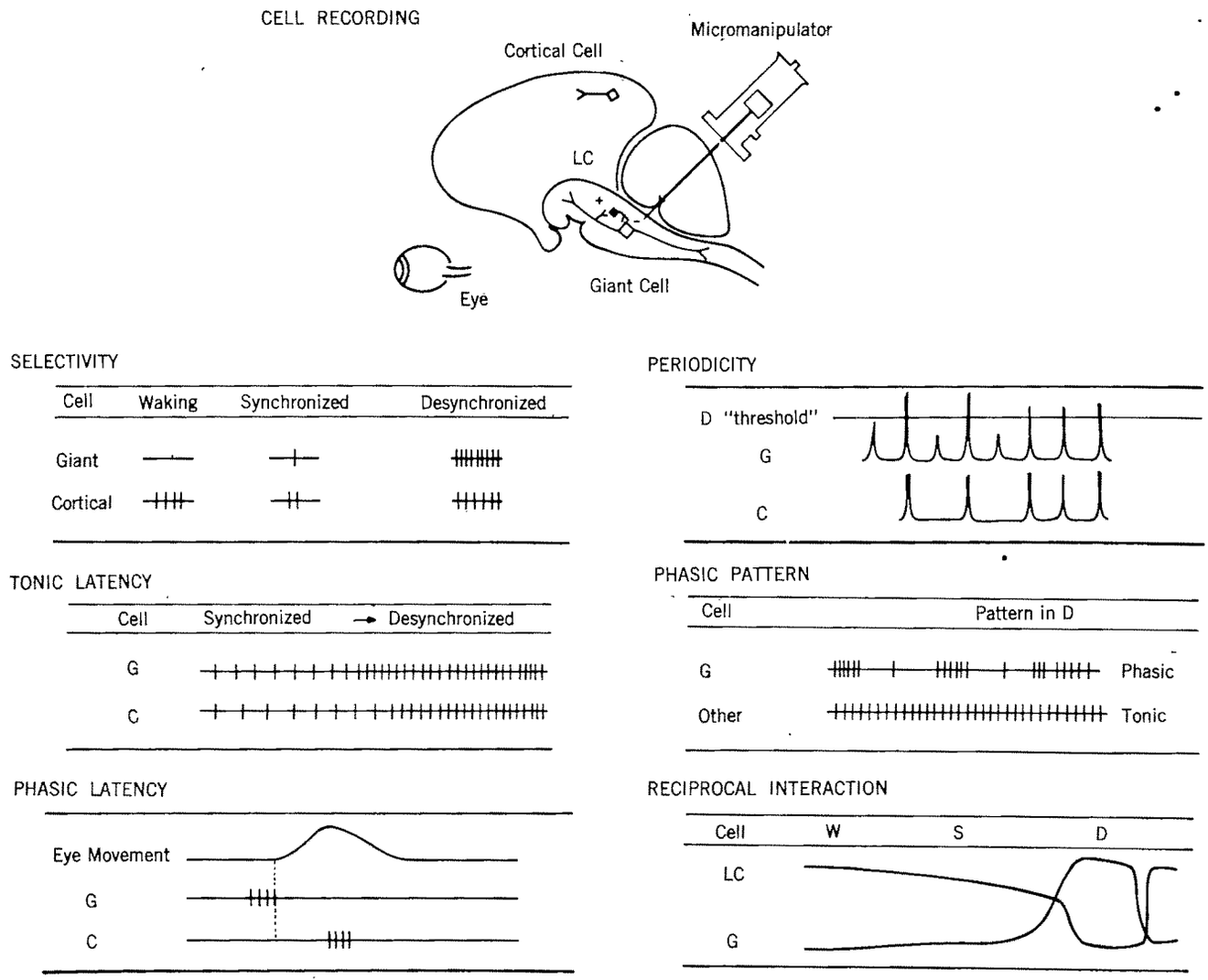
HISTOLOGICAL FEATURES OF RELEVANCE TO THE PERIODIC TRIGGERING OF THE DREAMING SLEEP STATE GENERATOR

Several structural details of the pontine brain stem are notable as possible elements of a D sleep control device with rhythmic properties (see figure 3 for an illustration of the anatomy discussed).

In his discussion of the histology of the pontine brain stem, Cajal (23) emphasized three points:

1. The paramedian reticular giant cells, with their rostral and caudal axonal projections, are admirably suited to serve as output elements of the generator; when excited they could influence many other cells. The work of Brodal (24) and the Scheibels (25) shows that the spinal cord and thalamus receive projections from these elements. Although they are relatively few in number, conservative estimates of their postsynaptic domain indicate that each directly projects to nine million (9×10^6) postsynaptic neurons. Thus the 3,000 pontine reticular giant cells in the cat might make many billions of synapses (2.9×10^{10}). Since the giant cells also project to other brain stem nuclei and have recurrent axons to themselves, mutual interaction with raphe-type elements (see below) and self-reexcitation

FIGURE 5
Cellular Neurophysiology of Dream State Generation*



*The cell recordings are made from hydraulically driven microelectrodes that can be stereotaxically directed at neurons in the cat brain during natural sleep. Two classes of brain stem neurons are represented by the reticular giant cell (G in the physiological models) and the LC cell; the synaptic interactions suggested are detailed in figure 8. A cortical cell is also shown.
The results of the cell recording experiments are shown in six models representing the criteria used to quantify discharge properties: selectivity—giant cells concentrate their discharge in D to a greater extent than cerebral cortical or other brain stem neurons; tonic latency—giant cells show rate increases that precede those of cortical neurons during the S to D transition; phasic latency—giant cells fire before the REMs of D, while cortical neurons fire after them; periodicity—peaks in the giant cell activation curves are periodic and the higher peaks are associated with D sleep episodes and peaks of cortical activity; phasic pattern—giant cells show a higher degree of clustered firing in D than do other neurons; and reciprocal interaction—the rate curves of giant cells and LC cells are reciprocal over the sleep cycle.

are both possible. These two features could be used to create excitability variability, with powerful consequences for the whole nervous system.

2. The raphe neurons of the midline are ideally situated and connected to regulate excitability of paramedian elements, and they also have extensive projections to other brain regions. The discovery that these cells concentrate the biogenic amine serotonin (26) gives this regulatory hypothesis an attractive corollary: these cells might regulate excitability of their postsynaptic neurons via specific transmitter substances. Another brain stem cell group, in the locus coeruleus, has been shown to concentrate the amine norepinephrine (26). There are thus at least two neuronal candidates for a level setting role, and both are probably inhibitory. Since the giant cells are excitatory (and probably cholinergic; see below), a substrate for reciprocal interaction is established.

3. Cajal (23) suggested that input to the central reticular core might be via small stellate cells in the lateral zone. This input channel, which we now know to be more diffuse than was originally suspected, could be used to abort or damp the core oscillator at critical ambient stimulus levels. This is an important feature,

since adaptation depends on the capacity to interrupt the cycle and not to incorporate all exogenous stimuli into the dream plot.

CELLULAR ACTIVITY IN THE PONTINE BRAIN STEM DURING THE SLEEP CYCLE

A direct experimental approach to the question of D state control has been made with cats by recording from individual neurons in many parts of the brain as the sleep cycle normally evolved. In this experimental paradigm, the frequency and pattern of extracellular action potentials, which are the signal units of nerve cells, are taken as indices of a cell's excitability; the influence of a recorded neuron upon other cells and that neuron's own control mechanism may also be inferred from the data. This method has the advantage of being relatively physiological since it does little to alter or damage the properties of the system under study. When cats are kept active at night, they will sleep under the necessary conditions of restraint during the daytime. The microelectrodes can then be stereotaxically directed at the brain stem and individual cell activity recorded for as long as 20 hours, allowing many successive sleep cycles to be studied (see figure 5).

The pontine brain stem control hypothesis has been tested in three ways at the level of single cells.

Selectivity criterion: which cells change rate most in D? We assumed that cells which showed pronounced alterations in discharge rate over the sleep cycle were more likely to be playing a controlling role than those showing minimal change. We further assumed that those cells having peaks of activity in phase with the D phase of the cycle were more likely to be specifically and actively involved in dreaming sleep state control than those with multiple peaks. We found that the giant cells of the pontine tegmentum concentrated their discharge in the D phase of sleep to a greater extent than any other group of neurons (27). They became our prime candidate for a generator function.

Tonic latency criterion: which cells change rate first in D onset? If the cells with positive discharge selectivity were driving the dreaming sleep phase of the sleep cycle, then their rates would be expected to increase in advance of the behavioral state change. Such phase leads might well be longer than those of the follower neurons under the control of the giant cells. The giant cells, when recorded over entire sleep cycles and through repeated sleep cycles, were found to change rate continuously (28). Significant rate increases occurred *as long as 5 minutes* before a desynchronized sleep phase. When the 2 minutes just prior to desynchronized sleep onset were studied, a rate increase in a pool of giant cells was observed 10 seconds before a similar increase in a pool of cerebral cortical neurons.

The rapidly accelerating limb of the giant cell activity curves at D sleep phase onset indicated that this was a time of maximal excitability change in this pool of neurons. The goodness of fit of the data by an exponential curve indicated that reexcitation within the pool might be superimposed upon disinhibition from without. The positive tonic latency indicated that the activation of the forebrain might be a consequence of activation of the brain stem but that the converse could not be the case.

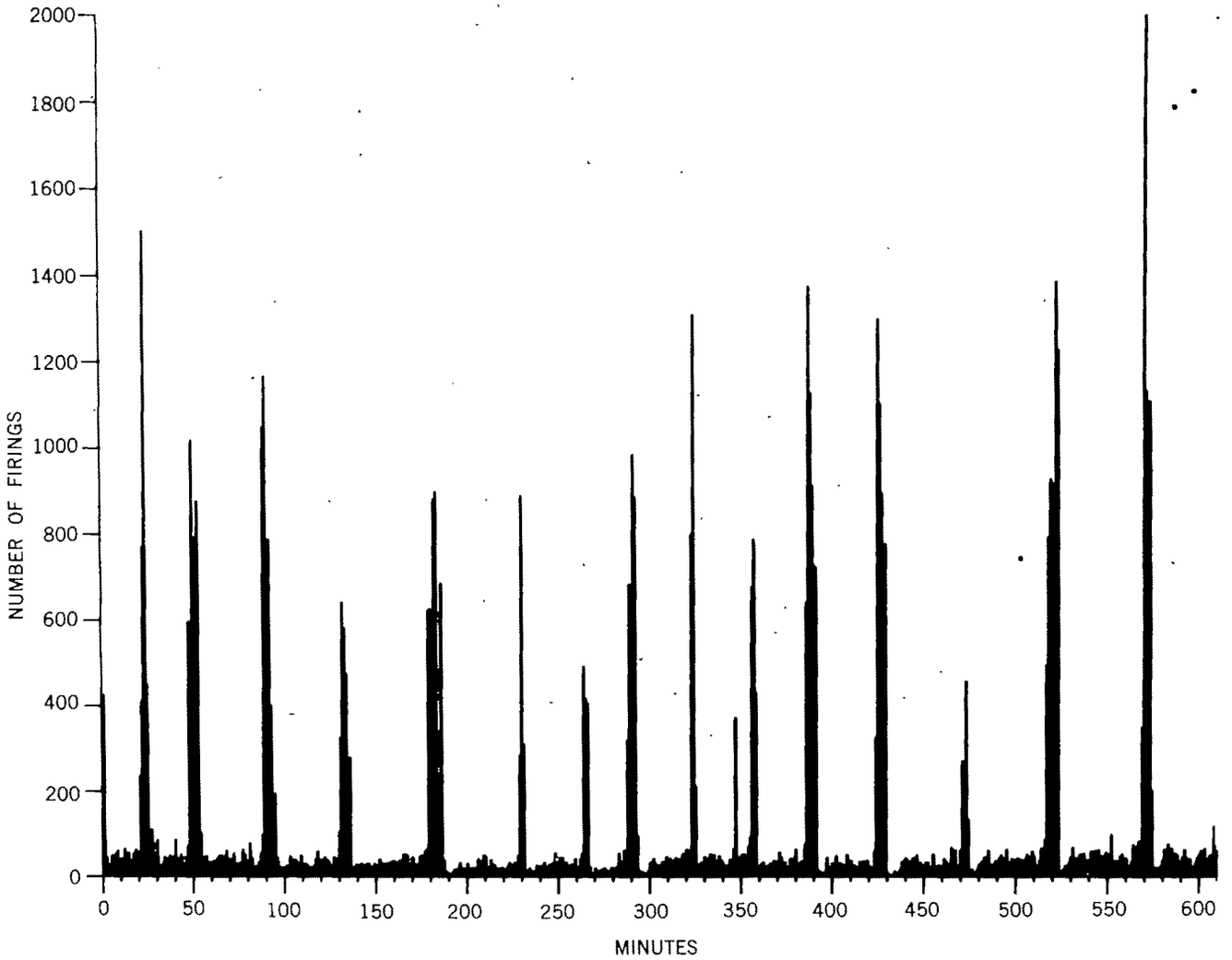
Phasic latency criterion: which cells fire before eye movements of D? Because of the proximity and direct projections to oculomotor neurons from giant cells, we tested the possibility that they might be generating the REMs so characteristic of the desynchronized phase of sleep by determining the time of occurrence of short-term rate increases by the giant cells in relation to eye movement onset. On the average, such rate increases were more prominent and anticipated eye movement by longer intervals than other brain stem neurons (29). Rate increases by presumed follower elements (in the posterolateral cerebral cortex) *followed* the eye movements by many milliseconds. It could therefore be concluded that the eye movements might be initiated by giant cells but could not be generated by cortical neurons. This finding practically wrecks the scanning hypothesis and strongly favors the idea that visual cortical events are determined by events in the oculomotor brain stem.

At this point we felt justified in concluding that the giant cells of the pontine tegmentum were critical output elements in a sleep cycle control mechanism. More particularly, we proposed that they might be generator elements for some of the tonic and phasic excitatory events in the desynchronized sleep phase of the cycles: most important to the activation-synthesis hypothesis of dreaming are the determination of EEG desynchronization (activation of the forebrain) and REMs (provision of forebrain with internally generated information). At the very least, we felt that we had found an important avenue to understanding sleep cycle control, since we could now examine the properties and possible mechanisms of giant cell excitability regulation. In this regard there are three additional points worthy of emphasis.

Periodicity criterion. Long-term recordings of giant cells revealed peaks of activity in phase with each full-blown desynchronized sleep episode (30) (see figure 6). Less prominent peaks were associated with abortive episodes and were rarely seen with no electrographic evidence of desynchronized sleep. Spectral analysis of these long-term data confirmed the impression of powerful periodicity in the discharge peaks, indicting that 1) sleep cycles are periodic, 2) underlying cell activity is probably even more so, and by definition, 3) cell excitability is under the control of a neurobiological clock. The possible mechanisms of excitability control are thus of great interest.

Phasic pattern criterion. The pattern of giant cell discharge within each D sleep episode indicated that classical pacemaker mechanisms are *not* involved in giant cell excitability regulation (31). Regular inter-

FIGURE 6
Discharge Activity of a Giant Cell Neuron Recorded over Multiple Sleep-Waking Cycles*



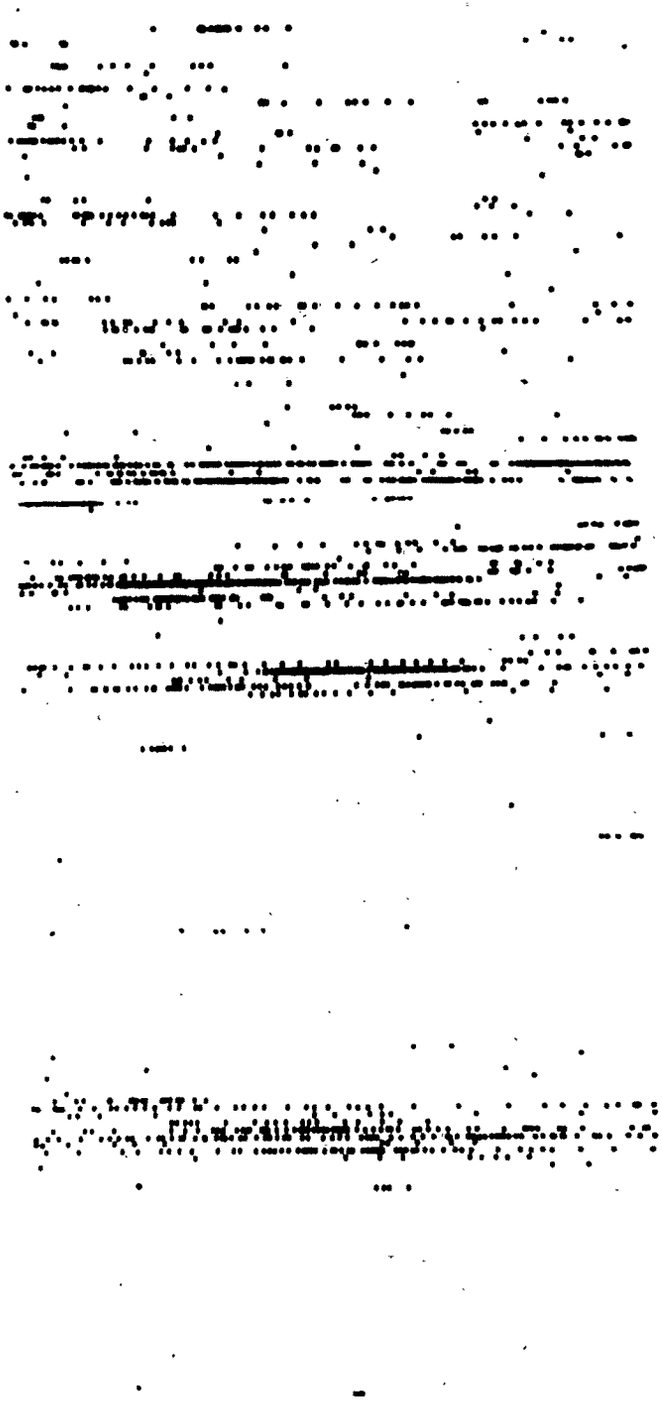
*Each peak corresponds to a desynchronized sleep episode, and a regular trend of discharge activity over a cycle is observable: a peak in desynchronized sleep; a rapid decline at the end of the desynchronized sleep episode; a trough, often associated with waking; a slow rise (in synchronized sleep and preceding all electrographic signs of desynchronized sleep); and an explosive acceleration at the onset of desynchronized sleep. Note also the extreme modulation of activity and the periodicity (30).

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spike intervals were exceptional, indicating that the rate increases were not caused by endogenous membrane depolarizations. The tendency, rather, was for giant cells to discharge in intermittent, prolonged clusters of irregularly distributed spikes as if the cells were responding to excitatory postsynaptic potentials from other neurons (see figure 7). In our view, a likely source of much of this input, especially as the longer clusters developed, was other giant cells. Once other neurons were excited, feedback from them is to be expected. It also seemed likely that the clusters of giant cell discharge were causally related to the eye movement bursts of the D sleep phase.

Reciprocal interaction criterion. If giant cell excitability change is not an intrinsic property of the giant cells, what other cell group might regulate it and in what way might that regulation be effected? Since all indices showed giant cells to discharge first in relation to both the tonic and phasic events of desynchronized sleep, we considered the possible contribution of inhibitory neurons. Since interneurons do not appear to exist in the giant cell fields, such cells should be discrete from but proximal to the giant cell. To be effective, projections should be abundant and should have inhibitory transmitter action upon the giant cells. Reciprocal rate changes during the sleep cycle are to be expected if such cells exist. We have discovered just such changes in a small number of unidentified cells in the region of the posterior locus coeruleus and the nucleus subcoeruleus (32). Not only is discharge concentration of these elements quantitatively inverse to those of the giant cells in the phases of the cycle, but their decelerating rate curve is the approximate mirror image of that of the giant cells

FIGURE 7
Temporal Clustering of Extracellularly Recorded Discharges of Cat Giant Cell Neurons During D Sleep*



*Each discharge is represented by a dot; the time sequence runs left to right and top to bottom, with each line 1 second in duration. The figure encompasses about 200 seconds of D sleep activity. Clustering is visible as closely spaced dots and, over longer durations, as "bands" of activity, some of which appear to occur rhythmically. Note the various durations of clusters and the presence of shorter duration clusters of activity within longer duration clusters. Clusters are delimited by periods of relative inactivity. Such sequences of giant cell neuronal activity are temporally associated with runs of eye movements and ponto-geniculo-occipital waves, and similar sequences of executive neuron discharges may represent the neuronal substrate of dream sequences in man (Hobson and McCarley, unpublished data).

at desynchronized sleep onset as seen in part C. We called such cells "D-off" cells to contrast their activity curves with those of the giant cells, prototypes of the "D-on" species of neurons. We do not know if the "D-off" cells are catecholaminergic but their location and discharge properties make this possible.

McGinty and associates (33) have found similar reciprocal rate changes in the dorsal raphe nucleus (DRN) neurons and we have recently confirmed this finding. The low regular rates of discharge by these cells in waking suggest a level-setting or pacemaker function. Their location and discharge properties are the same as those cells thought to be serotonergic on the basis of pharmacological experiments (34). Since both the LC and DRN are adjacent to and project to giant cells, and since giant cells receive abundant serotonergic and catecholaminergic endings, we thought that the mutual interconnections of these D-on and D-off cells could form a substrate for reciprocal interaction which regulated sleep cycle oscillation (30).

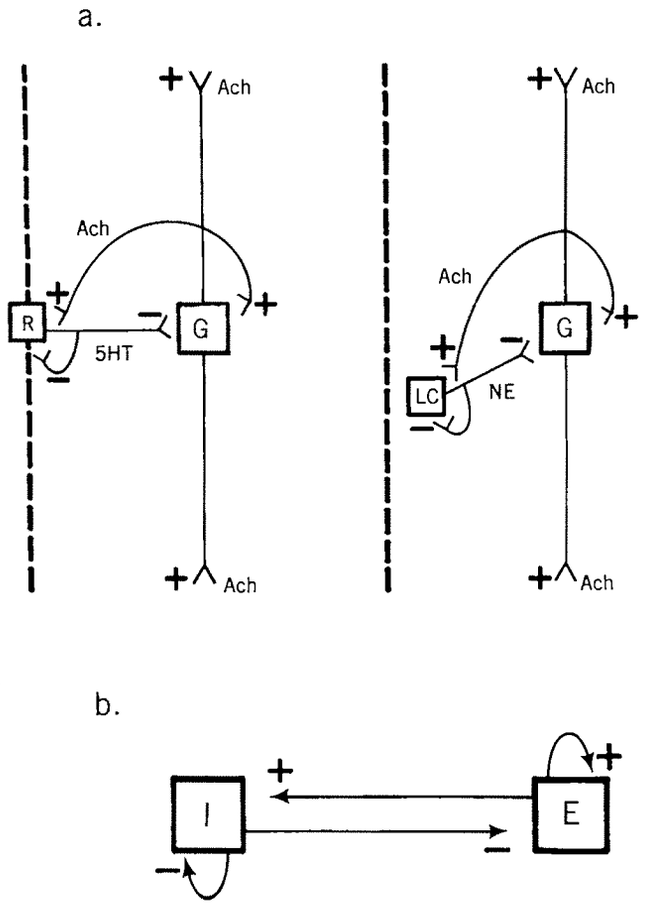
A MODEL FOR A BRAIN STEM SLEEP CYCLE OSCILLATOR

Restricting attention to within-sleep changes, we constructed a physiological model that bears a striking resemblance to the a priori schema derived from Cajal (see figure 8, top portion). Most of the connections have been demonstrated but many of the synaptic assumptions are as yet unproven physiologically. In addition to being explanatory, the model suggests experiments, particularly those employing pharmacological methods, the results of which will lead to its future modification. Since the LC, DRN, and giant cell groups are chemically differentiated, we deduced that their action and interaction may involve specific neurotransmitters.

In preliminary tests of the model, we have found that microinjection of the cholinomimetic substance carbachol into the giant cell zone not only gives more potent desynchronized sleep phase enhancement than injections into the adjacent tegmental fields but simultaneously activates giant cells. The results also indicate that an opposite effect is obtained at locus coeruleus sites (as if an inhibitory cell group were being activated). We have not yet tested this last hypothesis directly, but the LC cells do resume firing before the end of D sleep. We assume that as FTG excitation declines and LC inhibition grows, the cycle ends. In the decerebrate cat, physostigmine-induced D episodes are associated with activation of neurons in the giant cell and suppression of firing by cells in the LC and DRN (35).

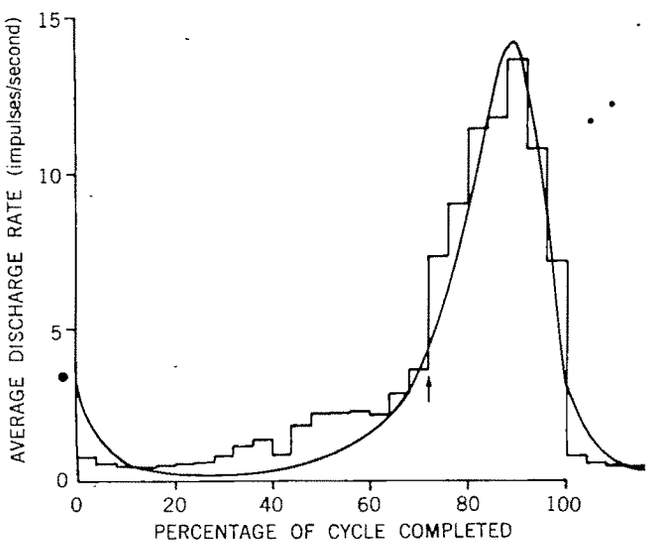
The physiological model can be reduced to a simple unit susceptible to mathematical analysis (see figure 8, bottom portion). Cell group E (giant cell) and cell group I (raphe and/or LC) are assumed to be mutually interconnected; cell group E is excitatory to itself and to group I, which inhibits itself and group E. Growth of

FIGURE 8
Reciprocal Interaction Model of Generator Process*



*Physiological models used to organize and interpret results of pharmacological experiments on desynchronized sleep. The G cells are seen as executive elements; they excite with and are excited by acetylcholine (Ach). They interact reciprocally with two aminergic cell groups, the LC and raphe (R), which utilize norepinephrine (NE) and serotonin (5HT) respectively. Both amines are hypothesized to be inhibitory to the G cells. D sleep will therefore be enhanced by increasing G cell excitability, and this can occur by either adding cholinergic drive or subtracting aminergic inhibition. Conversely, D sleep will be suppressed by subtracting cholinergic drive or by adding aminergic inhibition.
Formal reduction of the elements in the top portion of the figure yields the general model of reciprocal interaction, of inhibitory (I,-) and excitatory (E, +) populations, each of which contains a self-loop as well as a projection to the other set. The resulting oscillation of activity in the two sets can be mathematically described by the Lotka-Volterra equations.

FIGURE 9
Time Course of Giant Cell Activity over the Sleep Cycle*

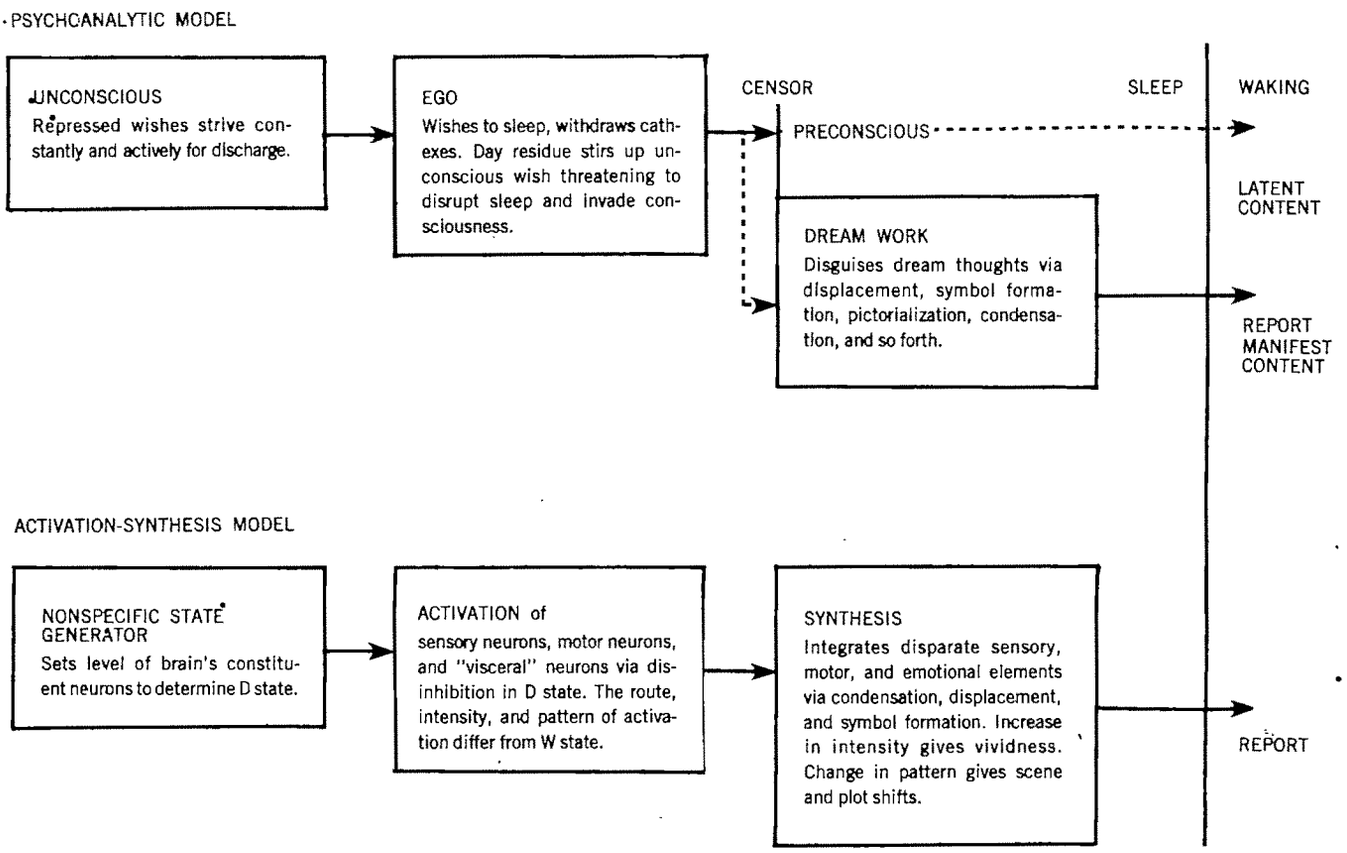


*The histogram shows the average discharge level (impulses/second) of a giant cell neuron over 12 sleep-waking cycles, each normalized to constant duration. The cycle begins and ends with the end of a desynchronized sleep period. The arrow indicates the average time of D sleep onset. The smooth curve is derived from a mathematical model of sleep cycle control and shows a good fit to the experimental data. The probability of obtaining dream-like mentation reports might be expected to show the same trajectory as these curves (30). Reprinted by permission from *Science*, volume 189, pages 58-60, July 4, 1975. Copyright 1975 by the American Association for the Advancement of Science.

activity in one group occurs at the expense of growth in the other, and vice versa. As such the cell groups are analogous to two populations, prey and predator, whose interaction can be described by a set of nonlinear differential equations, the Lotka-Volterra equations (30). As shown in figure 9, the time course of activity of cell group E closely resembles that predicted by these equations. It is now possible to plot the activity curves of cell group I and compare the actual data with the curves predicted by the model. The phase lag between the reciprocal cycles remains to be explained and the previously noted fact that cycle length is proportional to brain size suggests that a distance factor may be at work. The distance between the two cell fields could be such a factor through its determination of protein transport time. Assuming an average LC-FTG internuclear distance of 2.5 mm and a fast protein transport time of 96 mm/day, a period length of about 35 minutes is predicted for the cat. This figure is within limits normal for that species. Another possible substrate for the long, size-dependent time constant of the cycle is the recently discovered class of long-duration postsynaptic transmitter actions (36) that may be mediated by second messengers such as cyclic AMP (37). Since the cyclic nucleotides activate protein kinases, the metabolic activity of the neuron, including the synthesis of neurotransmitters, can be linked to and entrained by membrane events.

An important point is that the mathematical model parallels, but is not identical to, the physiological model. This means that even if the specific assumptions about physiological interaction are incorrect, the mathematical model may be viable and useful in another system—for example, the coupling of the circadian and ultradian oscillators (14) or, at another level of analysis, in a molecular system. This is particularly important to keep in mind since it is also at the molecular level that time constant elements necessary to explain the long periodicity of the sleep-dream cycle may be found.

FIGURE 10
Two Models of the Dream Process*



*In the psychoanalytic model the motive force of the process is the dynamically repressed unconscious wish that is released from control in sleep. The dream thoughts that emerge threaten consciousness and sleep; they are deterred by the censor. The "dream work" transforms the unconscious wish by the processes that are listed. The product, or manifest content, that becomes conscious thus contains only disguised elements of the original (latent) dream thoughts.
The activation-synthesis model is designed to contrast activation-synthesis theory with the guardian-censorship theory illustrated in the top portion of this figure. The motive force of the process is seen to be nonspecific neural energy or excitation hypothesized to arise from a nonspecific generator. This excitation affects the component systems of the forebrain represented in the upper box: sensory systems generate scene frames, structural fragments, and qualitative features; cognitive systems generate ideas that may be conscious (day residue thoughts) or unconscious (instinctually determined); emotion is also generated at this first stage. The dream report, easily obtainable if a state change to waking occurs, is seen as an accurate reflection of the integrated product of disparate, internally generated elements.

PSYCHOLOGICAL IMPLICATIONS OF THE CELLULAR NEUROPHYSIOLOGY OF DREAM SLEEP GENERATION

Hallucinoid dreaming is regarded as the psychological concomitant of D sleep. Brain activity in the D state has been analyzed to account for activation of the forebrain, occlusion of sensory input, blockade of motor output at the spinal cord level, and the generation of information within the system. The evidence that the pontine brain stem contains a clock-trigger mechanism that contributes to activation of the forebrain, occlusion of sensory input, and the generation of internal information has been reviewed. The periodicity of the triggering mechanism is hypothesized to be a function of reciprocal interaction of reciprocally connected, chemically coded cell groups in the pontine brain stem.

The psychological implications of this model, which we call the activation-synthesis hypothesis of the dream process (schematically represented in figure 10), contrast sharply with many tenets of the dream theory provided by psychoanalysis (also represented in figure 10) in the following ways:

1. *The primary motivating force for dreaming* is not psychological but physiological since the time of occurrence and duration of dreaming sleep are quite constant, suggesting a preprogrammed, neurally determined genesis. In fact, the neural mechanisms involved can now be precisely specified. This conclusion does not, of course, mean that dreams are not also psychological events; nor does it imply that they are without psychological meaning or function. But it does imply that the process is much more basic than the psychodynamically determined, evanescent, "guardian of sleep" process that Freud had imagined it to be; and it casts serious doubt upon the exclusively psychological significance attached to both the occurrence and quality of dreams.

2. *Specific stimuli for the dream imagery* appear to

arise intracerebrally but from the pontine brain stem and not in cognitive areas of the cerebrum. These stimuli, whose generation appears to depend upon a largely random or reflex process, may provide spatially specific information which can be used in constructing dream imagery; but the unusual intensity, intermittency, and velocity of the eye movements may also contribute to features of the dream experience which are formally bizarre and have been interpreted as defensive by psychoanalysis. Thus such features as scene shifts, time compression, personal condensations, splitting, and symbol formation may be directly isomorphic with the state of the nervous system during dreaming sleep. In other words, the forebrain may be making the best of a bad job in producing even partially coherent dream imagery from the relatively noisy signals sent up to it from the brain stem.

The dream process is thus seen as having its origin in sensorimotor systems, with little or no primary ideational, volitional, or emotional content. This concept is markedly different from that of the "dream thoughts" or wishes seen by Freud as the primary stimulus for the dream. The sensorimotor stimuli are viewed as possibly providing a frame into which ideational, volitional, or emotional content may be projected to form the integrated dream image, but this frame is itself conflict free. Thus both the major energetic drive for the dream process and the specific primary stimulus of the dream content are genotypically determined and therefore conflict free in the specifically psychodynamic sense of the term.

3. *The elaboration of the brain stem stimulus* by the perceptual, conceptual, and emotional structures of the forebrain is viewed as primarily a synthetic constructive process, rather than a distorting one as Freud presumed. Best fits to the relative inchoate and incomplete data provided by the primary stimuli are called up from memory, the access to which is facilitated during dreaming sleep. The brain, in the dreaming sleep state, is thus likened to a computer searching its addresses for key words. Rather than indicating a need for disguise, this fitting of phenotypic experiential data to genotypic stimuli is seen as the major basis of the "bizarre" formal qualities of dream mentation. There is, therefore, no need to postulate either a censor or an information degrading process working at the censor's behest. The dream content elaborated by the forebrain may include conflictually charged memories, but even this aspect of dream construction is seen as synthetic and transparent rather than degradative and opaque.

4. *With respect to the forgetting of dreams*, the normally poor recall is seen principally to reflect a state-dependent amnesia, since a carefully effected state change, to waking, may produce abundant recall even of highly charged dream material. There is thus no need to invoke repression to account for the forgetting of dreams. This hypothesis is appealingly economical, and in the light of the reciprocal interaction hypothesis dream amnesia can now be modeled in a testable way as the result of a different balance between cholinergic and aminergic neuronal activity and the resulting effects on second messengers and macromolecules (5). Among its other surprising gifts to psychophysiology, dreaming sleep may thus also provide a biological model for the study of memory, and a functional role for dreaming sleep in promoting some aspect of the learning process is suggested.

SUMMARY AND CONCLUSIONS

Assuming that isomorphism, or identity of form, must characterize the simultaneous physiological and psychological events during dreaming, we have reviewed the general and cellular neurophysiology of dreaming sleep in search of new ways of accounting for some of the formal aspects of dream psychology. We have noted that the occurrence of dreaming depends upon the periodic activation of the forebrain during sleep. We have hypothesized that the activated forebrain synthesizes the dreams by fitting experiential data to information endogenously and automatically generated by reticular, vestibular, and oculomotor neurons in the pontine brain stem. A specific physiological and mathematical model of the pontine generator, based upon single cell recording studies in cats, is described: the model posits reciprocal interaction between inhibitory aminergic (level-setting) and excitatory cholinergic (generator) neurons.

Some of the "bizarre" formal features of the dream may directly reflect the properties of the brain stem neuronal generator mechanism. The physiological features of the generator mechanisms and their corresponding psychological implications include the following: the automaticity and periodicity of activation indicate a metabolically determined, conflict-free energetics of the dream process; the random but specific nature of the generator signals could provide abnormally sequenced and shaped, but spatiotemporally specific, frames for dream imagery; and the clustering of runs of generator signals might constitute timemarks for dream subplots and scene changes. Further, the activation by generator neurons of diffuse postsynaptic forebrain elements in multiple parallel channels might account for the disparate sensory, motor, and emotional elements that contribute to the "bizarreness" of dreams; the suppression of motor output and sensory input simultaneous with central activation of both sensory and motor patterns could assure the maintenance of sleep in the face of massive central excitation of the brain; and the change in the ratio of neurotransmitters affecting forebrain neurons might account for dream amnesia and indicate a state-dependent alteration of neural plasticity, with implications for the learning process.

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Psychiatry, the Healthy Invalid

BY JEROME D. FRANK, M.D.

The author examines the reasons that have been offered in support of a pessimistic appraisal of psychiatry's future. The consequences for psychiatry of increasingly detailed knowledge of the interaction of psychological and physiological processes are emphasized, and the author concludes that psychiatry will continue to play a dominant role in its traditional domain and an increasing role in the study and treatment of medical and surgical illness. Thus, although the field is in a transition period, it is certainly not moribund.

THE IMPRESSION is abroad that psychiatry is seriously ill, if not actually at death's door. The thesis of this paper is that, although psychiatry is indeed experiencing distressing symptoms, they are not dangerous. Rather, most are analogous to the growing pains that accompany maturation.

Pessimists both within and without the profession support their judgment by enumerating various signs of illness. High on the list appears to be a widespread public disenchantment with psychiatry. It has become abundantly clear, for example, that psychiatrists do not have the answers to race relations, truancy, criminality, and war, and it is demonstrated repeatedly in courts of law that psychiatrists cannot agree on the mental state and degree of responsibility of defendants. Another criticism is that psychiatrists, in the guise of diagnosing and treating the mentally ill, are actually agents of social control, imprisoning deviants in mental hospitals by labeling them "ill" and then punishing them with electroconvulsive treatment, seclusion, and the like under the label of therapy. This accusation, most recently dramatized in the film "One Flew Over the Cuckoo's Nest," has led to a blizzard of lawsuits and judicial decisions concerning patients' rights.

The prophets of psychiatry's early demise also assert that psychotherapy conducted by psychiatrists is not cost-effective and that medical training does not provide any particular expertise in the use of psychotherapy. As a result, many people who previously sought psychiatric aid are flocking to nonmedical professionals such as psychologists, social workers, and mental health counselors, as well as to charismatic leaders who have developed their own therapeutic cults outside the boundaries of any profession.

Another development interpreted as a sign that psychiatry is in bad shape is the progressive withdrawal of public support, reflected, for example, in the phasing out of federal financing of psychiatric residency training programs, as a result of which the number of residents in training has been on a virtual plateau since 1972 and may be decreasing slightly. At the same time, the number of medical students has increased, so the proportion of medical school graduates who become psychiatrists has declined from about 10.5% to 9% (1).

A final apparently ominous sign is related to the rapidly increasing knowledge of biochemical and neurophysiological components of psychoses. It is claimed that nonpsychiatric physicians will increasingly take over the treatment of such illnesses, as they have done with the psychoses of pellagra and general paralysis of the insane.

Publicly discredited, harassed by the legal profession, suffering progressive shrinkage of federal support and of new recruits, and facing increasing competition from both nonmedical therapists and nonpsychiatric physicians, psychiatry would indeed appear to be in a parlous state.

THE ILLS OF PSYCHIATRY IN HISTORICAL PERSPECTIVE

In attempting to evaluate the validity of the dire diagnosis of psychiatry's state, it may be helpful to look briefly at the historical development of the current predicament. About two centuries ago psychiatry emerged as a medical specialty whose function was the care and treatment of the hospitalized insane. Psychiatry did not share in the spectacular gains of knowledge brought about by cellular pathology and the germ theory of disease and therefore became increasingly separated from the mainstream of medicine—mental hospitals became warehouses for segregating the mentally ill and havens for the less successful physicians. Public indifference to these hospitals permitted the growth of abuses such as ignoring patients' rights and failing to provide treatment. In the United States, reformist efforts to promote humane treatment of the mentally ill, led primarily by laymen, temporarily mitigated these abuses, but they were soon overwhelmed by the massive influx of immigrants from different cultural back-

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grounds. Most public mental hospitals reverted to human warehouses. Despite spasms of temporary improvement following public exposés, this deplorable state of affairs has only recently begun to change as a result of the introduction of antipsychotic medications and the development of community mental health programs.

In the meanwhile, toward the close of the nineteenth century, Sigmund Freud, on the basis of astute clinical observations, promulgated an intellectually dazzling, aesthetically appealing, and endlessly fascinating conceptualization of mental illness. Psychoanalysis created a brand new field of knowledge and therapy out of what had previously been chaos and demonstrated that a host of hitherto inexplicable symptoms and signs such as functional paralyses and anesthesias, compulsions, phobias, and obsessions could be treated psychologically. As a result, many patients with these complaints, especially the more educated and affluent, flocked to psychoanalysts for relief. The status, as well as the income, of psychoanalysts soared and, because of its intellectual challenges, psychoanalysis soon attracted the best minds in the field.

Psychoanalytic theories emphasized the role of stressful interactions with other persons in the production of neurotic symptoms. Initially, emphasis was on traumatic experiences within the family, which led to concern with child-rearing practices and then gradually to interest in the pathogenic role of all social stresses. Concomitantly, psychoanalytic theory expanded into a general theory of human nature. These developments created incentives for psychoanalysts to study and try to ameliorate all social problems. The high regard in which psychoanalysts were held, especially by the intelligentsia, contributed to the grandiose delusion of some that application of psychoanalytic principles could cure, or at least ameliorate, practically all social ills. Psychoanalysts offered their services as policy advisors to political leaders, without any first-hand knowledge of the problems to which they offered solutions. An extreme example was the assertion that the way to bring about world peace was to require that all political leaders be psychoanalyzed. Understandably, the credibility of psychoanalysts suffered.

The large number of psychiatric casualties of World War II, in conjunction with the overly optimistic evaluation of psychiatry by the public, led the federal government to provide lavish support for psychiatric training, research, and practice, resulting in a more than five-fold increase since 1948 in the number of psychiatrists. In the last decade the increase has been 51%—double that of physicians in general (1).

The discovery that bodily symptoms were often related to emotional states soon emboldened psychoanalysts to turn their attention to psychological aspects of medical illnesses, and by the 1930s they had established a firm beachhead in this area. The initial hope of relating specific psychodynamic conflicts to specific diseases remains to be fully realized, but psychiatrists did establish their usefulness to their medical colleagues, as reflected by the rapid spread of psychiatric liaison services.

An evaluation of the current criticisms of psychiatry in the light of this historical review suggests that the charges of grandiosity and arrogance were partly warranted in the past but have become increasingly ill-founded. Psychiatrists have become appropriately more cautious about hospitalizing mental patients involuntarily and have taken the lead in efforts to make mental hospitals genuine treatment centers and to promote community mental health care programs. They have also become more careful to inform themselves about the social and political problems for which they offer psychiatric insights and more modest about their contributions. In so doing, they are gradually regaining credibility.

The malpractice suits and administrative regulations that currently threaten to strangle all medical practice will almost certainly subside to an appropriate level as their damaging consequences for patient care and treatment become increasingly apparent.

The reduction of funds for psychiatric residency programs could be less a reflection of public disenchantment with psychiatry than a recognition that the specialty no longer needs and should not continue to receive special care and feeding. Psychiatry is now sufficiently well established for its training programs to be put on the same financial footing as those of the other branches of medicine.

Although members of other helping professions often can do as good a job of psychotherapy as psychiatrists, the fact is that there is no evidence of slackening of demands for psychotherapy by psychiatrists. The healing aura that surrounds the medical degree, the psychiatrist's indoctrination into the assumption of full responsibility for patients, and his residual familiarity with disorders of the body all assure that the psychotherapeutic services of psychiatrists will be sought for some time to come.

The prediction that nonpsychiatric physicians will take over the treatment of psychoses seems highly unlikely. Psychotics are uncomfortable patients to deal with, and most physicians are only too happy to relinquish their care to psychiatrists. Moreover, pellagra and paresis have a single, easily eradicated organic cause. There is ample evidence that the great majority of psychoses, including the group of disorders called schizophrenia, have multiple causes and that proper management requires continuing attention to their psychological aspects. More fundamentally, the assumption that any disease in which bodily disorders play an important role is best treated exclusively by internists or surgeons implies a dualistic view of human functioning that is increasingly untenable.

Interactions of psychological and bodily processes form a seamless web; this is as true of medical and surgical conditions as of psychiatric ones. This recognition is the result of the explosive advances in detailed quantitative knowledge of the relationships of

so-called mental and bodily phenomena made possible by new instrumentation and the use of computers for multivariate data analysis. Among the new instruments are the electron microscope, fluorescent dyes that permit visualization of neuronal tracts and cells containing different neurotransmitters, microassays of circulating hormones, and computer analyses of EEG.

RESEARCH ADVANCES IN MIND-BODY INTERACTIONS

A few representative examples of newer findings concerning the interaction of psychic and bodily processes may indicate the variety and promise of advances in the area of mind-body interactions. I have grouped the material in four categories: clinical research in certain diseases, harmful and beneficial psychological effects of medical and surgical procedures, interactions of psychological states with hormones and immune responses, and some relations of brain functions to consciousness.

Clinical Research Studies

Considering first advances in the specification of the relation of bodily and psychic states, an intriguing example at the clinical level is reversible somatotropin deficiency, or psychosocial dwarfism—a lag in growth and maturation found in boys who have been raised in a stressful home environment; they promptly start to grow when removed from the home. A likely explanation is that production of the pituitary growth hormone increases during sleep, suggesting that the child's failure to grow may be related in part to inability to sleep in the disturbed home (2).

That mental states may affect the course of infectious diseases was shown by studies of the relation of depression to delayed convalescence in brucellosis and influenza (3). The influenza study was prospective and therefore more conclusive. All the personnel of a military installation were given a paper-and-pencil test of depression before an anticipated outbreak of influenza. Those who became ill were divided into two groups: those who recovered in an average of about a week and those who continued to complain for at least three weeks. The severity of the initial attack, judged by a variety of tests, was the same for both groups, but the slow recoverers had scored significantly higher on the scale of depression.

Since symptoms of influenza resemble those of depression (lassitude, weakness, vague somatic complaints), this finding could be explained by the assumption that patients and doctors were mistaking depression for delayed convalescence. To exclude this possibility, a strictly objective criterion of improvement would be needed. One such carefully designed study exists, conducted by three clergymen who were also trained psychologists. The criterion was speed of healing of a detached retina. Patients were interviewed before the operation and their "acceptance" was rated on a paper-and-pencil scale that included such items as trust in the surgeon, optimism about outcome, confidence in ability to cope regardless of outcome, and preparedness to accept the bad with the good. Speed of healing was rated by the surgeon independently and without knowledge of the test scores. A significant correlation was obtained between acceptance and speed of healing ($p<.001$), leading the authors to conclude that "high acceptance and rapid healing occur . . . when the patient has faith in the healer, his methods of healing, and feels that these methods are relevant to the cause of his illness . . . the person seeking to help the slow healer . . . should focus primarily on what variables enhance or destroy the patient's attitude of expectant faith" (4, p. 140).

If psychological states are related to susceptibility to infection and speed of healing, it should not come as a surprise that they apparently also affect the course of cancer. Western medicine has conceptualized cancer as an invasion of the body from inside by cells that have gone wild, and treatment consists of an effort to remove or destroy them by surgery, radiation, or drugs. The outcome of this struggle is determined by whether the cancer cells or the destructive agents are stronger. The patient is simply the arena in which the battle is fought—he cannot influence the outcome. However, this theory cannot explain spontaneous remissions of definitely diagnosed cancer. Two surgeons have accumulated 176 examples of such remissions from the literature but offer no explanation for them (5).

Recently, there has emerged the so-called surveillance theory of cancer, which offers a possible explanation for at least some of these spontaneous remissions and suggests additional therapeutic approaches. According to the surveillance theory, cancer cells are formed throughout life but are promptly detected and destroyed by the person's immune system. Clinical cancer appears when this system fails. Hence, factors that influence immunity, including psychic ones, are relevant to both the emergence and treatment of cancer.

What is the evidence in support of this hypothesis? First, an increasing number of studies have found that persons with a certain personality style are especially prone to cancer. Compared with matched healthy controls or those suffering from other illnesses, they repress or deny unpleasant affects such as depression, anxiety, and hostility (6). Under a facade of cheerfulness and self-confidence, they feel isolated and view life as a hard struggle. The finding that these chronically stressed persons may have heightened susceptibility to cancer is consistent with the relation of adrenal cortical activity to immunity, which I will consider presently.

The cancer patient's attitude toward life seems related to early life experiences that led him or her as a child to feel that close emotional relationships brought pain and destruction (7). This retrospective finding has

recently found indirect confirmation from a prospective study of male medical students who on entering medical school were given a battery of tests that included a rating scale of closeness to parents. Those subjects who had developed malignant tumors 10–20 years later were found to have scored significantly lower on this scale than controls matched by age and class who were in good health (8).

Psychological Effects of Medical and Surgical Treatments

As is suggested by the study of surgery for detached retina mentioned earlier, medical and surgical treatments have psychological repercussions that may significantly affect their outcomes. Two medical procedures with noxious psychological concomitants that warrant the attention of psychiatrists are hemodialysis for renal failure and intensive care treatment for coronary disease. That hemodialysis is enormously stressful emotionally is evidenced by a suicide rate of 6%, which is approximately 400 times greater than the overall incidence in the general population. This finding cries for more study of personality attributes of dialysis patients that predispose them to or protect them from such a tragic outcome, as well as methods for helping them to endure the strain. In this connection, it is striking that patients undergoing hemodialysis in hospitals have about 9 times the suicide rate of those receiving treatment in their own homes (9). An analogous finding emerges from the investigation of the treatment of acute myocardial infarction. Data are accumulating which indicate that the death rate at 28 days following the infarction is as high for patients treated in intensive care units as for those who remain at home (10). Apparently the psychologically damaging effects of being transferred to a hospital and, perhaps, of some of the procedures in the intensive care unit outweigh the benefits of the availability of equipment for monitoring cardiac function and combating ventricular fibrillation and cardiac arrest.

On the positive side, all medical and surgical procedures incorporate the beneficial psychological effects of the patient's belief in the physician's healing power. This is customarily labeled the placebo effect, an unfortunate term because it implies that the effect is illusory and based on deception. Investigations have shown that the patient's response to placebo results from a complex interaction between his momentary state, the physician, and other aspects of the situation (11). Overall, however, placebo is a powerful remedy. In fact, it can be argued that until this century the reputation of physicians rested primarily on the placebo effect because, with the exception of a few drugs such as quinine, digitalis, and morphine, most of the concoctions they administered were at worst toxic and at best pharmacologically inert (12).

Double-blind studies comparing placebo with active medication for the relief of subjective complaints have repeatedly found the effect of placebo to be about half that of the medication, regardless of the latter's pharmacological efficacy. For example, placebo is about half as effective a pain reliever as both propoxyphene HCl and morphine, although pharmacologically morphine is a much more powerful analgesic. Apparently, the doctor somehow conveys to the patient his knowledge of the potency of the active drug (13).

If medication can have powerful placebo effects, one would expect surgical operations to have even stronger ones. Compared with a person seeking medical treatment, the surgical patient is more apprehensive and is totally dependent on the surgeon—he literally places his life in the surgeon's hands. Furthermore, the operation is a single dramatic act that is expected to produce a prompt cure. This description applies especially to operations on the heart, so it should not come as a surprise that their psychological effects may account for much of their success. This was first suggested by the fact that approximately 40% of patients who underwent ligation of the internal mammary artery for relief of anginal pain experienced marked symptomatic relief, including reduced pain and increased exercise tolerance without ECG changes. Unfortunately, a sham operation produced identical results (14), so the procedure was abandoned; it has been superseded in recent years by coronary bypass, which is currently enjoying a widespread vogue. Its rationale is completely convincing: angina is caused by poor circulation to part of the heart muscle resulting from partial occlusion of a coronary artery and therefore can be cured by restoring the circulation through bypassing the diseased portion of the artery with a segment of vein.

The reported surgical results have been spectacular. About 90% of patients report reduction of symptoms and improved quality of life and about 75% also can tolerate more exercise without pain or ECG changes. It would appear absurd to question that this improvement is caused by better blood flow to the heart as the result of the bypass. Comparisons with patients receiving medical treatment, however, have raised questions as to how much benefit is actually due to improved cardiac circulation. When compared with a matched group of patients treated medically, patients given surgery showed no definite evidence of improvement in life expectancy (15). Furthermore, on an objective test of ventricular function, about 44% showed no change, 38% were actually worse, and only 18% showed some improvement (16). In short, although symptomatic improvement occurred in 90%, there was improvement in heart function in only about 20%; therefore, 70% improved in the face of an unchanged or further deteriorating heart muscle. Although the issue is currently controversial, a reasonable conclusion, especially in light of the earlier experience with ligation of the internal mammary, is that improvement in a significant proportion of patients is due to the psychological effects of the operation, especially the patient's expectant faith. This is powerfully reinforced by a convincing rationale according perfectly with the scientific Weltanschauung of our culture and a spec-

tacular procedure that involves stopping the patient's heart and then reviving him. In a sense, the surgeon kills the patient and brings him to life again. Few shamans can demonstrate equal healing power.

Neurochemical and Neurophysiological Studies

Findings in neurochemistry and neurophysiology create new potential clinical roles for psychiatrists in the treatment of patients whose emotional states may affect the course of their organic illness. Their full contribution, however, depends on the ability to elucidate the biochemical and physiological processes, especially in the central nervous system, that mediate the interactions between bodily and psychological states. It is in specifying these intermediate processes that progress, although still fragmentary, has been especially promising.

Findings from an extensive series of studies on the interaction of circulating norepinephrine and epinephrine with psychological stress may serve as examples. If army recruits with different levels of circulating hormones under stress are placed in a group for an extended period of time, the levels gradually cluster as if under the influence of emotional contagion. Another provocative finding is that recruits who have lost a mother in childhood show a much greater activity of the adrenal cortical system than those who have lost a father (17, 18). This accords well with findings in groups of working women that 1) circulating levels of blood cortisol are increased in depression and 2) loss of the mother before age 12 predisposes the child to depression as an adult (19).

The finding that stress during basic training, which is accompanied by a rise in circulating cortisol, increases the susceptibility of recruits to virus infections accords with the clinical material presented earlier on the relation of depression to delayed convalescence from influenza and to possible susceptibility to cancer. These are a few examples of a variety of findings that may be summed up as follows:

> Psychosocial processes influence the susceptibility to some infections, to some neoplastic processes and to some aspects of humoral and cell-mediated immune responses. These psychosocial effects may be related to hypothalamic activity . . . there is no single mediating factor. (20, p. 439)

Brain Function and States of Consciousness

One exciting area of research is the exploration of differences in function of the right and left cerebral hemispheres. As is now widely known, the left hemisphere mediates analytical thinking and language, and the right mediates intuitive processes and responses to patterns of stimuli. For example, activation, as indicated by suppression of alpha, occurs in the right cortex when a person hums the tune of a song, in the left when he recites the words, and in both when he sings the words (21). Such findings may explain why some intense aesthetic experiences and mystical states, which seem to be accompanied by predominantly right hemispheric activity, cannot be described in words.

A realm of research into bodily concomitants of states of consciousness that may have far-reaching therapeutic implications relates to Transcendental Meditation, Zen, Yoga, and other spiritual exercises originating in the mysterious East. Because of their origins, these techniques had little influence on Western research until advances in instrumentation made them legitimate objects of scientific research. Recent investigations have revealed that all forms of meditation produce changes in oxygen consumption, heart rate, blood pressure, skin resistance, and other physiological functions. These changes, collectively termed the relaxation response, are indistinguishable from those accompanying any state of relaxation coupled with focused attention (22). Whether various forms of meditation differ with respect to other bodily concomitants remains undetermined.

Biofeedback training as an adjunct to relaxation enables a person to gain extraordinarily precise control of body functions. Among promising clinical applications of relaxation and biofeedback are reduction of systolic blood pressure, correction of cardiac arrhythmias, relief of migraine, and control of fecal incontinence and of painful spasms of skeletal muscles. It is important to stress that biofeedback training is essentially a form of psychotherapy. As the Yogis have shown, the same results can be achieved without the gadgetry, and clinical success depends largely on the strength of the patient's motivation, which in turn is strongly influenced by his relationship with the trainer (23). Moreover, much of the therapeutic benefit of biofeedback training may be in its demonstration to the patient that he can control a bodily function, which heightens his self-confidence in other areas of his life.

States of relaxation are precursors of the more profound alterations of consciousness that occur with meditation. In these, persons may experience phenomena outside the realm of normal waking consciousness, including telepathic experiences, precognition, "out-of-the-body" experiences, and mystical revelations. This brings us to the edge of the seductive and fascinating realm of the paranormal, which is so studded with booby traps and quicksand that one must approach it with the greatest circumspection. A full consideration of the paranormal would lead us too far afield, but it can be said that an increasing number of studies of paranormal phenomena such as telepathy and healing by laying on of hands, which may be relevant to psychiatry, have been conducted by researchers of unquestionable integrity and meet acceptable standards of scientific validity (24–26).

More spectacular than the growth of specific information concerning the effect of psychic states on bodily functions is the exponential increase of detailed information on the effects of brain function on psychic states. Since reports of psychopharmacological studies currently provide much of the content of psychiatric journals, I will not attempt to review this bur-

geoning field. A single example will suffice: the discovery that drugs which decrease dopamine activity, such as phenothiazines, may be antipsychotic and drugs that promote dopamine activity, such as amphetamines, may be psychotomimetic, has triggered a veritable explosion of research guided by the hypothesis that schizophrenic symptoms may be related to a relative excess of dopamine-dependent neuronal activity. Although these studies have not yet produced irrefutable support for the hypothesis, the cumulative findings have been described as "compelling" (27).

DISCUSSION AND CONCLUSIONS

This sampling of studies illustrating the close interaction of mental and bodily processes sharply reveals the shortcomings of the dualistic view of human functioning, which has never solved the dilemma of conceptualizing just how mind and matter can influence each other. A possible way out, sufficient at least for present purposes, is provided by the so-called holistic view of the organism as an open system interacting with the environment through continuous exchanges of matter, energy, and information. Informational input affects biological processes and, if sufficiently intense and enduring or occurring early enough in life, can actually alter structure, as is suggested by psychosocial dwarfism. Biological changes, in turn, modify the individual's perceptions and behavior, providing new inputs into the system to complete the circle. The division of this process into causes and effects is essentially arbitrary, resulting from the need to categorize experience. All aspects of human functioning are simultaneously causes and effects of other aspects.

In holistic terms, health is defined as a system in harmony with itself and its environment. All diseases disrupt both the individual's internal integration and his interchanges with the environment at both biological and psychological levels. The task of the healer is to help to restore the harmony of this system within itself and in its interactions with the outer world.

Although most of the studies reported in this paper were conducted by psychiatrists, psychiatric training programs must be modified considerably if psychiatrists are to take full advantage of the new opportunities and challenges emerging from holistically oriented research. Current emphasis on psychological aspects of personal functioning must be balanced by more attention to neuroendocrine and other bodily processes, with particular emphasis on detailed studies of their interactions.

Psychiatrists should be in the best position to take advantage of these developments. In contrast to other medical researchers whose training stresses biological processes and to nonmedical researchers whose expertise lies in the understanding of psychological states, psychiatrists are concerned with both biological and psychological phenomena. They sit astride the mind-body boundary, as it were—a somewhat uncomfortable position, but one that is proving to be increasingly important for many aspects of medical research and practice. As the contributions of psychiatry in this realm increase, an increased interest by medical students in psychiatric residency training can be confidently predicted.

With respect to other aspects of psychiatry's future, the information I have presented supports the prognosis that psychiatry, rather than being on the verge of death, is about to enter a period of vigorous health. Psychiatrists are becoming more realistic, as befits the attainment of maturity. We are shedding some of our infantile omnipotence and our dependence on father figures like Sigmund Freud and Uncle Sam.

With respect to social and political problems, it is sobering to realize that human social behavior now looms as the greatest potential cause of disability and death. Uncontrolled breeding, reckless pollution of the biosphere, inadequate control of radioactive wastes from nuclear power plants, and resort to nuclear weapons in war or terrorism are forms of behavior that pose a far greater threat to humanity than naturally occurring pathogens and environmental disorders. While preserving a becoming modesty about their ability to resolve these dangers, psychiatrists should not allow their regret for earlier mistakes to prevent them from accepting the responsibility to contribute what they can. As students of maladaptive behavior and communication failures, with some experience in how to correct them, psychiatrists should be able to make significant, if limited, contributions toward the control of these man-made health hazards.

With respect to psychiatrists' traditional roles, although a growing number of the unhappy and alienated may gravitate to nonmedical helpers, there will be no dearth of individuals seeking psychiatric help. Further, it is not likely that nonpsychiatric physicians will be eager to assume responsibility for treatment of the severely mentally ill. In this connection, studies of population trends for the coming decade project an increase in schizophrenia of from 5% in whites aged 15–24 to 87% in nonwhites aged 25–34, so psychiatrists will not suffer from lack of patients (28).

While psychotherapy will continue to be our major form of treatment, our methods will expand to include behavioral techniques, biofeedback, and other psychobiological approaches. We will also continue to incorporate into our treatment armamentarium the growing body of information about psychoactive drugs, including their interaction with psychotherapy. In this connection, a recent study has found that some severely depressed patients respond to cognitively oriented psychotherapy alone at least as well as to imipramine (29), and a leading researcher in the treatment of schizophrenia has concluded that although the efficacy of pharmacotherapy is unquestionable, psychologically oriented forms of treatment are also necessary (30).

Finally, psychiatrists will contribute increasingly to the understanding and treatment of all disease, based

on expanding knowledge of interactions of mental states, behavior, and bodily processes as mediated by the nervous and endocrine systems.

All in all, while psychiatry may emerge from its current travails somewhat altered in appearance, its future is bright.

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Social Discrediting of Psychiatry: The Protasis of Legal Disfranchisement

BY PARK ELLIOTT DIETZ, M.D., M.P.H.

The author believes that recent legal decisions against psychiatrists (including class action suits) have their genesis in writings by sociologists and other behavioral scientists, including psychiatrists, who have examined psychiatry in the context of social structure and social processes. The discrediting of the medical model could have been accomplished using theoretical assertions and research observations that had been published before 1940, but it did not occur until the human rights movement of the 1960s. The author reviews the assertions on which the discrediting of the medical model has been based. He stresses the distinction between the testing of a theory and the discrediting of a model and observes that the recent wholesale discrediting of psychiatry has relied less on theory testing than on polemical rhetoric and unwarranted extrapolation from empirical facts.

PSYCHIATRISTS HAVE become increasingly concerned about the proliferation of class action suits (such as *Wyatt v. Stickney* [1] and *Donaldson v. O'Connor* [2, 3]), legislative efforts (such as the Vasconcellos bill in the California Assembly [4]), and judicial decisions (such as in the Tarasoff case [5]) that would restrict their activities. No formal survey is needed to gauge psychiatrists' attitudes toward these developments. Most psychiatrists perceive such legal actions as unjustified attacks on psychiatry by attorneys and other outsiders, and they fear that the success of such actions will seriously handicap their efforts to care for patients.[1] Whether this perception is accurate does not concern us in this paper. The issue to be addressed here is the origin of this perception, which has an important influence on the professional identity and morale of many psychiatrists. This paper identifies sociological writings as a major source of both actions in mental health law and the perception that such actions comprise an attack. "Sociological writings" refers not only to the writings of sociologists, but also to a number of seminal works by psychiatrists, psychologists, and others who have examined psychiatry in the context of social structure and social processes.

The thesis of this paper is that the controversial actions in mental health, and psychiatrists' perceptions of these actions as an attack, are direct consequences of such sociological writings as those of Faris and Dunham, Edwin Lemert, Hollingshead and Redlich, Thomas Szasz, R.D. Laing, and Thomas Scheff. Empirical evidence for this thesis would lie in a study of the reading habits of attorneys, judges, legislators, and psychiatrists, and such a study has not been conducted (although some related evidence will be presented). But before the thesis is rejected out of hand for lack of sufficient evidence, it should be noted that Birnbaum (6) wrote the first article on the right to treatment while living in the company of sociologists as a research fellow in social relations at Harvard.

Insofar as mental health law advocates are motivated by humanitarian concerns, their major concern would seem to be that psychiatrists and psychiatric institutions may do harm to patients. One of our tasks, then, is to identify the intellectual roots of this concern. But the concern itself is not enough to produce social change. Illich (7) has recently argued that medicine in general does more harm than good, but it is safe to predict that his concern will not result in the closing of general hospitals or in attempts at legislative control of appendectomies or hemodialysis. The prerequisite for such change is a discrediting of the scientific and moral status of the profession. This prerequisite was fulfilled in the case of psychiatry by a host of writings

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[1]For example, the theme of the meeting for which this paper was originally prepared was "Psychiatry Under Siege," a reference to this spate of legal actions.

condemning the use of the medical model[2] in psychiatry and implying that psychiatrists are malevolent creatures who have conspired to impose their world view on unsuspecting patients. Our basic task, then, is to identify the intellectual roots of the discrediting of the medical model in psychiatry and the discrediting of the moral character of psychiatrists.

DISCREDITING OF THE MEDICAL MODEL

A compendium of papers edited by Rose and published in 1955 (8) marked the end of an era in psychiatric sociology, the era in which conventional psychiatry was given the benefit of the doubt. The papers in this volume were not influenced by Lemert, Goffman, Szasz, Laing, and Scheff; they made no mention of the medical model, labeling theory, or the social control functions of psychiatry, all of which characterize later works. Bowman's paper (9) provides an extraordinary example of the extent to which the legitimacy of psychiatrists was taken for granted by sociologists of this era. Bowman argued that "just as the psychiatrist is competent to judge degrees of reality or unreality in the patient's interpretations, so the social scientist is equipped to determine the distortions of collective thought . . ." (9). Thus not only was the psychiatrist's role as the official judge of individual reality reaffirmed, but an attempt was made to adduce an analogous status to social scientists. This contrasts sharply with later writings, which contained the common theme that psychiatrists lack both the valid means and legitimate authority with which to judge reality.

As we shall see below, the discrediting of the medical model in psychiatry drew support from several theoretical assertions and research observations. It is noteworthy that the essence of this theory and research had already been published by 1940, but received little attention at the time. Eliot's paper on "Interactions of Psychiatric and Social Theory Prior to 1940" (10), also in the Rose volume, provides many examples of the kinds of assertions that later were used to discredit the medical model in psychiatry.

In the 1920s Eliot had written of "insanity" as a social situation rather than as a personal trait and had dealt with the cultural defining of "mental disease" situations and with the situational causes of "mental diseases." In the 1920s and 1930s Anton Boisen studied religious ideologies, ethical conflicts, and social life histories as factors of causation among institutionalized mental patients. In the 1920s Herbert Adolphus Miller coined the phrase "oppression psychosis" to describe typical reactions observed among minority groups. In 1930 Robert E. Park suggested that the study of cultural conflicts would shed light on functional disorders. In 1932 a session of the American Sociological Society's annual meeting was devoted to the cultural patterning of "psychotic" states peculiar to certain cultural areas. In the 1920s and 1930s Trigant Burrow, Lawrence Frank, and the essayists contributing to a volume edited by Schmalhausen all suggested that it is the civilized world which is insane and that we have no absolute criteria by which to differentiate mental illness from genius, sainthood, or vice. It was in the 1930s that Faris and Dunham and Lee Brooks documented ecological variations in the distribution of psychiatric disorders. In the 1930s Maurice Krout described the task of social psychiatry as the discovery and control of social causes of abnormal states. All of the examples in this paragraph are cited in Eliot's historical review, arbitrarily limited to the period 1915–1940; no doubt many earlier examples could be cited by a psychiatric historian.

Except for Faris and Dunham, the authors just cited are unfamiliar to psychiatrists today and are probably equally unfamiliar to mental health law advocates. Yet these same ideas, as they reemerged in the late 1950s and thereafter, were used to discredit the medical model. That they were not so used in the 1930s and 1940s is amply demonstrated by the success of Deutsch's 1937 book, *The Mentally Ill in America* (11), which is both a documentary of the unsatisfactory conditions provided for the mentally ill and a celebration of the emergence of the medical model in psychiatry.

As mentioned above, the discrediting of the medical model drew support from several theoretical assertions and research observations. The critical ones seem to be the following.

1. Interpersonal relationships are important in the pathogenesis of mental illness. This idea, which was common to Freud and any number of other psychiatric theorists as well as to the social theorists of the 1920s and 1930s, is written about today as though it were first discovered in the 1950s and 1960s, in the important family studies of Bateson and associates (12, 13) and Lidz and associates (14, 15).

2. The frequencies of different types of mental illness vary among socioeconomic classes. This observation, noted as early as 1856 (16) and well established in the 1930s, was replicated and expanded in the 1950s and 1960s, most notably by Hollingshead and Redlich (17) and by Srole and associates in the Midtown Manhattan study (18).

3. "Mental illness" is in the eye of the beholder. A relativistic concept of mental illness was put forward by a number of authors in the 1920s and 1930s, but it was not widely diffused until the publication of widely read books by Szasz (19) and Laing (20, 21) in the 1960s.

4. The chronicity of mental illness is ensured by the official labeling done by psychiatrists and the process of institutionalization occurring in mental hospitals.

[2]The phrase "the medical model" has a variety of referents in the sociological and psychiatric literatures. It is used in this paper to denote a predominantly organic ideology regarding the etiology, pathogenesis, and treatment of psychiatric disorders. Most of the writers whose works are cited in this paper have supposed or implied that this is the most prevalent ideology among psychiatrists; the fact that this has not been true for several decades has not lessened the impact of their writings.

Although it has long been obvious that facilities of the snake-pit variety could only serve to prolong the necessity for confinement, it was not until the 1950s that a major theory arose which suggested that any public or official categorization of a person as being "mentally ill" or "a mental patient" is a strong determinant of chronicity. This thesis grew from Lemert's effort (22) to account for the tendency of radicalism, criminality, alcoholism, mental disorder, and other conditions to progress or worsen. Lemert's work, which became widely known in the 1960s (e.g., through Becker [23]), has been extremely influential in contemporary sociology. Lemert is generally credited with constructing "labeling theory," which has been applied most extensively to psychiatric disorder by Scheff (24, 25). The pervasive effect of institutionalization on the lives of mental patients, rationalized largely by means of the medical metaphor, has been described most persuasively by Goffman (26).

These four propositions underlie the wholesale discrediting of the medical model in psychiatry. It is important to distinguish between the testing of a theory and the discrediting of a model. Theory testing relies upon logic and controlled observation, both of which have been applied to selected aspects of the medical model. Examples of logical and observational approaches include some of Szasz's original arguments in *The Myth of Mental Illness* (19), the derivative arguments of Albee (27, 28), and the empirical studies by Scheff (29), Mendel and Rapport (30), and Greenley (31).

In contrast, the wholesale discrediting with which we are here concerned relies upon polemical rhetoric and unwarranted extrapolation from empirical facts. Thus the first and second propositions, that interpersonal relationships are important in the pathogenesis of mental illness and that the frequencies of different types of mental illness vary among socioeconomic classes, are transmogrified into the assertion that the causes of mental illness are interpersonal and social, not medical.

Similarly, the third proposition, that "mental illness" is in the eye of the beholder (a proposition that reflects difficulties of diagnosis, cross-cultural and other variations in conceptualization, and the widely acknowledged difficulties of applying a disease model to personality disorders, sexual deviations, and situational adjustment reactions), is transmogrified into the assertion that mental illness (including schizophrenia and the affective disorders) is mythological and does not exist. The behaviors that under the medical model are called mental illness thus come to be seen as "problems in living" (19), "residual rule violations" (24), or "interpersonal disjunctions" (32, p. 67).

Finally, the fourth proposition, that the chronicity of mental illness is ensured by the official labeling done by psychiatrists and the process of institutionalization occurring in mental hospitals, is transmogrified into the assertion that psychiatrists and mental hospitals create, perpetuate, and exacerbate "mental illness."

These transmogrifications of the four propositions described above, if accepted, provide ample reason to reject the medical model in psychiatry. Taken together, they lead to the conclusion that social and interpersonal factors cause problems in living which, under the influence of psychiatrists and mental hospitals, are transformed into chronic "mental illnesses" such as "schizophrenia." This conclusion entails more than a scientific rejection of a theory; it constitutes what we have called the wholesale discrediting of the medical model.

MORAL DISCREDITING OF PSYCHIATRISTS

The discrediting of the moral character of psychiatrists has occurred much more abruptly. As illustrated previously, the legitimacy of psychiatrists was taken for granted for many years. As recently as 1966 it could be said that there were only a few sociologists "who tend to picture the psychiatrists' relationships with patients less as treatment than as work in the interest of the psychiatric establishment" (33). This has changed dramatically in the course of a decade, and there now are a great many sociologists and a large cohort of college graduates who view psychiatrists in an unfavorable light. Some of the better-known sources serving to discredit psychiatrists are as follows.

1. Hollingshead and Redlich's finding (17, pp. 253–303) that patients from lower socioeconimic classes are disproportionately more likely to receive organic therapies than are patients from upper socioeconomic classes, who are disproportionately more likely to be treated with psychotherapeutic methods. (These researchers specified that they did not believe that this was the result of deliberate policy.)
2. Goffman's observation (26, pp. 155–162) that the psychiatric case history is a dossier of discrediting facts and statements about the patient.
3. Kesey's fictional account (34) of the punitive use of electroconvulsive therapy and psychosurgery.
4. Szasz's claim (35) that "coercion and deceit are rampant in the legal and social uses of psychiatry—beginning with the pseudomedical definitions of mental illness, and ending with the tyrannical abuses of psychiatric power in cases of political and other offenses."
5. Laing's description (32, pp. 121–122) of the labeling of "schizophrenia" as a political event in which a person is inaugurated into a patient career "by the concerted action of a coalition (a 'conspiracy') of family, G.P., mental health officer, psychiatrists, nurses, psychiatric social workers, and often fellow patients."
6. The Medvedevs' account (36) of the use of psychiatry for political repression in the Soviet Union.
7. Halleck's thesis (37) that no aspect of psychiatry is politically neutral.
8. Torrey's description of preventive psychiatry as "the road to psychiatric fascism" (38).

Each of these eight sources casts doubt on the morality of psychiatric practice without even invoking the additional controversies posed by involuntary commitment. To these may be added the findings of Rosenhan's study (39), generally interpreted as indicating that psychiatrists cannot even distinguish the "sane" from the "insane." These assertions are not merely criticisms of psychiatrists; if accepted, they constitute a wholesale discrediting of psychiatrists. It is a discrediting that seems to have been anticipated by Davis (40), who in 1938 wrote a paper documenting the extraordinary bias of the mental hygiene movement (and, by implication, psychiatry) in promoting conventional values, a bias that could not be tolerated indefinitely from a profession with scientific pretensions. Although the above cited sources have been most influential, it should be noted that the discrediting process has been carried even further by a number of self-proclaimed radicals (41–43).

DISCUSSION

The basic task of identifying the intellectual roots of the discrediting of both the medical model in psychiatry and the moral character of psychiatrists having been fulfilled, it remains to be shown how these various assertions, which are herein claimed to be sociological, relate to the perception that psychiatry is under attack. In this regard there are three points to be made.

First and foremost is the fact that these assertions, their transmogrifications, and their radical extrapolations could only be accepted in a society characterized by a plurality of values and growing concern for human rights. Risking the fallacy of *cum hoc, propter hoc*, I suggest that the widespread acceptance of the discrediting views discussed above, many of which were shown to have been espoused in the 1920s and 1930s, did not occur until the 1960s because it was not until then that political activism and concern with human rights were sufficiently widespread. It is noteworthy that Rappeport (44) recently attributed major importance to activism and the human rights movement in the genesis of the mental health lawsuits with which we are concerned; this theme has been extensively developed by Stone (45). It is suggested here that the social climate of activism and concern for human rights also contributed indirectly to the propagation and success of these suits by fostering acceptance of the discrediting ideas that stripped psychiatry and mental institutions of their thin cloak of scientific and moral legitimacy.

Second, the discrediting sources described above have had a direct bearing on mental health lawsuits and are considered to be resource material by mental health lawyers. Hollingshead and Redlich (17) were cited in an amicus curiae brief in *Legion v. Weinberger* (46). Goffman (26) was cited in the respondent's brief in *O'Connor v. Donaldson* (47). Szasz (19, 35) was cited in the petitioner's brief in *Donaldson v. O'Connor* (48). Further, the Midtown Manhattan study (18) was cited in an amicus curiae brief in *Glassman v. New York Medical College* (49). The three-volume book *Legal Rights of the Mentally Handicapped*, edited by Bruce Ennis and Paul Friedman under the auspices of the Practising Law Institute and the Mental Health Law Project (50), is a unique sourcebook of materials on mental health law. The annotated bibliography of this sourcebook includes many of the discrediting sources described above, namely, Goffman (26), Halleck (37), Kesey (34), Laing (20, 32), Rosenhan (39), Scheff (24), Szasz (19, 35), and papers from the Radical Therapist Collective (41), as well as a variety of discrediting sources not mentioned above.

Finally, the third way in which the discrediting assertions reviewed above have contributed to the perception that psychiatry is under attack is that they have become inextricably linked to those legal actions which pose practical problems for psychiatrists. McGarry (51) and Rappeport (44) have noted that the optimism of psychiatrists and administrators in the early days of the right-to-treatment lawsuits was short-lived, for it soon became clear that the means by which judicially mandated reforms might be implemented were not forthcoming. Thus there developed what McGarry has called "The Holy Legal War Against State-Hospital Psychiatry" (51) and what Rappeport has called the "belegaled" state of contemporary psychiatry (44).

Although it is too early to judge adequately the effects upon our patients, there are psychiatrists who believe, as I do, that sociological criticisms and mental health legal actions will have a net beneficial effect on patient care. Nonetheless, it seems clear that a substantial proportion of the psychiatric community has an understandable disdain for the discrediting and disfranchisement described in this paper. To the injuries of increased malpractice hazards, expensive and time-consuming paperwork and court appearances, and increasing limitation of the discretionary power of psychiatrists have been coupled the insults of scientific and moral discrediting. It is no wonder that psychiatrists perceive themselves as being under attack in an era in which their discreditors are cited as authority in briefs for the courts.

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Emotional Causes of Sudden Death

BY JOEL E. DIMSDALE, M.D.

The author reviews concepts important to understanding the emotional causes of sudden death, which occurs in the psychological setting of hopelessness, powerlessness, and intense emotions. The cardiovascular system responds to stress in a way that makes sudden death more likely. Studies pertinent to the effect of stress on blood pressure and arrhythmia in animals and man indicate that sudden death as a response to stress can occur only when certain factors are present simultaneously. Collaboration between psychiatrists and cardiologists is necessary for further progress in this field.

THE RELATIONSHIP of emotion to sudden death is an important area because of the widespread occurrence of sudden death and because of persistent theories about the role of emotion in its causation. Unfortunately, there is a paucity of work that combines consideration of emotional variables with their possible physiological mediators. I will discuss various theories of emotional causation of death with special reference to their possible physiological modes of action.

Sudden death from heart disease claims 300,000 Americans per year (1). The belief that sudden death can be associated with emotional flooding has enjoyed a long popularity in common folklore. In what must be one of the earlier case reports, dating from 50 A.D., St. Luke reports that when Ananias was charged by Peter, "You have not lied to man but to God," Ananias fell down dead (Acts 5:1–2).

Anecdotal evidence has accumulated on sudden death in humans and animals exposed to certain stress situations. The development of this research topic parallels much of the development of general stress studies. Initial interest was expressed by Cannon (2) and Selye (3), after which research bifurcated into psychological and physiological studies.

In 1942 Cannon wrote his now classic paper on voodoo death (2). He searched through the anthropological literature and found a number of reports of otherwise healthy people dying inexplicably after being hexed or violating some taboo. Cannon speculated that people died from suddenly flooding excessive adrenal discharge. As one example, he cites a case originally reported by Merolla in his 1692 diary of a Congo voyage.

> A young Negro on a journey lodged in a friend's house for the night. The friend had prepared for their breakfast a wild hen, a food strictly banned by a rule which must be inviolably observed. . . . The young fellow demanded whether it was indeed a wild hen and when the host answered "no," he ate of it heartily and proceeded on his way. A few years later when the two met again, the old man asked the younger if he would eat a wild hen. He answered that he had been solemnly charged by a wizard not to eat that food. Thereupon the host began to laugh and asked him why he refused it now after having eaten it at his table before. On hearing this news the Negro immediately began to tremble, so greatly was he possessed by fear, and in less than 24 hours he was dead.

Engel (4) collected a number of anecdotal case reports of people dying suddenly in the setting of acute stress.

> A 71 year old woman arrived by ambulance at the Emergency Room, accompanying her 61 year old sister who was pronounced dead on arrival. The patient collapsed at the instant of receiving the news. An EKG showed AV dissociation . . . left bundle branch block and myocardial damage. Shortly she developed ventricular fibrillation and died. (p. 774)

The explanatory models for this occurrence are still being formulated. It is my premise that a variety of factors combine to result in emotional sudden death and that a rigid division between psychological factors and physiological factors is inappropriate. However, I will present, as separately as possible, the psychological and physiological aspects of sudden death.

PSYCHOLOGICAL THEORIES

Overwhelming Stress

Engel (4) collected histories of 170 emotional sudden deaths. Death usually occurred around losses, situations of personal danger, and occasionally some paradoxical situations of great relief, pleasure, or triumph. All these situations had certain stress-related factors in common: 1) being impossible to ignore; 2) evoking intense emotions; and 3) involving a situation in which the person had no control.

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Hopelessness

Other studies concentrate on the hopelessness of the individual in the situation. Richter (5) described sudden death in rats that was apparently caused by hopelessness. He devised an experimental drowning apparatus in which normal rats could survive for many hours; however, there were always some rats who died within moments. In his next phase of research, he immobilized each rat in a bag while its whiskers and facial hair were shaved off; the rats were then dumped into the tank. Richter reports, "The first rat swam around excitedly on the surface for a very short time, then dove to the bottom where it began to swim around nosing its way along the glass wall. Without coming to the surface a single time, it died two minutes after entering the tank" (p. 194). Richter's initial conclusion was that trimming off the whiskers probably destroyed the rat's most important contact with the outside world. However, further studies revealed that restraint itself was sufficient to cause sudden death; some wild rats died while being held immobilized in the hand. He concluded that the situation of restraint, of being hopelessly trapped, led to a severe fear and rage reaction. The hopelessness of the situation caused the rats to give up. Interestingly, there was no adrenergic effect on heart rate, which slowed rather than accelerated. Richter speculated that the rats died a vagal death, and pretreatment with atropine indeed spared 3 rats of a group of 25 from prompt death.

A number of studies emphasize the hopelessness and helplessness that precedes sudden death. Schmale (6), Saul (7), and Engel (8) refer to this as "giving up-given up response." Bruhn and associates (9) described the "Sisyphus pattern"—the individual strives incessantly and with no chance for satisfaction or success.

Predilection to Death

Weisman and Hackett (10) described a predilection to death among surgical patients. They noted a small number of patients who accurately predicted their deaths. These patients regarded their impending deaths without anxiety or depression and were convinced that death was not only inevitable but desirable. The particular cause of sudden death varied from case to case.

> At the age of 41, the patient had undergone an abdominoperineal resection for cancer of the rectum, which kept her an invalid for the next 28 years. . . . The patient's misfortunes multiplied as the years of invalidism went on. Her favorite brother died from a painful rectal carcinoma leaving a large family destitute. . . . In rapid succession three of her sisters died. . . . Finally her loyal husband died . . . [When seen by the psychiatrist she had a striking] Leaden expression of anguish . . . which in its solemn immobility resembled a death mask. . . . She easily stated her conviction that death would occur in a few days and went on to say she had wanted to die for many years. . . . She told of the deaths in her family, of watching through the long dreary years the death of nearly everyone around her. . . . She thought of herself as a plump and sickening slug swallowing and feeding on death. . . . In speaking of her own death, she did not merely intimate that the long sought end was near, but avowed that she would be killed by surgery. After a previous operation she had a pulmonary hemorrhage. . . . Now she was certain another lung hemorrhage would recur except that this time she would certainly die. Eight days after surgery she unexpectedly succumbed to a massive pulmonary hemorrhage. (10, pp. 235–236)

Life Changes

Rahe and associates (11), who have explored the effects of life changes in illness, developed a scale of 42 items (schedule of recent events or SRE) and have investigated the correlation of medical illness with an increased number of these life events. Whereas the previously mentioned studies have looked at the subjective meaning and impact of events, the SRE focuses on the sheer accumulation of events that may be significant or novel to the individual. Rahe and Romo (12) looked at the life event scores of individuals dying suddenly from myocardial infarction and studied the histories of 61 individuals who died suddenly of coronary artery disease and who had previously been in good health. Spouses or next of kin were interviewed concerning the deceased's recent events. In the two years prior to sudden death they found a 143% increase in life events ($p<.005$, figure 1).

All of these psychological theories have assumed that stress is a sine qua non of sudden death—stress of flooding intense emotion that is impossible to ignore, stress of hopelessness and giving up, stress of predilection and longing for death, and stress of accumulating life events. In this sense sudden death is regarded as just another manifestation of the organism's response to stress. What is puzzling is that large numbers of people are under great stress, yet few die suddenly.

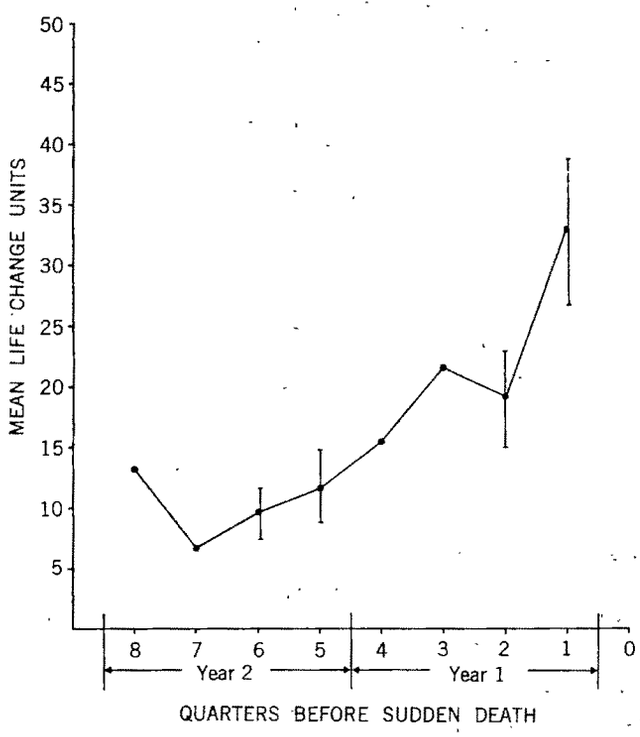
PHYSIOLOGICAL THEORIES OF SUDDEN DEATH

The physiology of response to stress is complex. I will focus on stress and the autonomic nervous system. It is clear that the so-called autonomic nervous system is not without higher cortical influence. Since most sudden deaths appear to result from cardiac events, I will review a number of articles that discuss the relationship of emotional stress to autonomic cardiovascular response.

Hypertension

The relationship of stress to hypertension is widely recognized. Among many articles, Graham's (13) is perhaps the most graphic. In 1943 he measured the blood pressure of soldiers resting on the beaches after 2 years of continuous desert warfare in Libya—26% had diastolic blood pressures greater than 100, and most of these returned to normal after a few weeks of rest.

FIGURE 1
Helsinki Study of Life Changes Units and Sudden Death



Cardiac Rhythm

There has been considerable documentation of the relationship of stress to heart rhythm. Gantt (14) has completed a number of studies on the effect of the mere presence of a person on the heart rate of dogs. He describes a famous neurotic dog named V3 who for his 8 years of life manifested a striking response to the approach of a person. His heart rate would drop from 200 beats per minute to 40 beats per minute. On occasion, V3 responded with asystoles up to 8 seconds. Even nonmisanthropic dogs have a marked physiological response to the presence of a person. The routine effect of petting dogs behind the ears is an initial increase in the heart rate and then a drop to 30–40 beats below the baseline rate.

The presence of a person also has a noticeable cardiac effect on other persons. Lynch and associates (15) described the effect of human contact on cardiac arrhythmia in coronary care patients. They noted heart rate changes of over 30 beats per minute, doubling of frequency of abnormal heart beats, and major changes in cardiac conductivity during routine interactions such as pulse taking.

There are a number of studies of heart rate in response to stress. Cardiac arrhythmia can occur in persons without cardiac disease when they are exposed to routine stress or to more extreme stress. Taggert and associates (16) continuously monitored the ECG of normal individuals driving in routine traffic and found ST depression, ectopic beats, and occasional premature ventricular contractions (PVCs). It can be inferred that the stress does not have to be unusually great to elicit cardiac abnormalities. Taggert and associates (17) also looked at the response of healthy subjects to the stress of public speaking. Six out of 23 healthy individuals developed ectopic beats of over 6 per minute while speaking. They found that these abnormalities could be suppressed by giving a beta-adrenergic blocking agent. Moss and Wyner (18) examined medical house officers presenting at grand rounds and found frequent tachycardia up to 180 beats per minute in the minute before giving their talk and the first minute of presentation. Lapiccirella (19) noted transient ST depression on the ECGs of healthy individuals before surgery and on initial imprisonment. Friedman (20) noted similar changes in bank accountants before audits.

Cardiac responses to stress situations may be stable over an extended time. Newton and Gantt (21) demonstrated a persistence in dog heart-rate response to a single-trial conditioning situation by ringing a bell and then administering a single strong electric shock to the dog. The stress of the shock led to a pronounced change in heart rate. When the stimulus of the bell ringing without the electric shock was repeated, the same heart rate changes were observed in some dogs for over a month.

Sigler (22) demonstrated that recall of emotionally upsetting situations was sufficient to produce transient ECG changes in patients with heart disease. One of his patients was a 69-year-old concentration camp survivor. The patient's ECG changed dramatically as he began recounting his experiences in the Nazi concentration camps. Specifically, he developed "changes in the QRS complex in V2 and greater depression of the RT segment and more markedly negative T in V3, V4, and V5, indicating greater ischemia" (22, p. 60).

A number of studies directly demonstrated that heart rate and arrhythmias can be modified by learning. Weiss and associates (23) used instrumental conditioning in 8 patients with chronic PVC. All showed some degree of learning heart rate control, and 5 showed decrease in PVCs in association with their decrease in heart rate. Four patients showed persistence of the lowered PVC frequency for up to 21 months after the conditioning trial.

Lown and associates (24) studied the effects of stress on cardiac rhythm in dogs. Heart rate was paced and a transthoracic shock was applied. The threshold for the amount of shock necessary to trigger a premature ventricular contraction was established under normal conditions. Under the stress condition of total immobilization, the threshold for triggering PVCs was lowered to one-third of its control value.

Myocardial Necrosis

The increased interest in animal behavior has brought a number of reports of animals dying suddenly of unknown causes in stress situations such as captivity. Gustafson and Petreman (25) noted spontaneous myocardial necroses in captive golden-mantled squir-

rels. Schmidt (26) looked at hearts of arctic ground squirrels shot wild and those killed after 2 weeks of captivity. The captive squirrels' hearts contained numerous areas of focal necrosis. Raab and associates (27) elicited focal myocardial necrosis in 25% of a series of 12 wild rats subjected to the frightening experience of listening to a taped cat-rat fight.

Selye (3) and Keegan (28) suggest that emotional stress leads to increase in catecholamines and glucocorticoids, which can cause hypoxia and disturbed cardiac conduction. Intravenous administration of large amounts of epinephrine in man can cause myocardial infarct or focal necrosis with subpericardial hemorrhage (3).

Overstimulation of the parasympathetic nervous system can also result in cardiac damage. In monkeys, continued electrical stimulation of the right vagus resulted in myocardial necrosis. Stimulation of the lateral hypothalamic nuclei led to subendocardial hemorrhage and microinfarct (29, 30).

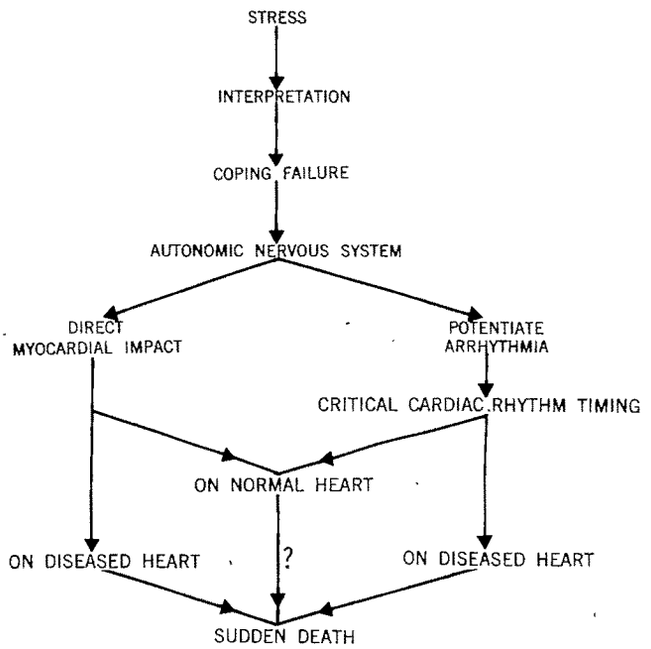
Arteriosclerosis

The presumptive cause of sudden death is arrhythmia. People with coronary artery disease are prone to develop arrhythmia. The prevalence of coronary artery disease is rather startling. Enos and Holmes (31) studied autopsies of Americans killed in the Korean War. The average age at death was 22; 77.3% of these young men showed evidence of arteriosclerosis. Hinkle and associates (32) studied a random group of 300 men aged 55 and recorded their ECG's for 6-hour periods of normal activity: 92% had asymptomatic periods of dysrhythmia and conduction disturbance, 76% had supraventricular premature contractions, 62% had PVCs, and 7% had deficits of cardiac conduction. In a 7-year follow-up study on this group they noted an increased incidence of sudden death in those men who had demonstrated PVCs or deficits of cardiac conduction.

Dive Reflex

Diving animals possess an interesting reflex that may be relevant to mechanisms of sudden death (33). They have a characteristic response to immersion of their face in water—bradycardia, decreased blood flow to skin and viscera, fall in blood pH, and rise in arterial pressure, lactic acid, blood CO_2, and potassium. This response is functional as an oxygen-conserving reflex. The diving reflex is rather inconsistent in man and can be lessened by higher cortical control. It is assumed that the bradycardia in the dive reflex is vagally mediated (34). Many deaths from myocardial infarction appear to be vagal deaths, i.e., the heart slowing to the point of asystole or ventricular fibrillation. Some researchers think that metabolic products from ischemic myocardium may activate the dive reflex as a misguided attempt to conserve oxygen (33). Hinkle and associates' study of 300 middle-aged men noted that 34 men showed a sustained relative bradycardia in daily life activity. After 7 years of follow-up

FIGURE 2
Conceptual Setting for Emotional Causes of Sudden Death



23% of the men with sustained relative bradycardia had died suddenly, whereas the incidence of sudden death in the total population sample was only 8% (35).

There may be subtle interaction between the vagal dive reflex and the adrenal response. Hartley and associates (36) suggest that

> Increased levels of catecholamines may predispose an individual to ventricular arrhythmias. The normal heart at exercise is under catecholamine stimulation. The high heart rate that occurs with exercise protects against ischemia. However, the rapid decline in heart rate after exercise exposes the myocardium to the effect of circulating catecholamines which disappear slowly from the blood.

This model suggests that sudden death occurs in the setting of simultaneous vagal and adrenergic excess stimulation.

DISCUSSION

Neither the psychological nor the physiological explanations of the causes of sudden death are firmly established. That there is a connection seems indubitable. The central question is why so few people die of an emotional sudden death. We must assume that sudden death occurs only when a number of factors are present simultaneously and that, fortunately, the odds of this occurring must be small. Figure 2, a flowchart of possible emotional causes of sudden death, illustrates that stress on a diseased heart at a certain stage in the cardiac cycle may trigger fatal arrhythmias; one must remain agnostic about the possi-

bility of stress-induced fatal arrhythmia in a completely healthy cardiovascular system.

Research Implications

This review has considered sudden death as a unitary phenomenon; thus I have discussed voodoo death, predilection to death, and sudden death. It is quite possible that these deaths do not result from the same mechanism. Future studies must be explicit in defining what constitutes sudden death. There is also confusion about antecedent causes of sudden death; the effects of sudden acute stress and prolonged accumulating stress may very well be different. It is not sufficient to state that the individual was "under stress" without specifying type and duration.

The most striking finding concerning research strategy is the absence of collaboration between cardiologists and psychiatrists. Cardiology journals are replete with studies describing the incidence and possible cardiovascular mechanisms of sudden death. Any attention given to emotional state is usually reserved for a brief diffuse sentence in the conclusion. The psychiatric journals contain case reports and an occasional attempt to summarize common emotional factors in sudden death, with little regard for the patient's prior cardiovascular status. Collaboration on psychosomatics of coronary disease is well underway with reference to personality types prone to myocardial infarction (37) and factors in rehabilitation of these patients (38). Unfortunately, this model of collaboration does not extend to consideration of sudden death per se.

This lack of collaboration results partly from the difficulties involved in studying sudden death. Sudden death is rare enough to make a prospective study in an unselected population unwieldy for both cardiologist and psychiatrist. However, collaboration should be possible in a selected population. Several approaches are possible: 1) patients in coronary care units, clearly at risk for fatal arrhythmias, could receive careful psychiatric evaluation; 2) activity of the peripheral sympathetic nervous system could be monitored by urinary assay; and 3) the methodology of Lynch and associates (15) could be adopted, i.e., a diary kept of daily occurrences matched with continuous rhythm strips. The last possibility would enable the clinician to study the occurrence of fatal arrhythmia with reference to preceding events—emotional, cardiac rhythm, and adrenergic discharge—and then interview the resuscitated patient. Until this sort of methodology is applied, we are likely to persist in work that is interesting but not sufficiently understood.

Clinical Implications

Because psychiatrists do not understand the exact composition of lethal emotional stress, we must routinely intervene in cases of patients at risk. Nowhere is this more pressing than in the coronary care unit, where emotional arousal, particularly anxiety, is not only distressing but potentially lethal and deserves treatment just as hypokalemia does. The treatment alternatives are eminently accessible, ranging from the supportive presence of the physician and antianxiety agents to more specialized techniques of psychotherapy and behavioral therapy.

COMMENT

This paper is not an exhaustive review of the emotional causes of sudden death. Instead it reviews concepts and key studies pertinent to an understanding of the phenomenon. The goal of this review is to stimulate interest in this very important and surprisingly neglected area. In both research and clinical practice, collaboration between psychiatry and medicine is sorely needed in the area of emotional causes of sudden death.

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A Survey of Tardive Dyskinesia in Psychiatric Outpatients

BY GREGORY M. ASNIS, M.D., MICHAEL A. LEOPOLD, M.D., ROGER C. DUVOISIN, M.D., AND ARTHUR H. SCHWARTZ, M.D.

The authors found a high prevalence—43.4%—of tardive dyskinesia in a sample of psychiatric outpatients, a population previously thought to be at nominal risk for development of this syndrome. There was no significant relationship between the presence of dyskinesia and age, sex, years of neuroleptic use, or various organic factors. The effects of dentures and of drug combinations are discussed, and it is noted that structured scales of dyskinesia and videotape recordings are important tools in diagnosing and following the course of dyskinesia.

TARDIVE DYSKINESIA, a neuroleptic-induced neurological syndrome, is characterized by involuntary movements of the tongue, face, jaws, and lips; occasionally, there are choreoathetoid movements of the extremities or trunk (1). Since the syndrome was first reported in 1956, various surveys have found its prevalence to be as high as 56%. These surveys focused exclusively on psychiatric inpatients, particularly chronic patients in geriatric, state, and veterans institutions (2, 3). The surveys have not included psychiatric outpatients, who are often younger, do not suffer the effects of chronic institutionalization, and have had shorter durations of treatment with antipsychotics.

Since tardive dyskinesia is often irreversible, it is essential that the treating physician be aware of its prevalence and of the factors that predispose to or in any way influence its occurrence. At present, the outpatient clinician does not have the necessary information to weigh this risk in treatment with antipsychotic medication. In recent years there have been reports of tardive dyskinesia in a few cases of younger patients who had been on neuroleptic medication for only brief periods of time (1). In this study, we surveyed a psychiatric outpatient population for tardive dyskinesia.

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METHOD

Sixty-nine patients were randomly selected from the group of 250 patients receiving antipsychotic medication in the psychiatric outpatient clinic of Mount Sinai Hospital at the time of the study. Patients were included in the study, regardless of diagnosis, if they had been taking antipsychotic medication for at least 3 months and if they were not taking medication at the time of evaluation, but their last dose was within the 3 preceding months.

After giving informed consent, all patients were interviewed and examined by one of the investigators (G.M.A. or M.A.L.). The presence of dyskinetic movements and their location and severity were rated on the Abnormal Involuntary Movement Scale (AIMS), a 5-point scale that has been found to assess tardive dyskinesia reliably (4, 5). Videotapes were made of all patients who appeared to have involuntary movements and of 8 patients who seemed to have no such involuntary movements and who served as controls for the final evaluations. Three members of the research team (G.M.A., M.A.L., and R.C.D.), including an experienced neurologist who served as a consultant in developing the AIMS (R.C.D.), then made independent ratings of all videotapes by using the AIMS. For the purpose of the survey, patients were considered positive for dyskinesia only if all three raters concurred.

All patients were also evaluated for organicity by means of the Mental Status Questionnaire (MSQ) and the Face-Hand Test (FH) (6). The MSQ consists of 10 orientation and information questions and was rated positive if more than 2 incorrect responses were made. The FH test assesses the patient's ability to perceive double simultaneous stimulation; errors have been reliably associated with brain damage in adults. Patients were rated positive if they made 1 error or more.

Other factors evaluated for possible association with involuntary movements were the duration and type of medication, number and length of hospitalizations, history of amphetamine or alcohol abuse, history of

ECT, personal or family history of neurological disorders, and history of drug-induced extrapyramidal side effects.

RESULTS

Of 69 patients sampled (53 women and 16 men), the three raters concurred that 30 patients (25 women and 5 men) were positive for dyskinesia on the AIMS. This represents 43.4% of our randomly selected outpatient sample. Although there was total agreement among raters on the presence or absence of dyskinesia, some variability existed in the severity of global ratings in 11 dyskinetic patients, with a grade of 1 being the largest difference between any two raters. Of the dyskinetic group, 60% were rated as minimally impaired, 30% as mildly impaired, and 10% as moderately impaired.

Demographic Characteristics

As can be seen in table 1, tardive dyskinesia was present in 47.2% of the women and 31.2% of the men surveyed, a difference that did not reach statistical significance (p=.40).

The mean age of the survey population was 45.5 years; the dyskinetic group had a mean age of 45.8 years and the nondyskinetic group of 45.3 years (p=n.s.). The percentage of patients diagnosed as dyskinetic was the same for those 50 years old or younger and those 51 or older (43.4%).

The mean age of the men with dyskinesia (37.8 years) was similar to that of those without dyskinesia (39 years). Mean ages for the two groups of women were also similar (47.4 years for the dyskinetic group and 47.8 for the nondyskinetic group). The sample is broken down by age and diagnosis of dyskinesia in table 1. The youngest patients with dyskinesia were 3 women aged 20, 22, and 28 years, and 1 man aged 20.

Medication and Organic Factors

The mean number of years on neuroleptics was slightly less in the dyskinetic group than in the nondyskinetic group (4.59 years versus 5.50 years, p=n.s.). It can be noted in table 2 that 46.7% of the patients with dyskinesia had been on neuroleptics for 2 years or less. Four patients (13.3%) with dyskinesia had received neuroleptics for 1 year or less, with the shortest duration being 8 months. All of the men with dyskinesia had been on neuroleptics for 5 years or less, the majority (4 of 5) for 2 years or less. Of the 30 dyskinetic patients, 2 had terminated neuroleptics 1 week prior to the evaluation. Both patients were immediately restarted on their medications, and 2 months later they were again found to be dyskinetic.

Despite careful assessment of hospital charts and interviews with patients, we were unable to assess with confidence all of the past neuroleptics and dosages patients had been given since their treatment began. Therefore, we could not assess which types of medications and dosage levels may be more often associated with tardive dyskinesia. The butyrophenones, phenothiazines, and thioxanthenes were all noted in the medication histories of patients with dyskinesia in this survey.

Neurological histories and history of ECT, alcoholism, amphetamine abuse, and cerebral dysfunction as reflected by the MSQ or FH test were approximately the same in the two groups (see table 3). Chart review revealed no active medical or neurological problems, no endocrinopathies, and no abnormal blood chemistries. Family history of movement disorders was found in 1 dyskinetic patient (Parkinson's disease) and

TABLE 1
Frequency Distribution By Age

	Dyskinetic Group (N=30)				Nondyskinetic Group (N=39)			
			Total				Total	
Age (years)	Women	Men	N	%	Women	Men	N	%
18–40	8	3	11	36.7	10	6	16	41.0
41–50	8	1	9	30.0	6	4	10	25.7
51–60	5	1	6	20.0	4	1	5	12.8
61–70	1	0	1	3.3	5	0	5	12.8
71–80	3	0	3	10.0	2	0	2	5.1
>80	0	0	0	0.0	1	0	1	2.6
Total	25	5	30	100.0	28	11	39	100.0

TABLE 2
Duration of Neuroleptic Medication

	Dyskinetic Group (N=30)				Nondyskinetic Group (N=39)			
			Total				Total	
Duration (years)	Women	Men	N	%	Women	Men	N	%
≤1	4	0	4	13.3	8	1	9	23.1
1–2	6	4	10	33.3	1	3	4	10.3
2–5	6	1	7	23.4	9	4	13	33.3
5–10	5	0	5	16.7	5	1	6	15.4
10–21	4	0	4	13.3	5	2	7	17.9
Total	25	5	30	100.0	28	11	39	100.0

TABLE 3
Organic Factors and Tardive Dyskinesia

	Dyskinetic Group (N=30)		Non-dyskinetic Group (N=39)	
Factors	N	%	N	%
Previous ECT	5	16.7	7	17.9
Alcoholism	6	20.0	7	17.9
Personal neurological history	2	6.7	2	5.1
Family neurological history	1	3.3	2	5.1
Amphetamine abuse	2	6.7	2	5.1
Positive MSQ or FH test	2	6.7	6	15.4

2 nondyskinetic patients (Parkinson's disease and familial tremor). No evidence of Huntington's chorea was present in patients or family members.

By chart review and interviews with patients, we verified past drug-induced dystonia in only 3 dyskinetic patients (10%) and 1 nondyskinetic patient (2.6%).

Miscellaneous Factors

The degree of psychopathology was assessed indirectly by the total number of psychiatric hospitalizations. The two groups had approximately the same mean number of hospitalizations (1.6 and 1.5). Medians were 2 and 1 for the dyskinetic and nondyskinetic groups, respectively. Statistical analysis indicates a trend toward more than 1 hospitalization among the dyskinetic group ($\chi^2=3.76$, $p<3.06$). Because patients' memories and charts were often vague about the duration of each hospitalization, exact times were not calculated, but all were for less than 1 year.

One factor that was frequently associated with dyskinesia was the presence of dentures—50% of patients with dyskinesia (N=15) had dentures compared with 18% (N=7) in the nondyskinetic group ($\chi^2=6.61$, $p=.01$). Of the 15 dyskinetic patients with dentures, 6 had vermicular movements of the tongue and 2 had choreoathetoid movements of the extremities. Only 6 of the dyskinetic patients with dentures had abnormal movements that were limited to their jaws.

An interesting finding was that in the total group of 69 patients surveyed, 9 patients were on neuroleptics alone and 5 of these (55.6%) had tardive dyskinesia. Of 22 patients on neuroleptics in combination with antiparkinsonians alone, 6 patients (27.3%) had tardive dyskinesia. Although a trend was noted, it was not statistically significant ($\chi^2=1.17$, $p=.28$).

DISCUSSION

Past surveys have reported the prevalence of tardive dyskinesia to range from 0.5% to 56%, with most recent studies citing the highest percentages (2, 3). These studies, which focused on chronically institutionalized populations, have given rise to the clinical impression that patients particularly at risk to develop dyskinesia 1) are female, 2) have organicity or prior brain damage (factors 1 and 2 are controversial), 3) are older (over 55 years old), and 4) have used neuroleptic medication for more than 2 years (1, 7, 8).

Before our outpatient survey of tardive dyskinesia, it was commonly felt that dyskinesia was infrequent in psychiatric outpatients being treated with neuroleptics and was mainly a problem of chronically institutionalized patients (1). The most striking result of our survey was the high prevalence (43.4%) of tardive dyskinesia in a population previously described as being at nominal risk for tardive dyskinesia. The dyskinetic group was young (average age of 45.5 years), had a short duration of neuroleptic medication (less than 2 years for 46.7%), and were not chronically hospitalized (median of 2 hospitalizations). There was no significant relationship between the presence of dyskinesia and age, sex, years of neuroleptic use, organicity, history of ECT, amphetamine or alcohol abuse, or of neurological disorders in the patients or their families. Perhaps the predisposing factors reported in previous studies were characteristic of their sample populations—chronically institutionalized patients.

Two associated findings of questionable significance were those related to dentures and antiparkinsonian medication. Dentures were worn by 50% of the dyskinetic group but only 18% of the nondyskinetic group, but only 6 dyskinetic patients who wore dentures had movements limited to the jaw, and none complained of discomfort. Since there were no denture problems and most of these patients had movements of the tongue and/or extremities, we believe that the movements were not induced by dentures.

Recently there have been reports that antiparkinsonians lower the threshold for amine stereotyped behavior in animals and when used in conjunction with neuroleptics may increase the risk of tardive dyskinesia in man (9). Our results suggested a contrary trend: 27.3% of patients on neuroleptics and antiparkinsonians had dyskinesia, whereas 55.6% of those on neuroleptic medication alone were dyskinetic. However, since our survey was not designed to evaluate the effects of antiparkinsonians specifically, it is difficult to draw conclusions from these data.

It appears that there is little to guide the clinician in predicting which outpatients may develop dyskinesia. We would agree with the present literature that the best way to reduce the occurrence is to give neuroleptic medication only to schizophrenic patients, using non-neuroleptics to treat other states, such as anxiety and depression. Since dyskinesia is often irreversible, it is incumbent on the physician to detect the syndrome early in its course so medication can be discontinued if the mental condition permits. Frequent examinations of the patient, assessing early oral-facial movements, particularly vermicular movements of the tongue, and temporary withdrawal of antipsychotic medication to evaluate an early dyskinesia that may have been masked have been suggested (10). We believe that early detection of dyskinesia is facilitated by using a structured dyskinesia scale like the AIMS and scales developed by Simpson and associates (11) and Crane and Naranjo (12), which can bring out subtle dyskinetic movements. This should be done before initiating treatment and at fixed intervals thereafter.

In the present study, the videotape recording of the dyskinesia examinations was invaluable in increasing diagnostic sensitivity, particularly of early signs of tardive dyskinesia. The videotapes included not only the examination itself but also a 1–2-minute interval of the patient sitting alone in the room. One patient did not show dyskinetic movements during the structured evaluation, but clearly had such movements during the period when he sat alone.

The videotape also allowed several observers to rate

the same examination at a later date. This is important since movement disorders can vary from one examination to the next in quantity and quality. The recordings can also be used to follow the development of these movements and to determine how they first appear or change over time. Most studies of tardive dyskinesia have not used videotape or movie recordings of the dyskinesia evaluations. Such recordings seem to be essential to future research on the clinical manifestations and progression of tardive dyskinesia.

CONCLUSIONS

This study demonstrates that outpatients as well as inpatients receiving neuroleptics are at a substantial risk for tardive dyskinesia. When neuroleptics are essential to the treatment of the patient, early detection of involuntary movements becomes critical. The present study suggests that the sensitivity of diagnosing tardive dyskinesia can be increased by examining the patient with a structured dyskinesia rating scale and videotaping the examination for evaluation by other physicians. Early, subtle signs can be detected more easily by following the patient's progress with subsequent videotaped evaluations. Further study is necessary to better understand the effects of medication combinations and other factors on the occurrence and development of tardive dyskinesia.

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An Evaluation of a Unique New Antipsychotic Agent, Sulpiride: Effects on Serum Prolactin and Growth Hormone Levels

BY DAVID H. MIELKE, M.D., DONALD M. GALLANT, M.D., AND CRAIG KESSLER, M.D.

Sulpiride is a new and chemically different neuroleptic. In a study of 16 severely ill schizophrenic patients, this compound displayed definite evidence of antipsychotic activity while producing few adverse reactions. The relative absence of extrapyramidal side effects may indicate that sulpiride has a low potential for producing tardive dyskinesia. Two patients who did not develop significant increases in prolactin levels did show a definite therapeutic response to sulpiride. Thus it appears that central dopaminergic blockade in the hypothalamic area is not a prerequisite for antipsychotic activity.

SULPIRIDE (±) is a new drug chemically unrelated to known neuroleptic agents. It is a member of a class known as orthopramides or *o*-anisamides and has the chemical name of N-[(1-ethyl-2-pyrrolidinyl)-methyl]-2-methoxy-5-sulfamoylbenzamide (1). The structural formula is shown in figure 1.

In animal studies sulpiride has displayed strong antidopamine effects (1), but its influence on norepinephrine is unknown (a review of the laboratory profile is available elsewhere) (2, 3). Acute and chronic administration of sulpiride to animals has resulted in toxicity and catatonia only at very high doses. Lower species excrete greater numbers of metabolites than do higher species.

Studies of human volunteers have shown that sulpiride is rapidly absorbed with oral and parenteral administration and that its half-life is relatively short (only about 8 hours) (1). Virtually complete elimination of a single dose occurs at 24 hours. Using ^{14}C labeled sulpiride administered orally, researchers found that approximately 40% of the drug was absorbed, and it was excreted renally in an unchanged form (1). To our knowledge, no metabolites have been found in man.

The clinical application of sulpiride began in the field of gastroenterology in 1966, and it currently is used as an anti-ulcer medication in other countries. Subsequently, sulpiride was used in the treatment of various psychopathological disorders, including psychotic states (3–7) and neurotic depression (3, 4, 8). The vast majority of studies suggest a broad therapeutic spectrum of activity and a low incidence of side effects. Jouglard and associates (9) found that toxicity is minimal, as evidenced by lack of mortality after single-dose ingestions of up to 7 g. These authors reported that some patients exhibited extrapyramidal phenomena for up to 48 hours.

In a previous evaluation of sulpiride in 10 schizophrenic patients, the Tulane research team found it to be an effective agent, free of side effects at moderate doses (2). Further trials were indicated to study the efficacy and adverse reactions at higher doses and to study the effects on prolactin and growth hormone over time.

METHOD

The study was conducted using 16 chronically hospitalized, severely ill schizophrenic patients under close supervision on a special research unit. The 6 men and 10 women were free of any significant disabilities of the cardiovascular, endocrine, hematopoietic, renal, and hepatic systems. None of the subjects had a history of alcoholism, cerebral trauma, leukotomy, epilepsy, recent ECT, or neurological disease. Informed consent was obtained from the patient and the closest living relative after the nature of the procedure had been fully explained.

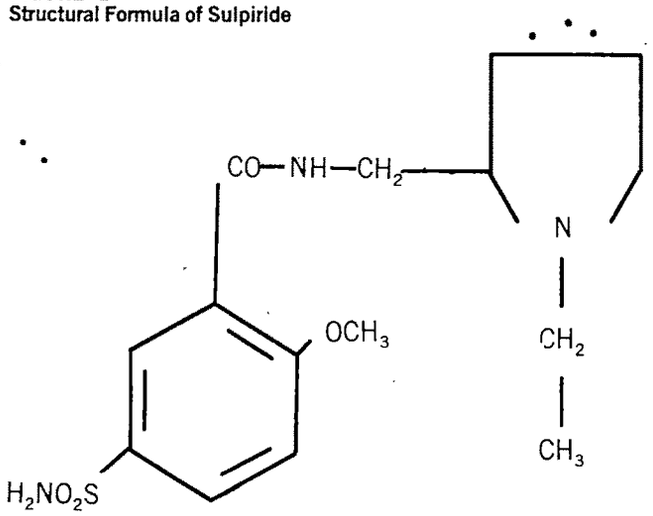
The average age was 38.6 years, and the duration of hospitalization averaged 15.5 years. After a 14-day drug-free period, sulpiride was administered twice daily for 12 weeks using a flexible schedule of increases dependent on therapeutic response and the appearance of adverse reactions. (Sulpiride was supplied by Warren-Teed Pharmaceuticals, Horsham, Pa.) The starting dose was 400 mg daily, with a planned maximum of 2600 mg per day. A small comparison group of 6 patients was administered standard neuroleptics in optimal dosages using the same schedule as that used with the sulpiride subjects. Prolactin and growth hormone blood levels were determined by radioimmunoassay procedures.

Clinical tests were performed as follows: supine and

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FIGURE 1
Structural Formula of Sulpiride



standing blood pressures three times weekly; ECG at baseline and weekly; and photosensitivity skin tests and complete eye examination, including slit-lamp examination, at baseline and termination of the study. Complete blood count, urinalysis, and SMA-12 were conducted at baseline and subsequently every other week. Serum prolactin and growth hormone levels were ascertained at 11 a.m. at baseline, on days 1, 10, 17, 24, 52, and 80 (4 hours after the morning dose); and 3 days after termination of active drug treatment. Psychometric measures included weekly global ratings (−1 to +3) based on a brief mental status examination. In addition, the Brief Psychiatric Rating Scale (BPRS), Clinical Global Impressions (CGI), and the Nurses' Observation Scale for Inpatient Evaluation (NOSIE) were rated at baseline, weeks 4 and 8, and termination. Adverse reactions were evaluated on a daily basis.

All data were analyzed by the George Washington University Biometric Laboratory, ECDEU section, using analysis of variance—repeated measures model.

RESULTS

Consistent with our first evaluation of sulpiride, the overall findings showed that this agent exerts a definite neuroleptic effect at doses above 400 mg daily. In the severely ill schizophrenic patients, some beneficial changes first appeared at doses above 2000 mg daily. Maximum daily doses administered were 1600 mg (4 subjects), 2400 mg (2 subjects), and 2600 mg (10 subjects).

The CGI, BPRS, and NOSIE strongly supported the clinical findings of improvement. Significant change scores ($p<.05$) across time were noted on all scales and each factor for which improvement was possible (core schizophrenic symptoms).

The study also indicated that sulpiride elevated serum prolactin levels and depressed growth hormone levels over time. It should be noted that the age difference between the sulpiride patients (see table 1) and the comparison group (see table 2) was significantly different ($p<.001$). Low doses of the neuroleptic rapidly elevated serum prolactin levels, but increasing the doses generally did not produce higher hormone concentrations. Serum prolactin values rapidly diminished after drug withdrawal.

In our first evaluation of sulpiride (2), serum prolactin levels were determined by a different laboratory using the same radioimmunoassay technique and were generally lower than those levels found in table 1. No correlation was found between increased prolactin levels and individual improvement in global ratings. Two subjects improved, but their serum prolactin levels did not show a significant increase above baseline levels.

ADVERSE REACTIONS

During this 12-week study only 1 of the 16 sulpiride patients (patient 2) exhibited side effects (a dystonic reaction on 2 separate occasions) when receiving doses of 1800 mg or greater. On each occasion he responded to a single dose of anticholinergic medication. No signs of sedation or antiadrenergic effects were observed in this group of patients. Laboratory values were not significantly abnormal, although the white blood cell count increased moderately in 8 patients without a shift in differential count.

For 7 subjects who were receiving doses exceeding 2200 mg per day, the ECG showed no changes from baseline configurations. For the remaining 9 patients, the only ECG change was a broadening and flattening of T-waves at various dosages over 1600 mg per day. As observed in our original sulpiride study, the only ECG alterations produced by sulpiride were those benign, nonspecific changes usually occurring at levels above 2200 mg daily.

Slit-lamp evaluation did not reveal any ophthalmological changes during treatment with sulpiride. There was no evidence of photosensitivity. No other significant clinical or laboratory abnormalities were noted.

DISCUSSION AND SUMMARY

The regulation of peptide hormones has been the subject of much investigation. Based on direct and indirect evidence, the modulation of prolactin is mediated by a prolactin membrane receptor (10), by the obligatory role of monoamines (11–14) in the hypothalamus and the pituitary (15, 16), and by the presence of a prolactin-inhibiting factor that has not yet been isolated (17). This prolactin-inhibiting factor may be dopamine itself, since high concentrations in the hypothalamus will inhibit the release of prolactin (15–17). There is evidence that both norepinephrine and dopamine modulate growth hormone release (14, 18).

Several commonly used drugs alter serum prolactin and growth hormone levels. Centrally acting com-

TABLE 1
Serum Growth Hormone (GH) and Prolactin Levels Before, During, and After Sulpiride Treatment of 16 Schizophrenic Patients

Patient	Age	Sex	Years Hospitalized	Factor Measured	Baseline	Day 1	Day 10	Day 17	Day 24	Day 52	Day 80	Post-drug
1	42	M	23	GH*	20.0	<0.2	<0.2	1.3	0.3	<0.2	0.2	7.7
				Prolactin*	3.2	54.0	50.00	88.0	112.4	65.0	4.7	—
				Dose/day**	0	400	1000	200	1600	1200	1600	0
2	36	M	14	GH	3.5	<0.2	0.2	<0.2	0.3	<0.2	<0.2	0.5
				Prolactin	3.3	20.0	43.0	56.0	34.7	51.4	43.0	1.6
				Dose/day	0	400	1000	1200	1600	2000	2400	0
3	34	M	7	GH	< 0.2	<0.2	0.2	0.3	<0.2	<0.2	<0.2	0.2
				Prolactin	3.6	6.2	5.0	4.9	2.3	7.2	7.0	5.7
				Dose/day	0	400	1000	1200	1400	2000	2400	0
4	37	M	3	GH	<0.2	0.2	<0.2	<0.2	0.3	<0.2	<0.2	1.9
				Prolactin	10.4	35.0	42.0	34.0	66.9	93.7	83.0	186.8
				Dose/day	0	400	1000	1200	1400	2200	1600	0
5	36	M	3	GH	0.5	<0.2	0.3	0.7	<0.2	0.2	<0.2	1.2
				Prolactin	7.1	31.0	50.0	68.0	60.3	124.2	52.0	10.3
				Dose/day	0	400	1000	1200	1400	2000	1600	0
6	33	M	3	GH	0.5	0.3	0.4	<0.2	0.4	<0.2	<0.2	0.2
				Prolactin	2.6	38.0	46.0	67.0	55.8	100.9	64.0	8.6
				Dose/day	0	400	1000	1200	1600	2200	2600	0
7	41	F	7	GH	5.9	<0.2	0.2	0.2	1.9	<0.2	0.5	<0.2
				Prolactin	30.0	124.0	108.0	255.0	62.3	158.6	213.0	124.4
				Dose/day	0	400	1000	1200	1400	2200	1600	0
8	33	F	17	GH	0.8	0.3	0.2	<0.2	0.2	<0.2	<0.2	<0.2
				Prolactin	10.5	78.0	90.0	105.00	64.7	101.4	86.0	4.5
				Dose/day	0	400	1000	1200	1600	2200	1600	0
9	40	F	12	GH	0.6	0.3	11.9	0.2	0.3	0.3	1.5	8.5
				Prolactin	28.0	132.0	112.0	219.0	253.6	188.8	133.0	228.0
				Dose/day	0	400	1000	1200	1600	2200	2600	0
10	49	F	29	GH	<0.2	0.3	4.3	<0.2	0.3	0.4	1.2	0.2
				Prolactin	3.6	38.0	76.0	108.0	63.6	89.5	86.0	36.8
				Dose/day	0	400	1000	1200	1600	2200	1200	0
11	51	F	21	GH	6.8	0.2	0.2	0.8	0.3	0.3	0.5	<0.2
				Prolactin	4.0	34.0	116.0	134.0	64.0	121.9	121.0	74.8
				Dose/day	0	400	1000	1200	1600	2200	1600	0
12	46	F	26	GH	0.2	0.4	0.4	0.3	0.4	<0.2	<0.2	<0.2
				Prolactin	13.6	92.0	108.0	177.0	199.6	122.0	111.0	3.8
				Dose/day	0	400	1000	1200	1600	2200	1600	0
13	47	F	20	GH	1.2	0.3	0.3	0.3	0.3	<0.2	0.7	<0.2
				Prolactin	11.0	60.0	62.0	117.0	49.3	67.0	80.0	4.1
				Dose/day	0	400	1000	1200	1600	2200	800	0
14	48	F	25	GH	1.9	0.2	0.6	0.3	0.2	<0.2	0.3	<0.2
				Prolactin	11.8	70.0	140.0	103.0	135.8	117.7	151.0	123.7
				Dose/day	0	400	1000	1200	1600	2200	1200	1200
15	43	F	19	GH	<0.2	0.6	0.3	0.2	0.3	<0.2	0.4	<0.2
				Prolactin	17.6	124.0	136.0	278.0	259.8	172.0	261.0	204.0
				Dose/day	0	400	1000	1200	1600	2200	1200	0
16	46	F	19	GH	0.6	<0.2	0.5	<0.2	0.2	0.3	1.0	<0.2
				Prolactin	7.0	74.0	80.0	126.0	87.6	94.4	108.0	12.3
				Dose/day	0	400	1000	1200	1600	2200	1600	0

*GH and prolactin are measured in nanograms per milliliter. Normal values are as follows: GH, 0–10 ng/ml and prolactin, 0–30 ng/ml.
**Sulpiride is measured in milligrams.

TABLE 2
Serum Growth Hormone and Prolactin Levels in 6 Patients Treated with Other Antipsychotic Drugs

Patient	Age	Sex	Drug and Dose	Factor Measured*	Baseline	Day 1	Day 10	Day 17	Day 24	Day 52	Day 80	Post-drug
A	43	M	Thioridazine, 600 mg/day	GH	<0.2	0.6	<0.2	<0.2	1.7	0.5	0.2	0.3
				Prolactin	8.4	15.7	6.3	6.8	9.7	11.5	5.3	5.5
B	53	M	Haloperidol, 40 mg/day	GH	<0.2	0.7	0.2	<0.2	0.3	0.3	0.3	<0.2
				Prolactin	4.8	50.4	51.3	51.9	46.0	22.4	3.5	6.6
C	57	M	Chlorpromazine, 300 mg/day; imipramine, 50 mg/day	GH	<0.2	<0.2	<0.2	<0.2	0.5	0.2	0.9	<0.2
				Prolactin	4.1	12.2	6.8	9.6	8.9	5.6	3.9	4.5
D	30	M	Loxapine, 100 mg/day	GH	<0.2	0.4	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
				Prolactin	4.8	56.3	33.7	32.0	43.0	13.4	7.6	4.3
E	55	F	Trifluoperazine, 60 mg/day	GH	0.3	0.5	0.2	<0.2	0.2	0.3	0.6	0.3
				Prolactin	4.7	56.4	62.5	74.0	74.0	78.8	79.7	17.4
F	48	F	Molindone, 80 mg/day	GH	1.1	1.9	0.2	<0.2	<0.2	<0.2	<0.2	<0.2
				Prolactin	14.8	237.8	345.1	509.0	297.0	1016.0	550.0	31.4

*GH and prolactin are measured in nanograms per milliliter.

pounds known to antagonize dopaminergic neurons, to stimulate serotonergic neurons, or to release thyroid-releasing hormone can profoundly increase serum prolactin levels. Such pharmacological agents include neuroleptics of all classes, tricyclic antidepressants, reserpine, and methyldopa (19–21). Dopamine agonists (e.g., L-dopa, apomorphine, ergot alkaloids) markedly inhibit prolactin secretion (19). It has been observed that antipsychotic drugs lower blood levels of growth hormone (22). However, it is not known whether a drug exerts the same degree of blockade in all dopaminergic pathways.

The consequences of drug-induced shifts in circulating hormones are unknown. Recently, there has been a controversy (23) over the possible increased risk of breast cancer in women exposed to rauwolfia derivatives (24–26). The relationship between chemicals and hormones and the development of tumors is complex. To date, there has been a failure to show an association between carcinogenesis and the use of non-rauwolfia drugs known to enhance prolactin release (24, 25). To our knowledge, neuroleptic-induced depression of serum growth hormone has not been shown to be harmful to adults or children.

In summary, sulpiride is a potentially valuable neuroleptic because of its very low potential for side effects and a wide margin of safety between therapeutic doses and toxic levels. It has been speculated that antipsychotic agents which produce few or no extrapyramidal symptoms have a diminished capability of producing tardive dyskinesia.

Sulpiride tends to elevate prolactin and depress growth hormone values. Increased serum levels of prolactin could not be correlated with individual clinical improvement. It is interesting to note that 2 patients who did not develop significant increases in prolactin levels (a reliable indicator of dopaminergic blockade) did show a definite therapeutic response to an antipsychotic agent. Thus it appears that dopaminergic blockade in the hypothalamic tuberoinfundibular area does not necessarily correlate with therapeutic responses to neuroleptic agents.

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Denying the Incredible: Unexplained Deaths in a Veterans Administration Hospital

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During July and August 1975 a large number of patients at the Ann Arbor Veterans Administration Hospital experienced unexplained respiratory arrests, and there were six deaths. These were later shown to be due to the deliberate injection of pancuronium bromide, a muscle relaxant. A questionnaire survey of the reactions of the hospital patients and staff to the deaths revealed that the patients had more confidence in the hospital and their medical treatment than did the staff, and those patients who were theoretically most vulnerable used denial to cope with the unprecedented and frightening situation.

THE ANN ARBOR Veterans Administration (VA) Hospital is one of the most active teaching hospitals in the VA system. The full-time attending staff is integrated with the University of Michigan Medical School, and all staff members hold academic appointments. The hospital trains residents, interns, medical students, nurses, and other professionals from the university. During the months of July and August 1975, an increasing number of patients began to experience respiratory arrests. Some of the arrests were among the chronically debilitated or terminally ill, but others caused more concern because they occurred among patients who apparently were stable or experiencing otherwise uneventful recoveries. Although the patients were found apneic and unresponsive, most often in the intensive care unit (ICU), seemingly superlative work by the evening staff managed to revive the majority. However, some patients did die, including a small number whom no one expected to have respiratory complications.

The hospital's medical and surgical staff soon became filled with a sense of unease. By the end of July the chief of anesthesiology had initiated a vigorous chart review to try to determine the cause of the breathing failures. However, the rash of respiratory arrests continued. Multiple hospital emergency calls would often be heard during the day and early evening. Some patients on the medical wards and the ICU began to experience second and even third episodes of respiratory failure. The intensive review of records failed to reveal any consistent medical or pharmaceutical error. The unthinkable slowly became tenable. Might someone be deliberately causing the respiratory arrests?

From July 1 to August 15, 1975, there were 51 respiratory arrests among 35 patients, 6 of whom died under suspicious circumstances (1). Before the outbreak there had been an average of 6 such arrests a month. The situation reached a crescendo on August 15 in the ICU. From 4:30 to 4:50 in the afternoon, first 1 patient, then another, and then a third suddenly stopped breathing. Teams of doctors, nurses, and orderlies successively raced from 1 patient to another, intubating them and placing them on respirators. The chief of anesthesiology was present and noticed the flaccid nature of the patients' muscles. A nerve stimulator was brought from the operating room and applied to one of the patient's arms. It showed evidence of a curarelike muscle blockade. Neostigmine was injected intravenously and there was a dramatic reversal of the patient's muscle and respiratory paralysis. Samples were then taken of serum, bile, and urine for further testing. The unthinkable—that someone was deliberately poisoning these patients—became the most plausible explanation. The Federal Bureau of Investigation (FBI) was called into the case a few hours later.

The nature of the depolarizing blockade made Pavulon (pancuronium bromide) the drug most immediately suspect. Pancuronium is an intravenously injected muscle relaxant that, ironically, is sometimes used in the ICU as an aid in placing conscious patients on mechanical respirators. On September 3, 1975, pancuronium or its metabolites were found in the bile and urine of 2 of the patients who had undergone arrests in the ICU.

The events in the hospital had been chronicled daily in the local press and on the radio and television almost from the moment the FBI had been notified. Speculations in the media were only later followed by

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confirmations. Front-page headlines such as "Killer at Large in Veterans Hospital?" were already appearing by August 20, 1975 (2). Both patients and their families expressed concern. The hospital was immediately closed to nonemergent admissions. Steps were taken to increase security. All patients receiving intravenous fluids were placed on 2 wards and closely monitored. Nurses could handle the intravenous apparatus only in pairs. The nursing staff was increased, identification cards were issued to employees, and strict controls were placed on the storage and use of pancuronium. The FBI took over a ward on one of the floors of the hospital, and it soon became common to see both FBI agents and television reporters in the hospital corridors. After being closed to nonemergent admissions for a month, the hospital slowly began to admit a reduced number of patients.

The department of psychiatry, which operates wards on 2 upper levels of the 9-story building, was somewhat removed from the turmoil on the medical and surgical services. The initial revelations were greeted by some in the department with frank disbelief, which only dissipated with the discovery of pancuronium in the body fluids of the ICU patients. The patients on the closed psychiatric unit had quickly begun joking about "Pavulon Pete," but they seemed to feel more secure behind the locked door. They suddenly began to view it as keeping someone else out rather than confining them. It became a matter of great interest as to how the patients and staff in the entire hospital reacted to the possible presence of a killer and the sudden, frightening notoriety of the hospital.

We decided that an anonymous, self-report questionnaire might well be the most unobtrusive manner of obtaining information on the current attitudes of a large cross-section of patients and staff. Those most directly affected by the events had undergone extensive FBI interrogations and were involved in a sensitive criminal investigation; further lengthy psychiatric interviewing at that time was not considered appropriate. The uniqueness of the event almost precluded the formulation of hypotheses about what, if any, pattern of responses might be found. Whatever pattern might develop would have to emerge empirically and inductively from a gathering of information.

METHOD

We developed a questionnaire that attempted to identify patient and staff attitudes and perceptions of the large number of respiratory arrests at the hospital. It consisted of 30 items divided among the following 5 categories: demographic data, the area in which the people were hospitalized or worked, attitudes toward the hospital, opinions of the origin or cause of the respiratory arrests, and attitudes toward the news media coverage.

During the period October 3–6, 1975, questionnaires similar in form and content were given to both patients and staff, to be anonymously completed and returned. Since admissions to the hospital had been severely limited, there were then only 195 patients, compared with an average daily census of 285. There also was a high percentage of extremely ill patients in the hospital, since the healthier patients had been quickly discharged after recovery. Questionnaires were given to 112 patients who were physically and mentally capable of responding; 90 were returned for a response rate of 80%. Seventy-three percent of the patients responding had been admitted after August 15, 1975. These patients may well have represented a self-selected sample of people who were strongly committed to the VA system and tended to minimize difficulties in their care. However, they were randomly distributed among all of the patient categories that were eventually compared. There were 632 employees available for sampling; 315 completed the questionnaire for a response rate of 50%. The occupational distribution of the responding employees was representative of the major job classifications in the hospital. The sample of patients and staff was divided by various criteria in such a way that 2 or more subgroups were formed. These were then compared using chi-square analysis (3).

RESULTS

Comparison of the Patient and Staff Groups

The patients appeared to have a great deal more faith in the hospital during the period of turmoil than did the staff. Only 26% of the patients, compared with 48% of the staff, would have requested the transfer of a relative to another facility. Ninety-three percent of the patients, but only 67% of the staff, stated that they would be willing to be a patient at the hospital in the future. Although 67% of the patients were reassured by the presence of the FBI, only 49% of the staff expressed similar confidence in the agency. These differences were all significant ($p<.005$ level). Seven percent of the patients and 9% of the staff felt that the hospital's reputation had been permanently damaged.

The patients' strong expression of confidence in the hospital came as a surprise. It could not be attributed to lack of information, since media coverage both in the local newspapers and on television was extensive, dramatic, and stressed the possible presence of a killer. We initially believed that much of this faith was realistically based on the hospital's 25-year history of professional service and that many of the patients, faced with limited financial resources, could not realistically contemplate transfer to private care. We also felt this confidence was the product of a sample that was skewed by psychiatric inpatients who were emotionally and physically remote from the events on the medical and surgical services. Far from being in a near panic, most patients seemed to deal with the crisis with relative equanimity. Even at the nadir of FBI fortunes, in the midst of the Hoffa disappearance and the failure to capture Patty Hearst, the patients retained their con-

fidence in the FBI while a more cynical or realistic staff remained skeptical.

However, the suspicion that denial mechanisms were partly responsible for the patients' more sanguine view of the hospital became apparent when the results of questions pertaining to the origin of the dramatic events were obtained. Of the patients responding to the question, only a bare majority (53%) felt that the cause of the events was a deliberate act of murder. Almost an equal number of patients were ready to attribute the origins to more benign, less threatening causes such as patient illness, pharmaceutical mistakes, or medical error. The staff members differed significantly from the patients in their assessment of the events; 70% of those answering the question felt that the respiratory arrests had been deliberately caused ($p<.025$). Of those who believed there was a murderer present, 75% of the patients and 82% of the staff thought that he or she was a hospital worker and not an outsider. Although they were reluctant to be specific, both patients and staff felt the killer had to be medically knowledgeable. The hospital staff was most suspicious of the medical outsider in their own midst—the medical student; the patients were concerned with the person who directly delivered the intravenous injections—the nurse. However, only a few people voiced these suspicions, and they could not be analyzed statistically.

The patients had a greater belief in the ultimate just resolution of events. Eighty-one percent of the patients, compared with only 47% of the staff, felt that whoever was responsible for the respiratory arrests would be apprehended ($p<.005$).

While the patients were about evenly divided as to whether the intensive media coverage was beneficial, the staff was extremely critical. Almost 80% felt that the coverage was exaggerated and not in the best interests of the patients or the public ($p<.005$). The investigators felt that the adverse reactions of the staff reflected employee defensiveness over the negative publicity given the hospital by the news media. Although they were sometimes sensationalized, the facts reported by the media were essentially accurate, but both the patients and staff often used the sensational tenor of the reporting to reject the idea that such coverage served the best interests of the public.

Comparison of Patients and Medical Staff in Relation to Their Proximity to the Events

It became apparent that comparing the total patient group with the total staff group elicited only the grossest of trends. It was difficult to imagine that the psychiatric inpatient could be as concerned about the respiratory arrests and deaths as the medical patient was, or that the radiologist could have the same sense of immediacy of crisis as a nurse or intern on the medical ward. We hoped that by reanalyzing the data in relationship to the subjects' proximity to hospital areas and medical procedures associated with the respiratory arrests, a more meaningful interpretation of attitudes could be made. The lack of concern on the part of the patients noted initially might not be denial but the responses of a large number of emotionally uninvolved patients and simply a form of emotional ignorance; they just did not understand the situation.

We therefore hypothesized that those patients and staff closest to the areas of difficulty would show less faith in the hospital, be more aware of the possibility of murder, and be more approving of the disclosures of the media than those patients and staff relatively distant from the events. It was further proposed that rumor and media coverage had effectively cancelled out any discrepancies in knowledge between patients and staff and that the nearby patients whose very lives were in danger would be even more concerned then their attending physicians. The hospital administration had been quite tight-lipped about the crisis and subsequent criminal investigation, and even the medical staff relied on local newspapers as the principal source of information about the deaths.

The patients were divided into 2 groups—close and distant. Patients were assigned to the close group if they gave a positive answer to any of the 6 questions concerning the medical care or procedures that they had experienced while in the hospital, e.g., receiving general anesthesia, undergoing surgery, receiving intravenous fluids, being hospitalized in the ICU or the coronary care unit, being close physically or emotionally to someone who had experienced a respiratory arrest, or personally having undergone an arrest. All other patients were placed in the distant group. There were 40 patients in the close and 45 patients in the distant group. The staff group was trimmed to just those who performed medical duties, i.e., doctors, nurses, and aides. Staff members were assigned to the close group if they gave a positive answer to 1 of the following 3 items: being involved in giving intravenous fluids, having worked on a ward where an arrest occurred, and knowing a patient who had undergone an arrest. The close staff group was composed of 43 members and the distant group, 49.

While our hypotheses basically held true for the staff, this did not prove to be the case for the patient group. The close patient group differed dramatically in the opposite direction from what had been expected. This group had more faith in the institution, tended to dismiss the idea of homicide, and was more disapproving of press coverage than was the distant patient group. When we compared the close patient group with the close staff group, with whom they supposedly shared a common environment but were expected to feel even more emotionally threatened because of their medical vulnerability, it was obvious that the two groups were not dealing with the same psychic reality. The close staff group demonstrated increased concern, while the close patient group showed remarkable confidence and faith. This was the exact opposite of what was hypothesized and was interpreted as denial on the part of the close patient group.

Although the trend did not reach statistical significance in the direction opposite that hypothesized, only 16% of the close patient group, compared with 35% of the distant patient group, would have transferred a relative to another facility. However, the staff responded in the expected direction, with 56% of the close group but only 27% of the distant group concerned enough to seek the transfer of a relative ($p<.025$). When the close patient and close staff groups were compared, the difference in the perception of danger in the hospital was even more readily apparent, with the two groups differing significantly in the direction opposite to what was predicted ($p<.005$).

A similar pattern held true for the question as to whether one would be a patient at the hospital in the future. Both patient groups showed nearly unanimous support for the institution (97% close, 86% distant), and the lack of difference between the close and distant patient groups again ran contrary to what was hypothesized. In contrast to the patients, the staff conformed to the hypothesis; only 55% of the close staff and 69% of the distant staff were willing to be a patient at the hospital in the future. The difference in faith in the institution demonstrated by the close patients when compared to the close staff group attending them ironically could not be expressed statistically because too few close patients fell into the category of being unwilling to return to the hospital. The relatively high regard the FBI was held in by patients regardless of category (about 67% overall) and the generally skeptical attitude of the medical staff (about 45% overall) was again evident in question 3.

The most interesting finding was the conception of events that the patients closest to the crisis chose to believe. They did not perceive themselves in danger from a murderer but felt instead that the respiratory arrests were nondeliberate—either coincidence or medical error. Only 35% felt that the cause of the respiratory arrests was deliberate (murder), compared with 69% of the distant patients. This difference was significant ($p<.01$). The distant patient group was apparently reading the papers and watching television and did not differ in its perception of the events from that of the 2 staff groups. Over 70% of both staff groups felt the respiratory arrests were deliberately caused. The perception of events expressed by the close patient group was once more at odds with that of their attending staff, differing in the direction opposite to that hypothesized ($p<.005$). This apparently was denial on the part of the close patients—perhaps the basis of all unrealistically trusting, but sometimes beneficial, "good" doctor-patient relationships.

The response to press coverage again showed the close patient group running counter to the hypothesis and differing from the distant patient group in being less, not more, appreciative of media coverage. Over 65% of the group felt the press exaggerated the situation and did not do either the public or themselves a service. However, the distant patients were more satisfied with the media coverage and saw it as useful; 60% felt that the coverage was not exaggerated ($p<.05$).

Medical staff reaction also ran counter to what had been expected. The close staff did not seem to feel that it had found a mutuality of interest with the media, who also seemed to grasp the gravity of events. Approximately 80% thought the coverage was exaggerated and of no benefit to the patients or public. This did not differ from the view of the distant staff, which was also highly critical. There was a feeling that the press had primarily served itself while serving the public. On this issue the close patient and close staff groups tended to view the situation in a similar manner, since both had a vested interest in dampening the effects of irrational personal panic or public hysteria.

DISCUSSION

Anna Freud (4) described denial as one of the earliest and most basic defensive maneuvers. In its original form, denial is naturally and constructively used by the young child to mitigate and adjust to an external world of powerful, potentially threatening forces with which it is physically unable to cope. Adults also use denial in much the same manner. Aitken-Swan and Easson (5) found that 19% of 231 British cancer patients who had curable lesions of the skin and had been informed of their condition denied having a cancerous condition on re-interview between a week and a month later. Lewin (6) noted the central place of denial in the dynamics of elation in order to generate unrealistic confidence and optimism. Hackett and associates (7–9) found a similar function of denial in the coronary care unit, where denial often served to allay anxiety, maintain hope in a life threatening situation, and generally aid in the patient's acute recovery. They developed a denial scale that was used by Froese and associates (10) in a subsequent study, which found that in the earliest stages of hospitalization for myocardial infarction, patients who used denial tended to gain control over their anxieties sooner than nondenying patients. During the acute stage of the illness, the defense of denial was not as dangerous a mental mechanism as such a blatant distortion of reality characteristically poses over the long term.

Weisman and Hackett (11, 12) explored further the process of denial and its dynamics in another area where it is often used: terminal illness. They saw it not only as a way of avoiding anxiety to one's physical person and internalized self-concept, but also as a positive mechanism for maintaining social contact and relatedness. They stressed the social context under which the denial occurred and the personally constructive ends an individual hoped to pursue by using such a defense. They also drew an important distinction between the process of denying and the act of denial, a distinction that points up the socially determined quality of much of human perception. The process of denying entailed the following four elements: a mu-

tually agreed-upon field of perception, a repudiation by an individual of part of this field of perception, the replacement of this repudiated section with a perception that is more congenial and which reinforces affirmative action, and a reorientation of the individual who has survived for the moment so that he can continue mutual interactions and relationships with others. The act of denial required a fifth step—the judgment by one person upon another that he has denied an event within their common reality. The individual is not aware that he is denying in the present and only becomes aware in retrospect. His principal aim has been to maintain himself and his social relationships.

These elements of the process of denial were very much in evidence among the close patient subgroup in the hospital. A threatening, murderous piece of reality was repudiated and replaced by a more benign view of events. This view of reality helped to ward off anxiety over routine medical procedures and once more allowed affirmative and active cooperation with the medical staff. The continued functioning of the hospital helped to reaffirm their relationship to an institution that had long been a source of medical care and support.

Unfortunately, there are no data on patient and staff attitudes toward the VA system, the FBI, or the media before the crisis. Social class differences alone may account for some of the variance. However, denial mechanisms often protect individuals and social groups from being overwhelmed by anxiety or panic that would otherwise paralyze all functioning and prevent even the initiation of constructive responses. Seemingly, both the close patient subgroup and the staff were critical of media coverage because they saw it as inadvertently, or for its own ends, stirring those forces of personal anxiety and public hysteria that they were attempting to contain. Although the hospital could not do business as usual, with the proper precautions the ill would continue to be cared for and efforts at medical, social, and legal restitution would be begun.

Fortunately, the denial of the patients was accompanied by vigorous preventive measures adopted by the hospital and did not serve as an invitation to further tragedy. While this paper was being written, a federal court issued a 16-count indictment including 5 counts of first-degree murder against 2 nurses who had served on the evening shift in the ICU (13).[1] This by no means marks a legal resolution to the events, since their trial will be closely contested and the eventual outcome is uncertain. However, with the imposition of tighter general security measures, the hospital has returned to normal patient care and functioning.

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[1]On July 13, 1977, after a lengthy trial, the 2 nurses were each convicted of conspiracy and 3 counts of poisoning. Efforts at appealing the verdict are currently under way.

Early Sexual Experience and Prostitution

BY JENNIFER JAMES, PH.D., AND JANE MEYERDING

The authors compared several aspects of early sexual experience found in two earlier studies of prostitutes with results of research on "normal" women. The prostitutes had in common many negative experiences not found or found less often in other populations of young women. These include incestuous and/or coerced sex, lack of parental guidance, intercourse at a young age, and few or no meaningful relationships with males. These women had discovered that sex could lead to a kind of status, even though that status is negatively labeled by the wider culture. In a society that values women on the basis of their sexuality, a woman who views herself as "debased" may see prostitution as a viable alternative—perhaps the only alternative.

IT HAS BEEN ARGUED by prostitutes and social scientists alike (1, 2) that prostitution is an aspect rather than a contradiction of the female sex role in our society. What then determines which women will act out the prostitution components of that role? According to Rosenblum (2), "access to prostitutes and perhaps specific incidents in the life of the individual" are the deciding factors. One of us (J.J.) conducted studies of streetwalkers in a large western city that seemed to indicate a common pattern of certain sexual experiences—many of them negative—among the 228 subjects studied (3, 4). Although there is more to the profession of prostitution than just sex (e.g., economics, customer demand, a subcultural ethos), it seems clear that a woman's self-concept of her sexuality must play an important part in her decision to prostitute and that sexual experiences unrelated to prostitution may have considerable impact on the development of this self-concept. In this article, we will present data on the sexual experiences of subjects in these two studies that we feel may be particularly relevant to the development of the deviant self-image of "prostitute." The experiences we will consider are age at first intercourse, early sources of information about sex, subsequent relationship with first sexual partner, number of sexual partners, number of "significant relationships," incest, and rape.

Both studies involved questionnaires, interviews, and ethnographic field observation; thus, data are limited to "self-report." The first study (referred to as study 1) was conducted between 1970 and 1972 with a sample of 72 adult prostitutes and 20 adolescent prostitutes contacted on the street and in jail (3). The second study (study 2), conducted during 1974–1975, included 136 prostitutes contacted in the same way; 68 of these subjects were also defined as addicts (4). In order to make our data more meaningful, we will use comparisons with studies of "normal" female sexual experience.

AGE AT FIRST INTERCOURSE

It is difficult to compare data from different studies on age at first intercourse because of varying methods of data presentation. For example, Sorensen's data (5) on age at first intercourse do not differ significantly from the results in study 2 except that a larger number of subjects in the latter study had their first intercourse at age 12 or younger (13% versus 7%). However, since 48% of Sorensen's total sample had not had intercourse, we can conclude only that there seems to be a section of the general adolescent population, of which the subjects of studies 1 and 2 are members, who are more sexually active at an earlier age than the norm.

Other studies support the conclusion that our subjects initiated sexual activity at a younger age than the general population. Kantner and Zelnik (6) compared age at first intercourse for the black and white members of their sample. They found that 54% of their black subjects experienced first coitus between the ages of 15 and 19 (74.9% of them by age 18), whereas only 23% of the white subjects had their first experience at those ages (19.9% by age 18). In the study 2 population, 56.6% reported first intercourse between the ages of 15 and 19 (36% had had intercourse before that age), and 91.9% of the sample had had coitus by age 18. In striking contrast is the figure of 17% nonvirgins at age 18 found by Simon and associates (7) in a sample limited to college students. Four-fifths of the adolescents in study 1 had their first sexual intercourse at age 14 or younger. Although information on sexual experiences before first intercourse was not elicited in the questionnaires in studies 1 and 2, extensive interviews revealed a pattern similar to the one found by Davis (8)

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in her study of 30 prostitutes: "The 'technical virginity' pattern typical of the middle-class female was not in evidence here. First sexual contacts typically involved sexual intercourse, with only one girl reporting an initial petting experience."

EARLY SOURCES OF INFORMATION ABOUT SEX

The first questions relating to sexual history in studies 1 and 2 attempted to elicit the subjects' major sources of early information about sex. In study 1 (3), 63.2% of the adolescent subjects—toward whom the questions on sexual history were aimed—learned about sexual intercourse from friends (36.9%) or personal experiences (26.3%). Study 2 produced more detailed information (4). The role of parents as sex educators was considerably less in study 2, the more recent sample, than in Wittels' 1951 study (9) (15.4% versus 34%). Friends continue to be the major source: Wittels found a figure of 42.7% and the study 2 figure was 37%, Adding the study 2 category of "siblings" to that of "friends" to produce an "other children" grouping increases the figure for this category to 44.9%. The role of teachers in sex education was somewhat smaller in study 2 than in Wittels' study (5.9% versus 9%), as was the influence of physicians (1.5% versus 3%). Wittels' final category, "other adults," has no counterpart in study 2. The 17% Wittels found in this category must balance against the categories of "books," 10.3%; "church," 0.7%; and—perhaps the most significant difference from Wittels' study and a possibility he apparently did not consider—"personal experience," 14%. Sorensen (5) found that 31% of his sample had learned about birth control and 33% about venereal disease from their parents. These figures approximate Wittels' 34% figure for the role of parents and reemphasize the contrast with the study 2 finding of 15.4%. The close agreement between Wittels' and Sorensen's data on this point is particularly striking given that the former study was conducted in 1951 and the latter in 1973.

This lack of parental guidance in sexual matters is one aspect of the general weakness of the parent-child relationship that is typical of many prostitutes (8, 10–16). Whether the parent-child relationship is marked by simple neglect by absence or by outright psychological or physical abuse, the result for the child is generally considered to be alienation from the parents and a consequent inability—the severity of which depends on the circumstances—to adequately socialize the conventional mores of "respectable" society.

SUBSEQUENT INTERCOURSE WITH FIRST PARTNER AND NUMBER OF SIGNIFICANT RELATIONS

The use of different groupings (e.g., "5 to 6 times" versus "5 to 10 times") makes it difficult to do a strict statistical comparison of the number of times subjects in various studies had intercourse with their first coital partner. It seems obvious, however, that the most significant difference between the results in study 2 and those from studies of "normal" populations is the percentage reporting no further intercourse with their first partner. In study 2, 33.8% reported they had no further sexual relationship with their first partner, whereas other studies found figures of 10%–15%. Table 1 compares findings from study 2 with those of Eastman (17).

TABLE 1
Subsequent Intercourse with First Coital Partner

	Eastman (17) (N=43)		Study 2 (4) (N=133)	
Frequency	Number	Percent	Number	Percent
Never again	6	14.0	45	33.8
1–2 times	6	14.0	15	11.3
3–10 times	12	27.8	25	18.8
11 or more times	19	44.2	48	36.1*

*Includes categories: "11–20 times," 8.3%; and "regularly," 27.8%

TABLE 2
Number of Sexual Partners in Two Samples of Women*

	Kinsey and Associates (18) (N=1220)		Study (2) (N=135)	
Partners	Number	Percent	Number	Percent
1	647	53	4	3.0
2–5	415	34	25	18.5
6–10	85	7	37	27.4
11–20	49	4	22	16.3
21 or more	24	2	47	34.8

*Customers are excluded in figures for study 2.

That the superficial, nonemotional nature of the first sexual intercourse of many of these women initiated a series of such encounters is supported by the fact that the mean number of private (i.e., not for profit) sexual partners of subjects in study 2 was 23. Even more significant is the fact that the mean number of persons with whom these subjects felt they had developed a "significant relationship" was only 5: 17.8% reported more than 10 and 71.6% reported 5 or less. Table 2 compares data from study 2 on numbers of sexual partners with data from Kinsey and associates (18).

INCEST AND RAPE

In their 1953 report on female sexuality, Kinsey and associates (18) found that 24% of their sample had experienced at least one sexual advance by an older male (defined as being at least 5 years older than the subject); they stated that the percentage would have been greater if the sample population had included more

TABLE 3
Adult Perpetrators of Sexual Advances Toward Children

Adult	Kinsey and Associates (18)		Gagnon (19)		Study 2 (4)		De Francis (20)	
	N	%	N	%	N	%	N	%
Stranger	559	48.7	195	58.5	13	20.6	63	25.0
Friend or acquaintance*	344	29.9	88	26.5	27	42.8	93	37.0
Father	43	3.7	5	1.5	4	6.4	33	13.0
Stepfather**	—	—	—	—	9	14.3	35	14.0
Foster father	—	—	—	—	3	4.8	—	—
Other relative***	204	17.7	45	13.5	7	11.1	28	11.0
Total	1150		333		63		252	

*In Study 2, the category was "family friend."
**De Francis' category was "parent surrogate." Offenders were virtually all male.
***Kinsey's categories included "uncles," 9%; "brothers," 3%; "grandfathers," 2%; "other relatives," 5%.

lower-class respondents. This figure and Gagnon's later figure of 28% (19), contrast sharply with the findings in study 2: 46% responded affirmatively to the question "Prior to your first intercourse, did any older person [defined as more than *ten* years older] attempt sexual play or intercourse with you?"

Kinsey and associates (18), Gagnon (19), and De Francis (20) attempted to ascertain the relative incidence of participation by various categories of males in such sexual advances (see table 3). Comparing data from study 2 with those of Kinsey and associates and Gagnon, it is apparent that the findings in the categories of "strangers" and "father figures" are very different: 3.7% of the perpetrators in Kinsey's study and 1.5% in Gagnon's work were fathers, whereas study 2 found that 25.5% were fathers, stepfathers, or foster fathers. Combining the percentages of fathers and "other relatives" in these three studies gives the following totals for sexual experiences with an "incestuous" character: Kinsey and associates, 21.4%; Gagnon, 15%; and study 2, 36.6%.

De Francis' data more closely approximate those from study 1. He found that 27% of such offenders were father figures; 38% were relatives.[1] In study 1, 13 of the 20 adolescent subjects reported having had a "forced/bad sexual experience,"[2] the majority of which (11 of 13) occurred when the subject was 15 or younger. In many cases the men responsible were fathers (23.1%) or other relatives (15.4%). Of the total sample of 20, one-fourth had experienced a negative sexual relationship with a relative. Sgroi (21) also reported that "The most frequently named perpetrator in cases of sexual abuse (of children) is the father or a male relative or [mother's] boyfriend."

None of the studies of "normal" female sexual experience available for comparison included statistics on the number of subjects who experienced the use of force in their first intercourse. In study 2, 23% of the subjects reported that they had been subjected to physical force in their first intercourse, and 7.4% felt they had been victims of emotional coercion. More than half (57.4%) of the study 2 sample reported that they had been raped at least once in their lives. Of these, 36.2% had been raped more than once, and 7.5% had been raped by multiple assailants.

DISCUSSION

Societal reactions to juvenile female sexual activity may influence the entrance of some young women into prostitution, especially those who are more sexually active and less discreet than the majority of their peers, as seems to have been the case with the subjects in studies 1 and 2. Choisy (11) asked, "At what number of lovers is a girl supposed to lose the status of a decent person?" Carns (22) stated that "A woman's decision to enter coitus . . . implies that she is creating for herself a sexual status which will have a relatively pervasive distribution . . . she will be evaluated downwardly. Such is the nature of the male bond." Girls learn early society's moral valuation of their sexuality. For example, in discussing her childhood sex education, one streetwalker stated, "I think the basic theme of the whole thing was that it was a dirty thing but that it was a duty for a woman to perform, and if you fooled around, you were a prostitute." Female promiscuity, real or imputed, virtually guarantees loss of status in our majority culture: one hears such statements as "I got pregnant and was kicked out of the house and school" or "I was accused of being promiscuous while I was still a virgin. They did that because I used to run around with a lot of guys." The labeling implied by such loss of status may be an important step in the process by which a woman comes to identify with a deviant lifestyle such as prostitution and thus begins to see it as a viable alternative. Davis (8) described this process in her discussion on the effects of institutionalization:

[1]It should be noted that De Francis' sample population is strongly biased in the opposite direction from those of Kinsey and associates and Gagnon, i.e., toward the lower class, and his sample consisted of "cases" serious enough to come to the attention of police or child protection agencies.

[2]Sexual experience is defined as intercourse. The force involved included both physical and emotional coercion in which the subject's self-report indicated she was sexually used against her will.

> The adolescent girl who is labeled a sex offender for promiscuity . . . may initially experience a conflict about her identity. Intimate association with sophisticated deviants, however, may provide an incentive to learn the hustler role . . . and thus resolve the status anxiety by gaining prestige through association with deviants, and later, experimentation in the deviant role. (8, p. 305)

Even in noninstitutionalized women, the labeling impact of status loss must have a strong effect on self-image. The woman may attempt to rebuild her self-image by moving into a subculture where the wider society's negative labeling will not impede her efforts toward a higher status—although that status itself will be perceived as negative by the wider society. Of the prostitutes in study 2, 44.9% reported that they had "gotten into trouble" because of their sexual activity while they were juveniles.

No matter how negative the long-term effects of juvenile promiscuity on a woman's social status are, the short-term effects may often seem quite positive to her. Young women who suffer from parental abuse or neglect, a common pattern for prostitutes, may be especially susceptible to the advantages of what Greenwald (15) called "early rewarded sex . . . engaging in some form of sexual activity with an adult for which they were rewarded. [These women] discovered at an early age that they could get some measure of affection, of interest, by giving sexual gratification." This type of positive reinforcement for sexual behavior, particularly when coupled with the cultural stereotype of women as primarily sexual beings, may cause some women to perceive their sexuality as their primary means for gaining status. Davis noted that "Sex as a status tool is exploited to gain male attention" (8). All women in our culture must in some way come to terms with the fact that their personal value is often considered inseparable from their sexual value.

> While men are also concerned with their sexual desirability, their opinion of themselves is not founded primarily on that desirability, for occupational achievement provides an important alternative to a self-identification based on sexual desirability. The alternatives available to females are fewer and generally carry lower social esteem, resulting in an inordinately high value being placed on sexual desirability. (2, p. 180)

The effect of incest on the child involved is virtually unknown. Some researchers, e.g., Jaffe and associates (23), preferred not to make a judgment, noting that "Little is known of the physical and emotional effects of incest." Ferracuti (24) stated that "it is hardly proved that participation in incest . . . results in psychological disturbances." He noted, however, that "Frequently [victims of incest] become sexually promiscuous after the end of the incestuous conduct." De Francis (20) found guilt, shame, and loss of self-esteem to be the usual reactions of the child victims of both incestuous and nonincestuous sex offenses. These feelings often led to disruptive, rebellious behavior, and some older (i.e., adolescent) victims later become prostitutes. Sexual abuse that continues over a long period of time, as is usual with incest, was found by Gagnon (19) to be "extremely disorganizing in its impact" on the victim. Weiner (25) echoed Ferracuti in stating that "girls who begin incest in adolescence frequently become prostitutes following termination of the incest."

The incidence of rape experiences among the study 1 and study 2 populations, especially among the adolescents in study 1, is so high that these samples present the characteristics of an especially victimized group. Although the subjects' identities as rape victims or nonrape victims were not in any way considered in the sampling procedures, 65% of the adolescent prostitutes had been the victims of coerced sexual activities. The 57.4% of the study 2 sample who had experienced rape, including 36.2% who were multiple rape victims, is also disproportionate compared with wider samples of women. When the societal stereotyping of women as primarily sexual beings is expressed through the violence of rape, the psychological effect on the victim may be one of reinforcing her self-concept as a sexual object and further isolating her emotions from her sexuality.

CONCLUSIONS

The prostitutes in the two studies we have considered shared a range of negative sexual experiences that—in conjunction with other circumstances and experiences—may have influenced them toward accepting prostitution as a lifestyle. The pattern that emerges includes 1) lack of parental guidance that leads to early, casual sexual intercourse to the exclusion of the more usual noncoital sociosexual experimentation; 2) the discovery that sex can be used to gain a kind of social status, coupled with the subsequent discovery that this status is perceived by others as negative, making the individual unacceptable to the majority culture; and 3) the emotionally destructive experiences of incest and rape. Because the range of acceptable sexual behaviors is much narrower for women than for men, and because women more than men are judged (by themselves and by others) on the basis of their sexual desirability and behavior, sexual experiences may be a more important factor in a woman's development of self-identity. A woman who views herself as sexually debased or whose sexuality is more than normally objectified may see prostitution as a "natural"—or as the only—alternative.

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Planning for a Mental Hospital Phasedown

BY M. POWELL LAWTON, PH.D., MORTIMER B. LIPTON, PH.D., MARK C. FULCOMER, PH.D., AND MORTON H. KLEBAN, PH.D.

The authors studied the patients and community facilities in an area for which a mental hospital phasedown was contemplated. They found that patients who were continuously hospitalized during a 9-month period were highly dependent and judged unlikely to be candidates for community placement. By contrast, the patients who had been discharged during this 9-month period were judged relocatable by staff and constituted a select group in terms of independence. The authors' survey of community facilities indicated few placement possibilities for the majority of the continuously hospitalized patients.

CONCEPTS OF WHAT constitutes desirable treatment for the psychiatrically disturbed have changed greatly since the publication of the report of the Joint Commission on Mental Illness and Health in 1961 (1). Realization of the deficiencies of public mental institutions led to the creation of the community mental health network and the search for community-based alternatives to institutionalization. Over the past decade a massive effort to deinstitutionalize psychiatrically disturbed patients and to forestall the admission of new patients has been made, resulting in an overall reduction of the public mental institution population from 527,000 in 1961 to 338,000 in 1970 (2), even though as of 1973 only 12 state hospitals had closed completely (3).

The consensus of most mental health professionals is that the emphasis on community-based treatment has been well placed. On the other hand, many problems associated with the deinstitutionalization process have arisen. "Community placement" has often meant transfer to nursing homes or boarding homes in which the environment is impoverished at best and more dehumanized than many mental hospitals at worst (4–6). Many reports, including those in the popular media, have also called attention to the isolation of the ex-patient and the failure of the community mental health system to provide services for the many ex-patients who live alone after their discharge from the hospital (4–6).

The concentration of large numbers of ex-patients in certain neighborhoods has created many problems for other community residents and has sometimes led to a backlash of attempted restrictive zoning and other unwelcoming behavior (4). Although a number of studies have suggested that there are positive outcomes associated with favorable community placements during a hospital's phasedown or closing (7, 8), national trend data showing increases in inpatient episodes (9) and in readmissions (3) suggest that there is some reality to the idea that the "revolving door" has replaced continuous institutionalization as the norm for chronically ill patients.

Policy that makes mental hospitalization more difficult has in some instances forced mentally ill patients to use acute-hospital care, which is far more expensive, for longer periods than necessary (6). There is some evidence to suggest that the shock of transfer itself has negative consequences in health and mortality for elderly mentally ill patients (10, 11), but this is not a consistent finding (12). In some cases, an interinstitutional transfer appears to stimulate later community placement (13). A comprehensive review of these and other issues may be found in *Deinstitutionalization* by Bachrach (14).

The point of this brief review is to suggest that there are enough negative features associated with phasedowns of mental hospitals to warrant giving increased attention to averting problems. Despite the fact that phasedowns have been implemented, there are few firm research findings to document the frequency with which problems have occurred or to assess long-term consequences for patients, communities, and treatment systems.

There are few reports on the processes by which large-scale movement of patients out of hospitals has been accomplished, and fewer yet on the use of ongoing data systems as the basis for planning. In this re-

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port we will describe the beginning of an effort to phase down an institution, highlighting the use of a formal program evaluation structure as the basis for decisions regarding the feasibility of the plan. We will emphasize some of the limitations of the specific method used in the hope that improvements might be incorporated into future efforts.

This research was done by the Program Research Department of Norristown State Hospital at the request of the Pennsylvania Department of Public Welfare to provide information regarding the feasibility of at least a partial phasedown of a 550-bed state hospital (not Norristown State Hospital). The physical facilities of this hospital were for the most part very old; it would be possible to bring them up to code requirements only at great expense. The hospital served two counties with a population of 361,000 that contained one moderate-sized city, several small cities, and a large rural area.

The task was defined as the determination of the number of currently hospitalized patients for whom an alternative other than the state hospital might be feasible and the types of non-state-hospital alternatives available in the geographical area.

The data on patients were obtained using the Systematic Treatment Evaluation Procedure (STEP), a psychometrically derived rating document designed to obtain information regarding each patient's current mental, physical, and behavioral status; current diagnosis; current and recommended referrals and services; estimated date of separation from the institution; and eventual expected residential location. The STEP has been described in greater detail by Lipton and Hespenheide (15), Fulcomer (16), and Kleban (17) as a component of an integrated treatment-oriented data system.

The STEP forms were completed by the treatment-unit team for 550 patients who were hospitalized continuously from May 1, 1975, through February 28, 1976, and for 104 patients who were discharged during that period. This represents all of the patients in the hospital during this 9-month period except for those who died before February 28, 1976, and 14 patients who were discharged before STEP ratings could be completed. All of the staff presumably knew about the potential closing of the hospital and had direct contact with the program research staff during the data-gathering period.

The primary purpose of the STEP document was to give a descriptive picture of the universe of patients, including those for whom relocation was a possibility.

CONTINUOUSLY HOSPITALIZED PATIENTS

The diagnoses of 531 of the continuously hospitalized patients are shown in table 1. Because of the probability that patients under 21 years old (N=8) and those hospitalized under a criminal court commitment (N=10) would have a short hospital stay, these patients were not classified by diagnosis. Among patients classified as psychotic (primarily schizophrenic, depressive, and manic-depressive psychoses), we made the further distinction between the 92% who were receiving continued treatment and the 8% who were receiving intensive treatment. A patient was considered to be receiving intensive treatment if he or she had either been hospitalized for less than 1 year or if the estimated date of separation was 1 year or less.

TABLE 1
Primary Diagnoses (N=531)* and Optimal Residential Expectations (N=550) for Patients Continuously Hospitalized for a 9-Month Period

Item	Number	Percent
Diagnoses		
Psychosis	352	64
Organic brain syndrome	111	20
Mental retardation	51	9
Psychoneurosis	8	1
Personality disorder	9	2
Optimal residential expectations		
Not in mental hospital		
Patient's own home	24	4
Home of relatives or friends	53	10
Supervised residence	48	9
Foster home	37	7
Long-term care facility	30	5
Other institutions	12	2
Other	1	
Total	205	37
In mental hospital		
Remain in same mental hospital or go to another	182	33
Not likely to leave hospital for any reason	163	30
Total	345	63
All continuously hospitalized patients	550	100

*Excluded are 8 patients under the age of 21 (1% of all 550 patients); 10 patients hospitalized by criminal court order (2%); and 1 patient whose diagnosis was missing.

Less than 2% of the continuously hospitalized patients were under 21 years old; 35% were 65 or older. Of the 325 psychotic patients receiving continued treatment, 56% were 65 or older, and 70% of the patients with organic brain syndrome were in this age range.

On a 7-point rating scale of general health on which 1=no apparent problem and 7=problem of devastating proportion, 29% of the 550 patients had a rating of 4 (relatively severe problem) or more in general physical health.

Two aspects of independence were rated on 5-point scales ranging from totally dependent to totally independent: capacity for basic self-care and degree of residential supervision required. Ratings on these two scales were combined into a single 4-point dependency rating: 1=very highly dependent (42% of the continuously hospitalized patients); 2=highly dependent (27%); 3=moderately dependent (20%); and 4=minimally dependent (11%). The patient population was thus highly skewed in the direction of dependency (median rating=1.8). The concurrent validity of the dependency rating was demonstrated by its highly sig-

nificant correlations with factor scores of cognitive status, withdrawal, self-care ability, disorganized communication, and social activity derived from STEP clinical ratings of 72 highly specific behavioral and symptom manifestations (16).

The treatment teams were asked to make estimates of each patient's probable date of separation on the basis of the patient's current condition, past history, and the possible environmental opportunities. The task was phrased in this manner to concretize the judgment of prognosis and thereby encourage maximum discrimination over a wide range of favorable to unfavorable prognoses. Eighteen percent of the 550 patients were judged likely to be able to leave the hospital within the earliest possible time frame for mass relocation (1 year), 23% after 2 years, and 29% after 3 years. Sixty percent were judged never likely to separate. Thus, staff were generally pessimistic about separation; this was consistent with their ratings of dependency.

The staff were also given a checklist of 25 possible residential locations (plus spaces for "other") to which a patient might be transferred should he or she leave this state hospital. Since one or more locations could be checked, the optimal choice was considered the one allowing the highest level of independence, as determined by research staff ratings of the whole list in terms of the degree of independence afforded in the average living situation represented by each choice. Only 37% of the 550 patients were judged capable of ever leaving a mental hospital (see table 1). Since "mental hospital" was listed as a residential expectation, separate figures are shown for this alternative and for when no change in type of residence was expected, although there is no substantive difference between these two categories.

DISCHARGED PATIENTS

During the 9-month study period, 104 patients were discharged to locations other than mental hospitals. These patients were also evaluated by the STEP document, but they are not included among the 550 continuously hospitalized patients. Eighty-eight percent of the 104 discharged patients had been judged capable of leaving the hospital within 9 months or less, a finding that supports the validity of the expected date of release estimate. Of the 104 discharges, 65% were to relatively "easy" placements, i.e., to their own home or the home of relatives. The median length of hospitalization of all 104 discharged patients was 11.3 months, and their median dependency level at the time of initial STEP evaluation was 3.1 (slightly better than moderately dependent), compared with the median of 1.8 for the 550 patients who stayed in the hospital during the study period. These figures demonstrate that patients who were discharged had short-term illnesses, were considered the most independent, and had environmental supports available.

In summary, the treatment team's assessment of the functional capacity and symptoms of the patients who were continuously hospitalized and of those who were discharged during the study period clearly documents the very low level of independence of the great majority of patients and the highly selected characteristics of those who were discharged.

AVAILABLE COMMUNITY ALTERNATIVES

We also surveyed existing facilities for discharged mental hospital patients for relocation possibilities. The social service personnel of the hospital compiled a list of all of the facilities in the two-county area that were known to have accepted discharged patients recently and were potential service resources. The survey thus omitted an unknown number of facilities that had not been used because they were of poor quality, charged fees that were beyond the state's or the patients' financial resources, or simply had not been discovered as possible resources.

A social worker or research assistant visited each facility and conducted a structured interview with the administrator or owner regarding administrative practices, staffing, treatment programs, physical features, finances, and policies regarding the acceptance of clients. There were 23 foster homes in the area, representing a combined resident capacity of 61 with 15 vacancies; 24 supervised residences, representing a capacity of 471 with 78 vacancies; and 22 long-term care facilities, representing a capacity of 1,469 with 56 vacancies.

Comparison of the number of vacancies (N=149) with the number of patients judged suited for placement in a foster home, supervised residence, or long-term facility (N=115; see table 1) indicates that, with the exception of foster homes, there are enough vacancies (other institutions such as medical hospitals, institutions for the retarded, or rehabilitation hospitals were not included in this survey of facilities). However, responses to a series of questions regarding possible criteria for excluding admissions revealed many reasons to expect great difficulty in matching a placeable patient with an available space.

Twenty-six of the facilities admitted either only men or only women; 3 would not admit patients who were mentally retarded; 65 excluded some patients on the basis of the degree of their mental illness; 27 excluded patients who were not ambulatory; 24 excluded patients who were not able to take care of themselves; 24 would not admit substance-abusing patients; 45 excluded patients with particular physical illnesses or disabilities; and 18 had other excluding criteria. No facility was without such restrictions, and most reported more than one criterion for exclusion.

One can see that the number of reported vacancies actually available for placement is drastically reduced if the exclusions are taken into account. This number would again be reduced by considerations not covered

in the present study, such as patient preference, relatives' preference, cost of the placement, and geographic location. The only factor that might operate to increase the number of placement opportunities would be the addition of new facilities, the discovery of additional existing facilities, or the active promotion of such additional resources as foster homes.

DISCUSSION

To summarize our major findings: 1) the great majority of patients served by this state mental hospital were highly dependent, long-term, chronic, and elderly; 2) a minority (18%) were judged capable of living in a location other than a state mental hospital within a period of 1 year; 3) the population actually discharged was primarily an acutely ill, relatively recently admitted group; and 4) existing community facilities would be hard put to accommodate even the small number of patients with the best prognoses. The deficits among the large majority of patients would almost certainly preclude their acceptance by any community facility.

These conclusions are entirely consistent with those of Deasy and Steele (18) in a study of the community residence potential of mental hospital patients in Florida. They also found an excess of demand over supply in both community mental health facilities and nursing homes.

Perhaps the most vulnerable point in this study is the fact that the source of much of the data was a group of employees whose jobs had been threatened and who knew that the survey results would be relevant to the ultimate outcome of the threat. Fortunately, the 72 clinical ratings of the STEP document had been used about 18 months earlier in large representative patient samples (N=747) from three other hospitals in the state system that have never been mentioned as candidates for the phasedown program. The levels of rated psychopathology in these nonthreatened hospitals (mean standardized factor score=2.837) were virtually identical to those found in the targeted hospital (mean=2.803). If bias had been present, mental state would surely have been judged poorer in the threatened hospital.

It was also impossible to make a specific estimate of the number of patients who could be accommodated in each type of facility or to make a projection of the number of additional resources that would be required over the next few years to accommodate the eligible patients. This gap was purely a matter of limited research resources. It is entirely within the capability of a larger research project to obtain more detailed patient and facility information and a near-100% sample of all facilities and to match individual patients with individual facility types, taking account of specific admission policies. These data would provide valuable information for short- and long-term planning.

This picture can never be complete without considering patient and family preference, the constraints of distance, and the financial matching of payer and vendor. Finally, considerable sharpening of our ability to measure the quality of treatment will be necessary before we can know whether a particular type of facility can really deliver the care needed by a specific patient. The matching of patients to facilities chosen without reference to their quality clearly risks mismatches.

Given the above limitations, one may conclude that this study of patients and community facilities gives considerable reason for caution in the planning of mental hospital phasedowns. One simply cannot wish out of existence the realities of chronic psychosis and chronic patienthood on the one hand and the short supply of community placement possibilities on the other, to say nothing of the deprived environments of many community facilities. The hard line of the past decade on institutionalization has unquestionably had a favorable effect on the unnecessary incarceration of large numbers of people. Although our data suggest that there is still a gap between the number of patients who are capable of living in the community and the number of opportunities for community living, the gap has greatly narrowed. The tasks for the future seem to be to close this gap completely by improving the quality of alternative treatment environments that are now so poorly serving patients released to the community and to relinquish the fantasy that all patients are better served in the community.

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Social Disability in Chronic Psychiatric Patients

BY JUDITH A. SYLPH, M.A., DIP.SOC.ADMIN., HELEN E. ROSS, B.A., B.J., AND HENRY B. KEDWARD, M.D.

The authors measured the social functioning of 147 chronic psychiatric patients using a rating scale originally devised for use with the mentally retarded, the Adaptive Behavior Scale (ABS). They found that social deficits were more widespread and generally more severe among chronic psychiatric patients than among institutionalized mentally retarded subjects. Chronic undifferentiated schizophrenic patients showed the most comprehensive deterioration; other groups, such as neurotic patients, women, and the elderly, had more limited impairments. The authors suggest that the ABS can be useful in establishing the limits of social functioning of chronic psychiatric patients.

SINCE THE ADVENT of community psychiatry, it has become commonplace for chronic psychiatric patients to be discharged from mental hospitals. Adequate preparation for discharge is critical for this type of patient, whose social skills may be atrophied or geared to the expectations of the chronic ward. Comprehensive patient evaluation is essential to rehabilitation planning and should include assessment of social functioning to measure patients against community norms. Quantifiable social data that are detailed and specific can contribute significantly to the choice of rehabilitation program. The study reported here measured the social functioning of a group of chronic psychiatric patients with a rating scale particularly suited to a population deficient in social skills, having been devised originally for use with the mentally retarded.

METHOD

We studied the social functioning of 147 chronic psychiatric patients from two mental hospitals in Toronto. Patients were aged from 25 to 65, were currently in full-time care, and had spent a minimum of two years in a hospital or alternative residential psychiatric facility.

Psychiatric diagnoses were made by research psychiatrists using the Present State Examination (1). Schizophrenia was divided into three subgroups—chronic undifferentiated, differentiated syndrome, and residual—according to *ICDA-8* criteria (2).

Social functioning was evaluated with the Adaptive Behavior Scale (ABS) (3), selected for its comprehensiveness, its specificity, and the appropriateness of its threshold. Below an upper limit of "normal" socially acceptable behavior, a broad range of deviant or socially conspicuous behaviors can be rated. The first part of the ABS (Part I) assesses instrumental daily living skills; the second (Part II) assesses interpersonal and expressive behavior. General categories of behavior (called "domains") comprise a number of individual items whose ratings are added together to form a domain score. ABS assessments were carried out mainly by psychiatric nurses who knew the patients well and who had received instruction in the use of the scale.

RESULTS

Individual Items of Behavior

For the most part the results reported refer to the domains of behavior (see tables 1 and 2). However, a clearer picture of the group's social functioning can be gained from the individual items making up the domains.

On Part I of the scale, over half of the 60 items assessing daily living skills were found to be beyond the capacity of at least 50% of the chronic psychiatric patients. One third of the patients had distasteful table manners. Almost all could dress and undress, but without supervision many would attract unfavorable attention in the community. About half were unkempt, were inappropriately attired or groomed, or would go about dirty unless reminded to wash. A slightly smaller proportion could not take a bath unaided, and 30% were unable to wash their hands and face adequately. A quarter wet or soiled themselves at least occasionally, and about 20% needed help at the toilet. Over three-quarters could not manage money satisfactorily, 57% had difficulties with public transportation, and almost half were unable to use the telephone or find their way about outside the hospital.

Impairment of work capacity was marked. Poor performance of domestic chores was universal, and only a third of the patients managed other types of work. Common types of work failure are suggested by the

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prevalence of slow and sluggish movements and lack of initiative, perseverance, and organizing capacity among 58%–75% and unreliability in carrying out assignments in 96%.

Between 10% and 30% of the patients had defects of sight, hearing, mobility, or manual control, and the body balance of 79% was rated as impaired. Language and communication handicaps were common: 10% of the patients were mute or unintelligible and a third had blocked or unclear speech. Vocabulary and sentence construction were severely limited in 60%–75%. One third could not understand complex instructions, and twice as many had difficulty reading.

On Part II of the scale the most prominent characteristics were in the domains of rebellion, withdrawal, and psychological disturbances. About half of the patients resisted instructions, ignored regulations, or neglected assignments. Over a third misbehaved in group settings, and a quarter ran away. Half of the patients were unable to tolerate frustration, 44% exhibited emotional instabilities, 41% demanded excessive attention, 37% resented criticism, 33% had outbursts of temper, 38% were violent, and 39% used profane, hostile language. Around 20% engaged in other forms of antisocial behavior, such as teasing others or disrupting their activities. Thirteen percent did physical violence to themselves.

The majority of the patients were described as withdrawn, 42% as profoundly withdrawn and inactive, and 40% as withdrawn but active. Bizarre, psychotic behavior occurred in various forms: over 40% had inappropriate interpersonal manners or disturbing vocal or speech habits, 37% performed stereotyped actions, and roughly 20% showed other oddities of behavior, such as chewing clothing or smelling everything. Sexually aberrant behavior was uncommon; between 2% and 8% of the patients were rated positively for each of the four items in this domain.

Social Functioning by Behavioral Domain

Tables 1 and 2 show the mean scores for the behavioral domains. For the purpose of comparison the optimal score for each domain is presented, together with the scores of a group of institutionalized mentally retarded adults of comparable age and IQ range derived from the data of Nihira and associates (4). No scores on normal adult populations are available. We emphasize that optimal score represents only the minimum social performance compatible with community survival.

On Part I the chronic psychiatric patients functioned at a consistently lower level than the mentally retarded subjects; the differences reached statistical significance in 9 out of the 10 domains (see table 1). The patients' best ratings were in physical development, number and time concept, and independent functioning, which covers personal care activities, and their worst were in domestic occupation, responsibilities, and socialization.

The results for Part II are presented in a different manner (see table 2) because of the difference in scoring method. Unlike Part I, high scores on Part II are indicative of maladaptive behavior; the optimal score for each domain is zero. The percentage of patients and mentally retarded subjects receiving adverse ratings and the mean scores of those who scored more than zero are presented in table 2.

Of the two groups, the proportion of psychiatric patients receiving adverse ratings was larger on all but two domains, and the differences were statistically significant on seven (see table 2). However, when subjects in the two groups who scored more than zero were compared, the retarded subjects received somewhat poorer ratings on the majority of Part II domains, although the differences were not tested statistically because the variance of the scores in the retarded population was not available. Thus, although behavior disturbance was more widespread in the chronic psychiatric patients, it was not usually more severe.

The patients' worst ratings were obtained on rebellious behavior, psychological disturbances, and withdrawal, in which the largest proportions of patients receiving adverse scores were also found.

Differences in Social Functioning Between Groups

Sex. Although there was a consistent trend for women to receive worse ratings on both parts of the ABS, this is largely a function of the greater age of the women. When age was controlled for, women performed significantly worse than men on only four domains, violent and destructive behavior, untrustworthy behavior, inappropriate interpersonal manners, and psychological disturbances. Women performed better on one domain, domestic occupation.

Age. Statistically, increasing age was correlated negatively with physical development, independent functioning, and economic activity. Older patients were also rated as more eccentric but less rebellious, less sexually aberrant, and less psychologically disturbed than younger patients.

Diagnosis. Differences between diagnostic groups were statistically significant for all Part I domains. The best scores were obtained by neurotic and affective patients throughout Part I, with the exception of physical development, general occupation, responsibilities, and self-direction, on which residual schizophrenic patients were rated best. Patients with organic syndromes or chronic undifferentiated schizophrenia were consistently placed last or next to last as least socially competent.

When age and sex were controlled for, 5 of the 14 Part II domains discriminated significantly between diagnoses. These were rebellious behavior, withdrawal, stereotyped behavior, psychological disturbances, and use of medication. Different diagnostic groups were characterized by different types of maladaptive behavior: violent and antisocial behavior was more predominant among neurotic and affective patients,

TABLE 1
Comparison of ABS Part I Scores of 147 Chronic Psychiatric Patients and 46 Mentally Retarded Subjects*

ABS Part I Domains	Best Possible Score	Chronic Psychiatric Patients		Mentally Retarded Subjects		Significance**
		Mean Score	SD	Mean Score	SD	
I Independent functioning	126	99.2	22.1	114.1	11.0	p<.001
II Physical development	26	22.0	3.8	22.6	3.3	n.s.
III Economic activity	19	10.5	6.0	12.2	4.3	p<.05
IV Language development	43	28.4	9.5	36.7	4.3	p<.001
V Number and time concept	14	11.7	3.7	13.1	2.0	p<.01
VI Occupation, domestic	19	6.3	5.1	11.6	5.5	p<.001
VII Occupation, general	13	7.4	4.9	11.0	3.2	p<.001
VIII Self-direction	33	20.3	6.7	28.4	4.5	p<.001
IX Responsibilities	6	2.8	1.8	4.6	1.4	p<.001
X Socialization	34	18.7	5.1	24.0	4.9	p<.001

*These subjects ranged in age from 41 to 60; their IQs ranged from 68 to 83.
**By t test of difference between means.

TABLE 2
Comparison of ABS Part II Scores of 147 Chronic Psychiatric Patients and 922 Mentally Retarded Subjects*

ABS Part II Domains	Chronic Psychiatric Patients Scoring More Than Zero**			Mentally Retarded Subjects Scoring More Than Zero**		Significance***
	Mean Score	SD	Percent of Patients	Mean Score	Percent of Subjects	
A Violent and destructive behavior	5.3	5.5	52.4	5.4	44.2	n.s.
B Antisocial behavior	5.8	6.1	63.3	7.1	56.5	n.s.
C Rebellious behavior	9.0	7.5	74.8	6.7	43.6	p<.001
D Untrustworthy behavior	3.3	2.9	36.7	4.1	26.9	p<.05
E Withdrawal	6.5	4.9	67.3	3.3	34.9	p<.001
F Stereotyped behavior/odd mannerisms	3.5	2.5	43.5	3.3	25.8	p<.001
G Inappropriate interpersonal manners	2.2	1.1	21.8	3.0	20.9	n.s.
H Inappropriate vocal habits	2.7	2.0	47.6	2.8	27.1	p<.001
I Unacceptable eccentric habits	3.6	3.1	39.5	4.6	36.4	n.s.
J Self-abusive behavior	1.8	0.9	12.9	3.3	15.0	n.s.
K Hyperactive tendencies	3.0	2.0	33.3	2.4	19.9	p<.01
L Sexually aberrant behavior	2.9	2.6	18.4	3.4	23.7	n.s.
M Psychological disturbances	7.3	7.2	74.1	7.7	67.8	n.s.
N Use of medication	3.0	1.6	74.8	2.6	44.4	p<.001

*These subjects were all over age 19. The number of mentally retarded subjects tested on domain E was 63; the number tested on domain M was 121; these two groups had IQs between 68 and 83.
**The optimal score for each Part II domain is zero.
***By z test of proportions.

but chronic undifferentiated schizophrenic patients were rated worst on inner-directed domains, such as withdrawal, stereotyped behavior, and self-abuse. With the exception of domain I of Part II, organic patients showed little behavioral disturbance. The best scores on 9 Part II items were those of residual schizophrenic patients. Maladaptive behavior was most widespread among chronic undifferentiated schizophrenic patients.

Placement and Duration in Care

The social functioning of patients receiving different levels of care was ranked consistently. The most competent, stable patients were in approved community foster homes, the most unmanageable and disruptive in the hospital, and the incompetent physically infirm in nursing homes.

Performance on the ABS was correlated with length of stay in care. Significant, generally negative associations were found for almost half of the domains, although no direct inferences can be drawn from this finding alone. Patients with the lengthiest hospitalization received significantly worse ratings on all but two Part I domains as well as on withdrawal, stereotyped behavior, unacceptable and eccentric habits, and self-abusive behavior, although they had fewer psychological disturbances and received less medication. Longer community stay was associated with significantly better ratings on 7 of 10 Part I items and with a decrease in withdrawal and unacceptable and eccentric habits.

TABLE 3
Relationship Between ABS Part I and Part II by Psychiatric Diagnosis

Psychiatric Diagnosis	ABS Part I (Daily Living Skills)	ABS Part II (Interpersonal Behavior)
Schizophrenia, residual (*ICDA-8* 295.6)	Good	Good
Neurosis, personality disorder	Good	Fair/poor*
Affective disorder	Good	Fair/poor*
Organic syndrome	Poor	Good
Schizophrenia, differentiated syndrome (*ICDA-8* 295.0–5)	Fair	Fair/poor**
Schizophrenia, chronic undifferentiated (*ICDA-8* 295.9)	Poor	Poor**

*Hostile, aggressive type.
**Withdrawn, bizarre type.

DISCUSSION

The ABS indicates different patterns of social functioning and identifies patient groups with special difficulties. The chronic patients' major deficiencies spanned both parts of the ABS and were essentially those noted by Paul as critical to outcome among chronic patients' "work, social participation . . . bizarre behavior and . . . management problem(s)" (5). The patients' performance on occupational domains was very poor, the majority were seriously socially withdrawn, and, although bizarre behavior was somewhat less common, behavior likely to create severe management problems, e.g., uncooperativeness (domain C), violent, destructive, and antisocial behavior, and various puerile and demanding types of behavior (domain M), was widespread.

However, when individual groups were examined, global uniformly poor functioning was uncommon. The two parts of the ABS appear to tap quite different strengths and weaknesses: on factor analysis (6, 7), two major dimensions of social functioning were found: one, personal independence, related almost exclusively to ABS Part I; the other, social maladaptation, related to Part II. Table 3 uses diagnosis as an illustration to demonstrate that good performance on instrumental role tasks was no criterion of success with interpersonal expressive domains. Both the neurotic and affective patients and the residual schizophrenic patients were rated highly on Part I, but, although the residual patients performed equally well on Part II, the neurotic and affective patients did very badly. Conversely, organic patients displayed marked ineptitude on Part I but were virtually free of serious disturbance on Part II. Alone of the diagnostic groups, the chronic undifferentiated schizophrenic patients appeared to be socially dilapidated chronic patients with unrelieved deficits in all areas. Two main types of maladaptive behavior on Part II were discernible, the aggressive antisocial and the withdrawn-bizarre type.

CONCLUSIONS

Target areas for rehabilitation may be suggested by examining relative levels of social functioning on the two parts of the ABS and the specific areas of greatest deficit. On the basis of ABS findings in conjunction with clinical data, patients may, for example, be selected for milieu or social learning programs with limited, specific goals or placed in patient-run residences where a reasonable mixture of social skills among the deficiencies would be desirable. Extension of the use of the ABS, e.g., to long-term outpatients, would be useful in establishing the limits of social functioning of chronic psychiatric patients.

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Restructuring Rehabilitation for Women: Programs for the Female Drug Addict

BY KATHLEEN M. DOYLE, MARK A. QUINONES, PH.D., M.P.H., GEORGE TRACY, M.A., DARLEEN YOUNG, AND JACQUELINE HUGHES

Two residential therapeutic communities for female addicts—one coeducational and the other all female—encountered serious problems shortly after their formation. The authors found that the male and female staff of the coeducational program had quite different perceptions of the purposes and characteristics of the women's part of the program. In both programs the female staff held such strongly ambivalent feelings toward their roles as women and authority figures that they had difficulty functioning effectively. The authors suggest the need for research that will identify the female addict's special needs, and a restructuring of programs to meet those needs. Further, training programs for female staff must enable them to distinguish between their own ideals and their clients' needs.

HOW, BY WHOM, and in what setting the rehabilitation of the female drug abuser is best accomplished is a subject of increasing concern to clinicians and researchers alike. In the early 1970s studies that focused on drug abusers and drug treatment programs generally reported only the percentage of women in treatment (20% to 25% of the total treatment population) and their retention rates, which differed little from those of their male counterparts (1–4).

Somewhat later studies explored psychological and social variables as they related to female addicts. Miller and associates (5) compared the value patterns of addicts by sex and found that female addicts tend to place a higher value on good interpersonal relationships and feelings of self-respect, while men place more value on qualities of competence and achievement. Richman and Levine (6) studied sex differences in the psychiatric backgrounds of narcotic addicts and found that significantly more women than men reported that they had had a "nervous breakdown" or had felt that one was impending. Both research groups concluded that these distinctions reflected general differences between the sexes rather than any differences specific to the drug experience. Indeed, these findings confirm those of Mellinger (7) and Suffet and Brotman (8), who reported that women use drugs for different social and psychological reasons than do men and exhibit significantly different patterns of drug abuse, more often abusing barbiturates and alcohol.

Other studies explored the types of treatment that women receive in traditional treatment programs. Levy and Doyle (9) examined both a methadone maintenance program and a therapeutic community in Newark and found that therapists regarded female clients as more deviant and "sicker" than the men and thus harder to help. The male clients regarded these women as having it easier both on the street and in treatment and thus being in less need of help. These researchers also found stereotypical treatment of women in terms of low therapeutic, vocational, and educational expectations and services. In the therapeutic community, for example, men, to attain graduation, were expected to have a job, an apartment, and a bank account; women were expected to have a man. Similar differential treatment of women has been documented in other representative drug treatment programs (10, 11).

Faced with evidence that the female addict presented different treatment problems and was apparently receiving inadequate and inferior treatment in existing programs, the National Institute on Drug Abuse (NIDA) and a few individual programs acted to correct the situation. In 1975 NIDA funded five 3-year demonstration programs for women—two methadone maintenance programs aimed at the pregnant addict, two therapeutic communities, and one outpatient drug-free program. These programs have built-in evaluation; they are now at the 3-year point and, with the exception of the methadone maintenance programs, are having great difficulty retaining staff and recruiting and keeping patients. (This material comes from conversations with researchers at the NIDA-funded women's demonstration programs.)

Recent large-scale studies have further confused the issue. Using files from the Client-Oriented Data Acqui-

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sition Process, Rosenthal and associates (12) studied the treatment termination rates of a random sample of 13,258 clients and found that the sex variable had no influence on retention rates. Sells (13), in a study of posttreatment outcome in a nationwide sample of programs, found that demographic variables (of which sex was only one) accounted for only 6% of outcome variance. Quinones and associates (14) explored posttreatment outcome in 6 programs in Newark's multimodality system and found that sex had no influence on outcome (although in the case of methadone maintenance it influenced retention rates in that women remained in treatment significantly longer than men).

The current situation may be summarized as follows: two bodies of literature exist, one indicating that women do as well as men in existing programs, the other that women have needs which are not effectively met in traditional rehabilitation systems. Based on the latter premise, several programs have been initiated specifically to treat the female addict. To date, their retention rates have been low, staff turnover has been high, and posttreatment client outcome studies are far in the future. Because of these difficulties, the existence of programs directed solely at the female addict is in jeopardy.

It would clearly be useful to determine the bases of the problem that these programs are having. Toward this end, the New Jersey Medical School's Division of Drug Abuse of the Department of Preventive Medicine and Community Health has analyzed an individual therapeutic community that has made extensive changes in order to better serve the female addict. Between the fall of 1974, when this program began making changes, and October 1975, its turnover of female staff was 100% and its retention rate for female clients remained the same as it had been before changes were instituted (29% after 6 months). In April 1975 the program opened an all-female facility staffed entirely by women. Six months later it faced a leadership crisis and loss of another 100% of the female staff. The retention rate for female clients remained unchanged. Thus this program faced the same problems as the demonstration programs do, and the analysis of its difficulties can be expected to elucidate the problems that other women's treatment programs are experiencing.

BACKGROUND

Integrity House is a large residential therapeutic community in Newark, N.J., with a capacity of approximately 125 clients and a treatment philosophy based on the Synanon/Daytop model. It was founded in 1968 by the same man who directs it today. In 1974 the division of drug abuse examined attitudes toward women in the program (9). The results of this study, which showed that women received less effective treatment than men, because of both derogatory staff attitudes toward women and differences in actual services, were shared with the program's director, who resolved to change his program. By October 1974 he had hired a female "director" and several female therapists and set them to reforming the treatment received by women.

By January 1975, 10 women held important clinical and/or supervisory positions. Among the innovations they had instituted were weekly female staff meetings and, for female clients, special therapy groups, nonstereotyped vocational counseling, and a program of activities.

As the female therapists exercised more power and increased both their support of each other and their influence on clients, they also began overtly questioning the policies of the male senior staff. At the same time, male senior staff were disturbed by what they saw as lack of progress, too much talking, and too little action. While admitting their sexism, the male senior staff began to make demands that the women saw as harassment, e.g., more paperwork and more evening hours. Female staff meetings were cut back. Some of the women left, angry and frustrated. By the end of August only two of the original group of female clinicians were left, and one of them was job hunting.

Later in 1975 Integrity's male senior staff decided to try again and set about creating an all female house with an all female staff. In April 1976, 18 women (Integrity's entire female population) and 3 staff members moved into the new facility. A new director of the house, an ex-addict graduate of Daytop Village, was appointed on a probationary basis. For the next 6 months she directed treatment activities and experienced increasing difficulty in retaining patients and increasing pressure from the male senior staff to conduct a more traditional program. In October, with the client population down to 7, she resigned rather than conform to mandates from Integrity's director that would have greatly changed the women's program. A month later the program closed altogether; the remaining 4 clients were moved to the coed facility.

From this brief history it might appear that the problems facing this program resulted from the interference and/or prejudice of the male senior staff. However, since other all female programs face identical difficulties, this cannot be the sole explanation.

METHOD

After the first failure, which resulted in the loss of all but 2 female clinicians (phase 1), all staff involved in trying to effect change during that 10-month period (10 men and 10 women) were interviewed by division staff, using a structured open-ended technique. A man interviewed the male staff, including all men in positions of authority and/or influence (the executive director; the directors of research, clinical activities, and social service; the administrator; and individual facility directors). A woman interviewed the female staff, including all of the female clinicians who had worked at the program during this time (the director of the

women's program, 5 counselors, 2 intake social workers, 1 teacher, and 1 nurse). All interviews were recorded and transcribed verbatim for analysis.

Interviews were structured to elicit perceptions in 7 general areas: initial goals and expectations, the authority and status given women (or taken by them), actual changes instituted by women, success or failure of these changes, ways in which difficulties were handled, reasons for eventual dissolution of the experiment in change, and views on how the program might be reformed effectively.

In analyzing the interviews, all statements relating to these areas were extracted, classified by the subject's sex, race, age, and position in the program, and tested for differences by each variable using the Cochran Q test for related samples.

In analyzing the all female facility (phase 2), 2 female research assistants spent an average of 2½ days per week observing therapy groups, staff meetings, individual counseling, and routine daily activities at the program. Each observed interaction was carefully described in writing as to purpose, participants, emotional content, intellectual content, and outcome and was coded for later statistical analysis.

FINDINGS

Analysis of interviews through which reasons for the failure of phase 1 were sought revealed significant differences between perceptions of the male and female staff in 6 of the 7 areas (there were no differences based on staff position, race, or age in either group). The only area in which perceptions were similar was that of goals.

Four findings are most relevant to the present inquiry. The women saw authority as an emanation from the male director that should be given by him and taken back by him, while men saw it as something that an individual can take for himself or herself ($Q=30.9$, $df=7$, $p<.001$). Second, the women felt that the failure of the changes was due solely to the men's prejudice against and fear of women, especially aggressive women, while the men felt the failure was caused in part by their own failure to give the women support ($Q=30.7$, $df=8$, $p<.001$).

Third, disagreement on the efficacy of the actual treatment changes instituted by women stemmed from the women's view that their innovations (e.g., trips and fashion shows) were important and effective clinical tools and the men's view of these activities as frivolous ($Q=51.3$, $df=16$, $p<.001$). Finally, all of the men stated that the successful treatment of women can only be accomplished in a coed setting, while all of the women said that it can only be accomplished in a separate, all female program.

In brief, then, data from phase 1 analysis indicate widely differing perceptions on the parts of directors and the male and female staff of the purposes and parameters of the women's program and, at the end, mutual rancor among the groups.

Analysis of phase 2 revealed substantive ways in which this all female therapeutic community was structured differently from the coed program from which it evolved.

1. The Synanon model, based on behavior modification through rewards and punishments, including harsh encounters and physical "learning experiences" such as haircuts and wearing signs, was rejected. The staff felt that reason rather than fear could best modify female behavior.

2. The staff viewed their clients as women first and addicts second. While this sounds perfectly proper, it led to a great deal of identification of staff with clients as women, to the point where roles became confused and treatment forgotten.

3. Motherhood (all but 2 of the clients had children) was viewed as a responsibility rather than a privilege, as it had been in the coed Integrity House. Thus a woman had the opportunity of having her children visit each weekend from entry into the program onward, rather than having to earn that privilege over a 3- or 4-month period.

4. The staff directed treatment much more toward the development of skills with which to succeed in the outside world (particularly work skills) rather than stressing the development of a feeling of reliance on the "treatment family," as was the standard in the coed facility. As a result, the female clients spent a great deal more time outside the treatment facility than did their male counterparts.

5. In an effort to avoid stereotyping women, the staff did not stress the "feminine" work of cleaning and cooking. As a result, the facility was often messy and/or dirty. But, also as a result, the female clients engaged in a great deal of carpentry and renovation work in the new facility.

After 6 months of operation, this all female program was in a crisis situation; it had lost 75% of its clients, and it closed soon afterward.

DISCUSSION

Juxtaposition of data from phase 1 and 2 analyses indicates that although the outward structures of the 2 phases were quite different, the operating dynamic was similar.

Perceptual data on female clinicians in phase 1 reveal that they held such strongly ambivalent feelings toward their roles as women and as authority figures that they had difficulty functioning effectively. Again, in phase 2, the female staff appeared torn between their roles as women identifying with the troubles of another woman and as treatment agents who must remain aloof in order to be effective therapists.

Data from phase 1 reveal that the female therapists believed that anything women do together as a group is clinically not only good, but highly effective. In phase 2, there was the belief that it is a woman, not an addict

or a personality disorder or a neurosis, that is being treated and that treatment is best accomplished by "togetherness" of the female staff and female clients.

In phase 1 the women's perceptions were characterized by hostility and anger toward the male staff, who were blamed for the failure of the attempt at change. After 6 months of phase 2 the same attitudes prevailed among the female staff because their director was resigning in the face of mandates by the male director of Integrity House.

Thus in both phases the dynamic of treatment moved from an initial determination to treat the female addict in a nonstereotypical manner with special attention to her unique social and psychological needs, to an overidentification of the staff with the clients' needs, to resentment of the confusion resulting from this overidentification.

The dynamic in operation was one in which social services became confused with a social movement. Social movements are characterized by cohesiveness of members, dogmatic beliefs, and merciless, often radical thought and action against an existing system. Social services, on the other hand, are activities that are only properly provided in an atmosphere in which categorization, differentiation, and logical decision making are possible. The goal of social movements is to change an existing system, that of social services, to modify circumstances or behavior within the existing system.

Several investigators (15, 16) have urged that therapists use the women's movement as a resource in the treatment of female clients. But this is quite different from the confusion we observed in the Integrity women's program and in others around the country. The staff at these centers have been influenced by the uniqueness of their programs so much that some unconsciously and others quite consciously (17) have transformed treatment centers into causes. As a result, staff become extra sensitive to criticisms, whether from outside or within, and at the same time expect support in their battles from those clients supposedly undergoing treatment.

If those clients were actually able to become members of this cause, programs would not break down, as is the case currently. It is clear that clients are not able to do so; their presence in rehabilitation programs and their status as recent addicts presenting a myriad of psychiatric problems including low self-esteem, depression, and anomie (5, 6, 9) preclude in ability to view their identify clearly, either as individuals or group members.

In his classic work on social conflict, Coser (18) noted that outside pressure on a group can only produce internal group cohesion and thus positive progress if the group is agreed on its goals. A group composed of female staff who, unconsciously or otherwise, have become fighters for the women's movement and clients who desperately need treatment is not cohesive. Thus when pressure from male directors (as in the case of Integrity House) or from outside funding agencies impinges on such a group, it disintegrates. In the cases under consideration, this has taken the form of client dropouts, staff resignations, and questions of whether programs by women for women can ever work.

CONCLUSIONS

Despite the present discouraging situation of women's programs, we cannot conclude that they should be eliminated. The body of literature which indicates that women have special treatment needs remains. What is needed is 1) research that will not only identify the female addict's special needs but also her strengths, and 2) a restructuring of programs along objective lines designed to meet those needs while using the strengths. Finally, because the evidence is strong that services and causes do not mix, training programs for female staff must be devised to enable these women to distinguish their own ideals from their clients' needs and abilities and to keep the two separate.

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Early Review of Annual Meeting Papers

In order to speed the review and publication process for annual meeting papers, the *Journal* will accept for review *final* versions of papers that are ready before the meeting. Authors must *not* submit their papers before the meeting if they expect any substantive revisions due to discussion at the meeting, further research, etc. The version received for review must be in final form and double-spaced throughout, and it must conform to *Journal* style as described in "Information for Contributors." The paper must be clearly labeled "final version," and the number assigned to it by the Program Committee, as well as the address and phone number of the corresponding author, must appear on the title page. Authors should also submit a cover letter indicating that their manuscript is ready for review and that no major changes are anticipated. These papers will be sent out for review as soon as possible; of course, none can be published before the meeting date.

If you do not use this early submission option but do wish your paper to be considered by the *Journal*, the process will be expedited if you can bring three copies of the final version to Atlanta. However, if you feel that the paper will need revision after the meeting, *do not* submit it to the session secretary.

We hope that these changes will expedite the processing and potential publication of work that is in final form before the meeting.

1977 Anniversaries

BY GEORGE MORA, M.D.

The author recalls this year's anniversaries of events and individuals prominent in the history of medicine, psychiatry, and psychology and examines their practical and theoretical contributions.

1577

Robert Burton was born at Lindley, in Leicestershire, England. He was the fourth of nine children. His mother was described as domineering and unaffectionate toward him, and his childhood was unhappy; this might have been at the root of his depressive character and the feelings of self-depreciation that permeated his entire life. He was graduated from Christ Church in Oxford with a degree of Bachelor of Divinity in 1599 and then worked as vicar of St. Thomas', Oxford, until his death in 1640. He led a very ascetic life, but he resented the celibacy then required of academicians. Typical of his time, he showed traits of misogyny.

In 1621 Burton's *The Anatomy of Melancholy* was published under the pseudonym of Democritus Junior. In spite of its noncommittal and elusive style, the book passed through six editions during the author's life and many more after his death. Burton said of the book, "I have laid myself open (I know it) in this treatise, turned my inside outward." He confessed that he got much of his knowledge from "melancholizing." Part of the book's success was unquestionably due to the extraordinary range it covered: medicine, psychology, philosophy, history, art, theology, folklore, astrology, and much more. It served as a source for many later writers, who freely copied from it until the end of the eighteenth century.

The Anatomy of Melancholy is divided into three main parts. The first deals with the causes, symptoms, and prognosis of melancholy; the second with its cure; and the third with love melancholy and religious melancholy. According to Burton, melancholy, in contrast to sorrow, trouble, fear, grief, and passion, "is an habit, *morbus somaticus*, or *chronicus*, a chronick or continuate disease, a settled humor. So have melancholy men an inward cause, a perpetual fume [cloud] and darkness, causing fear, grief, suspicion, which they carry with them." Characteristic of this condition is excess, i.e., the exaggeration of desires and feelings that are universal to everyone. Among the symptoms of melancholy, Burton mentioned hallucinations, perversion, compulsions, suspicion, jealousy, boredom, moodiness, anger, and a tendency to self-destruction. As for love melancholy, he felt that disturbances in matters of sex were not confined to the sexual union but were extended to the whole process of objectivizing love.

In matters of etiology, Burton stated that melancholy has its origin in the black bile; however, he did not exclude the influence of the stars and the devil. Among other causes of melancholy, he suggested sickness or disturbance of the body, epilepsy, jealousy, idleness, solitude, fears, and poverty. The important cause of melancholy is thus "inward," caused by "irascible" and "concupiscible" inclinations, which in turn might be made worse by three deeper causes, heredity, lack of affection in childhood, and sexual frustration. Of the three, Burton considered heredity the most important.

As for treatment, Burton disregarded bloodletting and a good number of the other major treatments of his time (e.g., witchcraft, charms, amulets, and philters). In particular cases he considered such treatments as purgatives, mild stimulants, narcotics, tobacco, diet, travel, sports (hawking, fowling, and fishing), occupation (but not assiduous study), music, merry company, and sexual gratification helpful. Because the melancholic person is not aware of the distortions of his mind, he must use another person as an external fixed point of reference. This person should have a certain knowledge of medicine, should carry a certain authority, and should be free from passions and perturbations in his own mind. The physician's influence over the patient—the "notable secret" of the successes obtained in many cases of melancholy—is paramount, especially if the patient is genuinely interested in regaining health.

Burton was quite aware of the fact that melancholy is often a symptom of the sickness of society: "The greatest enemy of man is man . . . the knaveries, impostures, injuries, and villainies, of men no art can avoid." As a remedy for this state of affairs, he proposed that property be controlled, profits be limited, privileges be abolished, equitable wages be instituted, the poor be given free care, and slums be eliminated. The government of this ideal society should be entrusted to an educated and intelligent group of trained administrators. Moreover, only people of sound mind

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and body should be allowed to marry and have children.

In essence, *The Anatomy of Melancholy* is impressive for several reasons: the carefulness and thoroughness of Burton's unprecedented survey of the literature, accompanied by an array of self-observations; his conception of the state of the human mind as a continuum from normal to abnormal; the central role he assigned to anxiety and to sexual disturbances in psychopathology; his anticipation of the unconscious theory of the mind; his compassionate and enlightened attitude toward the mentally ill; his delineation of the role of the therapist in terms close to contemporary ones; and, finally, his acknowledgment of the relationship between culture and neurosis.

In Basel three volumes by Johann Wier appeared, printed by Wier's friend Oporinus, a famous printer and humanist who in 1543 published Vesalius' *De Humani Corporis Fabrica*. Wier's volumes were *Pseudomonarchia Daemonum (The Pseudomonarchy of the Demons)*; *De Lamiis Liber (A Book About Witches)*, which also included *De Commentitiis Jejuniis (About the Feigned Fast)*; and *De Ira Morbo (On Anger as a Disease)*. By 1577 Wier had already acquired renown for his book *De Praestigiis Daemonum (On the Deceptions of Devils)*, published in 1563.

Johann Wier was born in 1515 in the town of Graeves, which is on the border between the Netherlands and Germany. He was influenced in his early years by Cornelius Agrippa, a physician and student of occult sciences. Wier studied medicine according to the Hippocratic and Galenic traditions, which divided mental symptoms roughly into phrenitis (delirium with fever), melancholia (delirium without fever), and mania (attacks of great agitation). Treatment at that time was essentially based on diet, baths, and cathartics. Mentally ill people were either kept in prisons or dungeons or left to wander on their own. Wier was graduated from the medical school of Orléans, France. For more than 30 years, from 1550 to the end of his life in 1588, he was active as personal physician to Duke William of Jülich, Cleve, and Berg.

In *De Praestigiis Daemonum*, Wier covered the topic of witchcraft from theological, psychological, medical, and legal viewpoints. He warned against the unnecessary belief in the supernatural to explain natural phenomena and bitterly condemned men who practiced occult sciences. He demolished absurd beliefs in the extraordinary powers of alleged witches and rejected the belief in the aphrodisiac effects of some magic philters. He insisted that in supposed cases of witchcraft the best thing was to ask the advice of a reputable physician and obtain a careful examination of all the people involved.

From the rich clinical material scattered throughout the volume, two points stand out clearly: the importance of affects—especially love and fear—in the pathogenesis of mental diseases and Wier's detailed description of the clinical cases of mental conditions from a refined psychopathological perspective. There are descriptions of toxic psychoses, epilepsy, senile psychoses, hysteria, delusions, full-fledged paranoia, depression, manic-depressive psychosis, schizophrenia, and collective psychopathology. In essence, this volume opened new vistas in the fields of psychopathology, psychotherapy, legal psychiatry, and pastoral psychology, which justifies the claim that Wier should be considered the first modern psychiatrist. Unfortunately, he was so far ahead of his time that the importance of his message could not be assessed properly until the recent advent of dynamic psychiatry.

1677

Baruch Spinoza died in The Hague. Spinoza was born in Amsterdam in 1632 to a wealthy Jewish family originally from Portugal. He was brought up in the strict Hebrew tradition but progressively parted from it under the influence of Latin and Renaissance traditions. He chose to live in poverty, working as a lensmaker, first near Leiden and then near The Hague. He refused academic positions in order to maintain his intellectual freedom. His main work, *Ethica Ordine Geometrico Demonstrata*, was published after his death.

Spinoza was obviously influenced by the Renaissance concept of the microcosm of man and the macrocosm of nature. He identified God with nature and has therefore been considered by some as a pantheistic metaphysician, by others as a materialist and atheist. For Spinoza, spirit and matter were the same thing viewed from two different perspectives.

It has been claimed that Spinoza anticipated many basic concepts of contemporary dynamic psychology. One of these is the assumption of a thoroughgoing psychic determinism ("For such emotions, as hate, wrath, envy, etc., considered in themselves follow from the same necessity and ability of nature as other individual things, and therefore they acknowledge certain causes through which they are understood"). Another is the idea of self-preservation ("To act absolutely according to virtue is nothing else in us than to act under the guidance of reason, to live so and to preserve one's being . . . on the basis of seeking what is useful to oneself"). Still another is the idea of libido as universal to human beings ("Pleasure and pain are desire itself, or appetite, in so far as it is increased or diminished by external causes, helped or hindered, that is they are the nature of everyone").

Spinoza also anticipated the concept of the unconscious in the affective life of man ("The human mind does not involve an adequate knowledge of the component parts of the human body; those ideas of modifications are like consequences without premises, that is . . . confused ideas"). Furthermore, he anticipated the idea of ambivalence and the belief that a better adjustment to reality can be achieved through conscious knowledge of our drives and emotions.

1777

Albrecht von Haller died in Bern, where he was born in 1708 to a distinguished family. He was an extremely precocious child, interested in all fields of knowledge. He attended the medical schools of Tübingen and Leiden, where he was greatly influenced by Hermann Boerhaave. In 1728–1729, after a trip to London and Paris, von Haller came back to Switzerland and composed the poem "The Alps," which brought him renown.

After several years of research on botany in Switzerland, von Haller was named professor of anatomy, surgery, and botany at the newly established University of Göttingen in 1736. Between 1743 and 1754 he published *Icones Anatomicae*, an important contribution to the knowledge of the vessels of the human body. In 1739–1744 he wrote *Hermanni Boerhaave Praelectiones Academica*, followed in 1747 by *Primae Lineae Physiologiae*, a basic work on physiology that was used until the end of the eighteenth century.

In the field of psychology von Haller is especially remembered for his famous lessons *De Partibus Corporis Humani Sensibilibus et Irritabilibus* (1758), in which he proposed his well-known differentiation between irritability and sensitivity. He posited that irritability is an irreducible property of the muscles due to their contractibility and elasticity; sensitivity, on the other hand, is a property present only in the living being endowed with a nervous system. Because sensations occur only in the brain, which is the seat of consciousness, sensitivity is a conscious phenomenon. This dichotomy between irritability and sensitivity caused a great deal of controversy that can be followed throughout the literature on the nervous system to the end of the eighteenth century.

1877

In Philadelphia S. Weir Mitchell's *Fat and Blood—and How To Make Them* appeared. Mitchell was born in Philadelphia in 1829 to a distinguished family. He was graduated from Jefferson Medical College at the age of 21. After further study in Paris, which included Bernard's lectures on physiology, Mitchell spent the next 10 years in Philadelphia in general medical practice; he also conducted some clinical research and experiments in toxicology during this time. In 1864, as a result of his experience as a physician during the Civil War, he coauthored *Gunshot Wounds and Other Injuries of Nerves* with G.R. Morehouse and W.K. Keen. This book has remained a classic in the field; it contains clear descriptions of atrophy, nutritional changes, alterations in joints, and, of course, motor and sensory abnormalities.

From 1879 on, Mitchell worked mainly at the Orthopaedic Hospital and Infirmary for Nervous Diseases in Philadelphia. During this period he wrote *Injuries of Nerves and Their Consequences*, a scholarly and detailed monograph dealing with such complex issues as the nutritive changes in denervated extremities, the conduction of pain, and the nature of the nerve impulse.

It was during this same period that Mitchell began to develop the concept of the rest cure for treating neurotic disorders, which were then considered primarily hysterical. In 1871 he wrote *Wear and Tear* expressing his concern for the steady increase in the incidence of nervous diseases, allegedly brought about by the rising number of jobs in the cities, by the adverse climatic conditions in the United States, and by the poor health of the American woman, exacerbated by the emphasis on female education. In 1875 he wrote *Rest in Nervous Disease: Its Use and Abuse*, in which he stated that most of the successes achieved with the rest cure were with women with hysteria, which, rather unsympathetically, he called "the nosological limbo of all unnamed female maladies."

Fat and Blood became very popular soon after its appearance in 1877. The title, recognized as unfortunate, was changed in later editions to *An Essay on the Treatment of Certain Forms of Neurasthenia and Hysteria*. The cases described in it are chiefly of young, thin, sensitive women from well-to-do families who had been treated unsuccessfully by many physicians.

The range of illnesses for which the rest cure was prescribed came to include forms of melancholia as well as hysteria. The five main components of the treatment were seclusion, diet, rest in bed, massage, and electrotherapy. Mitchell was aware of the importance of the individual relationship between patient and physician. As he put it,

> If the physician has the force of character required to secure the confidence and respect of his patients, he has also much more in his power, and should have the tact to seize the proper occasion to direct the thoughts of his patients to the lapse from duties to others, and to the selfishness which a life of invalidism is apt to bring about.

By 1881 Mitchell was the most prominent neurologist in the United States. He received honorary degrees from Harvard University, the University of Bologna, and the University of Edinburgh, and in 1891 he served as president of the Second Congress of American Physicians and Surgeons.

In his 1894 invited address at the 50th annual meeting of the American Medico-Psychological Association (now the American Psychiatric Association), Mitchell compared the limited advances achieved in psychiatry with the progress achieved in medicine and surgery and boldly criticized mental hospital administrators and staff for the lack of scientific research. He advocated an ideal mental hospital that would be architecturally well planned and staffed by plenty of well-trained nurses. The resident physicians of these hospitals should be appointed through competitive examinations and should be able to carry on psychological and neuropathological research projects. Needless to say, Mitchell's comments provoked a strong reaction in many psychiatrists.

Mitchell continued his research by publishing a collection of essays under the title *Doctor and Patient*. In 1897 his masterful *Clinical Lessons on Nervous Diseases* appeared. By that time he had also become known for his extensive literary production. Among his most popular novels were *Hugh Wynne* (1897), which sold 500,000 copies, *In War Time* (1898), *Dr. North and His Friends* (1900), *Circumstance* (1901), *Westways* (1913), and several others. He was elected president of the American Neurological Association in 1909 and continued to be professionally active until his death in 1914 at the age of 84.

Lasègue and Falret's description of folie à deux appeared in the *Annales Médico-Psychologiques*. Charles Lasègue was born in Paris in 1816. He was graduated in medicine there in 1839 and became physician to many hospitals, including the Salpétrière. His name is mostly known for the Lasègue sign in the diagnosis of sciatica. He died in 1883.

Jules-Philippe-Joseph Falret was born in Vanves in 1824. After receiving his medical degree he worked for many years at the Bicêtre (1867–1884) and then was active at the Salpétrière until 1897. He was elected president of the Société Médico-Psychologique in 1872 and of the Congrès International de Médicine Mentale in 1889. He died in 1902.

In their article on folie à deux, on the basis of solid clinical data Lasègue and Falret came to the conclusion that the contagion of insanity does not occur between two people except in very rare circumstances. It might occur when an individual with an active delusion imposes it on a passive individual in such a way that the delusion becomes their common cause. It might also occur in two people who have been very close to each other for a long period of time. A delusion might also be communicated if it has a character of probability. Lasègue and Falret stated that this variety of insanity was more common among women, that heredity can be a predisposing cause, that continuous reinforcement is necessary, that the content of the delusion often represents wishful thinking, that the main therapeutic indication is to separate the two patients, and that, in most cases, the second patient is not so deeply affected as the first.

Carl Westphal presented the classical descriptions of agoraphobia and obsession. Westphal was born in Berlin in 1833. He studied medicine at the universities of Berlin and Heidelberg. In 1858 he became assistant at the famous hospital Charité, where he was influenced by Carl Wilhelm Ideler (1795–1860) and Wilhelm Griesinger (1817–1868). Wesphal was appointed to succeed Griesinger after his death. He thus became the first professor of neuropsychiatry in a Prussian university. Westphal introduced psychiatry as a regular course of a separate specialty in medicine and established a flourishing psychiatric school. Always interested in correlating clinical with anatomical findings, he described the knee phenomenon now known as Westphal's sign. He was an extremely clear and simple lecturer, and he succeeded in isolating clinical entities that served as models for later presentations. He died in 1890.

Pliny Earle's pamphlet *The Curability of Insanity* was published in Boston by the New England Psychological Society. Pliny Earle was born in Leicester, Mass., in 1809 to a Quaker family. He was graduated in medicine from the University of Pennsylvania in 1837. He then spent two years in Europe visiting mental hospitals, notably the York Retreat in England, which was run by the Tuke family, who were Quakers, and the Bicêtre and the Salpétrière in Paris, where he met Esquirol and Leuret.

Back in the United States, Earle served for a time on the staff of the Frankford Retreat, which had been opened in 1817 as Friends' Hospital on the model of the York Retreat in England.

On October 16, 1844, Earle was one of 13 superintendents of mental asylums who gathered in Philadelphia and established the Association of Medical Superintendents of American Institutions for the Insane, which eventually became the American Psychiatric Association. He served as president of this Association in 1884–1885. Also in 1844, Earle was appointed superintendent of the Bloomingdale Asylum in New York, which had been opened in 1821 for rather wealthy patients under the leadership and guidance of Thomas Eddy, a Quaker merchant. Under Earle's vigorous leadership, many improvements were introduced in the system of care at this asylum. At that time he believed that "when the insane are placed under proper curative treatment in the early stages of the disease, from 75 to 90 per cent recover."

In 1864 Earle was appointed superintendent at the State Lunatic Asylum in Northampton, Mass., where he served for more than 20 years. When he took over the asylum, which was originally founded in 1858 for 450 pauper and incurably insane persons, it was in shambles. Earle's outstanding administrative skill, his innovative approach, and his excellent relations with laymen enabled him to introduce a vast program of work for patients within the large context of moral treatment. As a result of this the hospital became solvent.

While at Northampton Earle collected the statistical data on mental patients that resulted in the publication of *The Curability of Insanity* as a pamphlet in 1877 and as a book in 1887. In the book he took strong issue with the myth of curability. Earle's main contentions were that the word "recovered" was misused and that the words "case," "patient," and "person" were not differentially defined in most follow-up studies. In view of the fact that attacks of mental illness followed by hospitalization tended to recur more than once in a person's life, in many instances a patient was considered cured each time he or she was discharged as recovered from the hospital. Earle's findings gave a definite blow to the cult of curability of mental disorders;

they have acquired new importance in the recent controversies on the recovery rate of mental patients.

Karl Abraham was born in Bremen, Germany, to an old and well-established Jewish family. After finishing his medical studies in 1901, he worked for four years at the State Hospital at Dalldorf, near Berlin. In 1906 he was appointed psychiatrist at the famous Burghölzli Hospital in Zurich. There he was influenced to study psychoanalysis by Eugen Bleuler, then director of the hospital and an admirer of Freud, and by C.G. Jung, who was at the time very close to Freud. In November 1907 Abraham first met Freud in Vienna. A friendship developed almost immediately between the two men. In December 1907 Abraham settled in Berlin, becoming the first German physician with a private psychoanalytic practice. In 1910 he founded the Berlin Psychoanalytic Society and Institute.

Abraham's analysands included Karen Horney, Theodore Reik, Helene Deutsch, Edward Glover, James Glover, Sandor Rado, Ernst Simmel, and Melanie Klein. In 1920 Hanns Sachs joined Abraham and, with a few others, including Franz Alexander, founded the Berlin Psychoanalytic Institute. By 1913, Abraham had become a member of the inner circle called the committee of the seven rings (each member had been given a special ring by Freud), composed of Freud, Sandor Ferenczi, Ernest Jones, Hanns Sachs, Otto Rank, and Max Eitingon.

During World War I Abraham worked in a psychiatric unit on the Eastern Front. A man of great personal charm and of little ambition, he remained faithful to Freud even after some of Freud's most outstanding followers, such as Jung, Adler, and Rank, had left him. Abraham died in 1925 after a prolonged illness at the age of 48.

In spite of the brevity of his life, Abraham's contribution to psychoanalysis was outstanding. As one of the earliest psychoanalysts with a background in psychiatry, he was particularly interested in the study of psychoses and their etiology. He considered the loss of a sexual or love object central to psychotic despair. He characterized depression as ambivalence between love and hate and established three levels of development of the libido related to character formation and the different types of mental illness. These are the oral stage, the anal-sadistic stage, and the genital stage. Schizophrenia and manic-depressive psychoses were characterized by a fixation or regression to the oral stage, character disorders to the anal stage, and narcissistic neuroses to the genital stage. Abraham's views of the primal depression of infants as a precursor of later melancholia were further developed by Melanie Klein and René Spitz. Abraham's *Selected Papers* was published in English translation in 1927 (reprinted 1966), and his correspondence with Freud was published in 1966.

Ugo Cerletti was born in a small town about 80 miles from Venice. He studied in Rome and Turin and received further psychiatric training in Heidelberg, Munich, and Paris. In the first three decades of his scientific activity, Cerletti dedicated himself almost entirely to the study of the pathology of the nervous system. In 1925 he was named professor of neuropsychiatry at the newly established university at Bari. Three years later he founded the neuropsychiatric clinic at the University of Genoa, which became one of the best in Italy. In 1935 he became director of the neuropsychiatric clinic in Rome, where he remained until his retirement in 1948.

In the late 1930s Sakel's insulin shock and Meduna's pentylenetetrazol (Cardiazol) shock for the treatment of manic-depressive and schizophrenic conditions received wide recognition. Cerletti, however, was the first to attempt to use electricity to bring about unconsciousness in the patient. He divided the course of events related to his famous discovery into three periods: 1) the preparatory period, dealing with provocation of epileptic fits in dogs; 2) the actual invention of the electric shock, consisting of the extensive research on the effect of electric currents on pigs and the actual application of the first ECT (70 volts for 8.2 seconds) in 1938 to a schizophrenic patient with a machine built by his associate Lucio Bini (1908–1964); and 3) the scientific study of the mechanism of ECT, which was presented by Cerletti and his coworkers in a special 600-page issue of the *Rivista Sperimentale di Freniatria* in 1940. Unfortunately, this study passed largely unnoticed because of World War II.

Cerletti considered the electrically induced epileptic attack as consisting of two stages: the first is a discharge of the autonomic nervous system originating in the hypothalamus and brain stem; the second is a motor reaction and is far less essential to the therapeutic process. He further emphasized the innocuousness of ECT, the need to repeat the treatment in most patients, the positive results obtained—especially in cases of depressive and schizophrenic reactions—and, finally, the practical aspect of its low cost. Other studies dealt with the physiological reactions of the organism to ECT and with the amnesia resulting from it.

ECT continued to gain acceptance all over the world. It was unquestionably the most popular method of shock treatment. In recent years, however, controversies have developed concerning the use of ECT in relation to patients' legal rights and other issues.

Cerletti received wide recognition for his contribution to the treatment of mental illness. In Italy he was named president of the Italian Psychiatric Association. Outside Italy, he received honorary degress from the universities of Paris, Rio de Janeiro, São Paulo, and Montreal. In 1959 and 1961, at the invitation of the American Psychiatric Association, he attended the annual meetings in Philadelphia and Chicago; although he was over 80 years old, those who had the opportunity to meet him could not help but be impressed by his alert and inquisitive mind, by his kind and unassuming attitude, and by his gentlemanly demeanor. Cerletti died in 1963.

BRIEF COMMUNICATIONS

Lithium Treatment of Patients with Affective Illness Associated with Organic Brain Symptoms

BY LAURENS D. YOUNG, M.D., INGA TAYLOR, M.D., AND VALERIE HOLMSTROM, PH.D.

Lithium was used to treat the manic symptoms of affective disorder in three patients with associated organic brain syndromes. In all three cases neuropsychological testing permitted evaluation of the degree of dysfunction during the acute and follow-up periods of treatment. Lithium was effective in controlling the manic symptoms of all three patients, and there was no evidence that it caused further organic deterioration.

THE PURPOSE of this paper is to report in detail the lithium carbonate treatment of three adult patients with manic-depressive illness that occurred in association with organic brain dysfunction. Recent reports (1, 2) have indicated that lithium is a useful and safe treatment for the symptomatic control of hypomania and recurrent depressive symptoms in patients with definite signs of organic brain disorder and neurological disease. To our knowledge this report describes the only three cases of lithium treatment involving organic brain symptoms in which diagnoses of primary affective illness based on generally accepted criteria have been made (3). In addition, longitudinal observations of these cases allow inferences that the effect of lithium on the organic symptoms was partially independent of its effect on mania. In all three cases neuropsychological testing permitted comparison of the degree of dysfunction during the early and later periods of treatment.

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CASE REPORTS

Case 1. The patient was a 46-year-old crane operator. He had been diagnosed as manic-depressive, circular type, 3 years before the present hospitalization and placed on lithium carbonate. At that time he was functioning adequately on his job. He had a lifelong history of "bad temper" spells and heavy drinking. After 2 years of lithium therapy, with some relief of symptoms, the patient was admitted to the hospital for evaluation of headaches. The findings from brain scan, EEG, skull series, spinal tap, and CSF studies were normal. The pneumoencephalogram demonstrated a borderline shift of the septum pellucidum. The patient was diagnosed as having adult onset diabetes mellitus and hyperlipidemia of the Fredrickson type IV. Lithium therapy was continued, and clofibrate (Atromid) and appropriate diet were prescribed for the metabolic diseases.

After 1 year psychiatric referral was made because the patient had begun to complain that his "nerves were shot." His family had noted frequent angry outbursts. At work he felt that his supervisor was "out to get him." He admitted a 6-month history of diminishing ability to concentrate and of memory loss. He had confused the controls of his crane and allowed boxes to drop, causing damage. He was suspicious that others might be sabotaging his work area. Mental status examination revealed speech marked by circumstantiality and perseveration. He could not remember three words after 5 minutes. Findings from neurological examination were unremarkable except for confirmation of the mental status abnormalities described above. The findings from brain scan and EEG were normal. Neuropsychological testing was performed.

The patient's score on the full-scale Wechsler Adult Intelligence Scale (WAIS) was 72. The verbal scale score of 78 and the performance scale score of 67 showed a degree of scatter between subtests that was not quite significant. The digit symbol subtest score of 3 was definitely indicative of visual memory impairment out of proportion to the rest of the patient's testing. This finding was confirmed by the patient's score of 19 on the Graham Kendall Memory-for-Designs Test, a score that is in the critical range for organicity.

After 8 additional months of treatment, lithium was discontinued for a 2-week period on the chance that it might have contributed to the patient's dementia. After a few days his wife reported that he had begun the project of cutting down all of the trees on a large piece of land they owned. He was overzealous in his work to the point of carelessness, and he nearly severed his fingers in an accident. He also frequently drove off in his jeep in the middle of the night, saying that he "could not sleep." A second series of testing was done 2 weeks after lithium was discontinued. Lithium therapy was then reinstituted because it seemed clear that symptomatic control was necessary.

On the Memory-for-Designs Test the patient showed a nonsignificant improvement in score from 19 to 17, still in the critical range for organic dysfunction. His Wechsler Memory Scale IQ of 80 was comparable to his previous WAIS IQ of 72. His score of 6 on subtest VI reflected the weakness with visual reproduction previously noted on the digit symbol tests of the WAIS. Thus a degree of organic dysfunction, particularly in the visual memory area, had persisted over 8 months.

During the next month the patient's poor retention and recall persisted, and his family reported frequent verbal threats and occasional violent outbursts. Phenothiazines were added to his treatment regimen. After an additional 1½ years on this regimen, the patient has not deteriorated further, but he has been unable to work or return to his family for any substantial period of time.

Case 2. This 55-year-old woman, a high school teacher with a master's degree, was first hospitalized because of recent episodes of confusion, agitation, and bizarre behavior. The patient was described by friends as frequently driving her car off alone and unannounced, and picking up hitchhikers indiscriminately. The patient proclaimed that she was the Virgin Mary. She was unable to sit still for more than a few minutes. Word salad, flight of ideas, and loose associations were noted. Her affect seemed incongruously depressive. The findings from physical and neurological examinations were unremarkable.

The patient had a history of recurrent depression going back several years. She had been treated with psychoanalytic psychotherapy for 4 years, with some improvement. She had been treated somewhat successfully with phenothiazines and antidepressants for 3 years, until this hospitalization. In the past the patient had suffered from migraine headaches. She had been treated with methysergide (Sansert) for years until ascites and jaundice developed. Cirrhosis, diagnosed by open liver biopsy, was thought to be related to the methysergide, although the possibility that phenothiazines and antidepressant medication had contributed to the cirrhosis could not be excluded.

Therapy, first with haloperidol (Haldol) and then with thiothixene (Navane), was ineffective because doses high enough to remit symptoms could not be given without causing liver toxicity. The patient continued to have periods of agitation requiring seclusion and mood swings characterized by euphoria alternating with depression, irritability, and delusional religious preoccupations. She was placed on lithium carbonate, and within 5 days therapeutic blood levels were reached, with dramatic clearing of her psychotic symptoms.

With the clearing of the manic symptoms, it was noted that the patient's level of intellectual functioning was not consistent with that of a master's degree-level teacher. The patient's score on the full-scale WAIS was 117—in the bright/normal range. The degree of scatter between subtests, a verbal score of 121 and performance score of 109, was sufficient to suggest organic brain dysfunction. The patient's digit symbol subtest score of 6 was 1.93 standard deviations below her mean for all performance subtests, suggesting impairment in perceptual motor ability. Her score of 6 on the Memory-for-Designs Test was within the borderline range for organic dysfunction. Testing was repeated 2 months after her discharge to evaluate the effects of continued lithium therapy. This testing showed considerable improvement in perceptual motor ability. Her Wechsler Memory Scale IQ of 114 was consistent with her previous full-scale IQ, but her subtest VI score of 8 represented a considerable improvement in visual reproduction ability. Similarly, her score of 1 on the Memory-for-Designs Test represented enough improvement to place her in the normal range.

Later, because of her increasing frequency of headaches, the dose of lithium was decreased. When the patient became more agitated, lithium treatment was reinstituted at the original level. She was referred for biofeedback treatment of her migraine headaches and was followed for up to 1 year, with no return of the manic or depressive symptoms.

Case 3. The patient, a 71-year-old male retired civil engineer, had been scheduled for elective inguinal hernia repair. Transfer to the psychiatry service was arranged before surgery because of the patient's nervousness, agitation, and bizarre behavior. It was noted that the patient's speech rambled and that he paced about the ward in an agitated manner. He was confused about the transfer, insisting that the hospitalization was for surgery. His recent memory was impaired. The history obtained from his wife indicated that he had been hospitalized once years earlier for ECT and 3 years earlier, when he received antidepressant medications. Although in the past he had been told he was hypertensive, he was not hypertensive at this time. Physical examination revealed some weakness of the left hand, a left grasp reflex, and difficulty with tandem walking.

The patient was treated initially with chlorpromazine and brief periods of seclusion. He complained of vertigo, which the seclusion seemed to intensify. He frequently tried to unbutton his shirt, even when he was not wearing a shirt with buttons. He cried out gleefully at others, trying to touch them and their clothing. He usually had a bright and animated expression on his face. He repeated jinglelike phrases over and over again and used odd rhyming words. He began to insist that he immediately have off-ward privileges and made elaborate plans to accomplish his move.

A diagnosis of manic-depressive illness was proposed, and lithium carbonate therapy was started. Because of the patient's age the dosage was raised gradually. In a few days his rate of speech dropped markedly. He became more cooperative. By the time therapeutic serum levels were achieved, his bizarre touching and rhyming behavior had ceased. He continued to show occasional motor hyperactivity, short attention span, and lapses of memory. Although the weakness, grasp reflex, and ataxia noted on admission disappeared, neuropsychological testing and EEG were arranged as soon as the patient could cooperate.

The EEG showed bilateral slow waves over both temporal poles. Neuropsychological testing showed a full-scale WAIS score of 110. Scatter between subtests, a verbal score of 111 and performance score of 106, was not significant. The patient's digit symbol score of 6 was 1.73 standard deviations below the mean of the other performance scale subtests. His score of 10 on the Memory-for-Designs Test was in

the lower end of the borderline range for organic brain dysfunction. Together, these test scores suggested a degree of organic dysfunction during the early stages of lithium therapy.

The patient was later transferred for hernia repair. Before surgery he was taken off lithium and within a few days after the operation developed signs of confusion and hyperactivity. He described a feeling of being trapped in "the belly of a whale." He stayed up at night pacing and kept his family awake with his repetitive phrases. Lithium therapy was reinstituted. His symptoms subsided rapidly, and he was euthymic within a few days. After about 1 year of follow-up in lithium therapy, he has shown no signs of depression or mania. EEG repeated after several months of therapy showed no persistence of the slow waves. The patient was taken off lithium therapy for a 4-week period after 1 year of treatment, and neuropsychological testing was repeated.

At that time he had a score of 9 errors on the Memory-for-Designs Test, indicating an absence of signs of organic dysfunction. His score of 112 on the Wechsler Memory Scale was comparable to the previous testing, and his subtest VI score of 13 was also consistent with earlier results. The patient was taken off lithium after 6 months and, after 1 year, has needed no further treatment.

CONCLUSIONS

Although these 3 patients were similar in that they all had concurrent diagnoses of manic-depressive illness, manic type, and organic brain dysfunction, some differences in their response to treatment deserve comment. Patient 1 had a 2-year-old diagnosis of manic-depressive illness and had been treated with some success with lithium when he developed a slowly progressing insidious dementia. The relationship between the organic brain findings and the mania is unclear, but lithium appeared to ameliorate the manic symptoms and have little effect (positive or negative) on the brain dysfunction. Patient 2 had a history of recurrent depression, and her first acute manic symptoms responded to lithium dramatically. It is not clear whether lithium ameliorated the organic brain dysfunction since the symptoms cleared more slowly, but lithium clearly did not worsen the brain dysfunction.

Patient 3 appeared to develop manic symptoms and brain dysfunction concurrently. Both the mania and organic brain dysfunction cleared relatively rapidly with lithium treatment. There was some evidence in this case that lithium may have been beneficial to the symptoms of mania and to the symptoms of organic brain dysfunction, since both seemed to remit when lithium therapy was reinstituted. In spite of the individual differences, there was no evidence of progression of organic brain dysfunction attributable to lithium.

The evidence presented in these three cases indicates that lithium carbonate in the usual dosage range is effective in the treatment of hypomanic and manic symptoms when a manic-depressive illness is complicated by organic brain disorder. In this respect, our data tend to agree with previous reports (1, 2). However, in contrast to previous reports all 3 patients fulfilled the criteria for primary affective disorder (3, 4). That is, they all had illnesses of longer than 6 months' duration, with affective symptoms. It is not clear whether our group's disorders should be classified as unipolar with organic psychosis or as true bipolar affective disorder. During the period of time that these patients were maintained on lithium they were dramatically free of either manic or depressive symptoms, a response reportedly more characteristic of bipolar than unipolar patients (5).

In all 3 cases, follow-up a year or more later showed that lithium maintenance appeared to provide symptomatic relief without adversely affecting the associated organic brain syndromes. None of these patients showed any progression of organic dysfunction during follow-up. Patient 1's organic brain syndrome remained essentially stable, but lithium appeared to ameliorate his manic-depressive psychosis. Patients 2 and 3 showed improvement in affective symptoms that appeared specifically related to lithium, whereas improvement in their organic brain dysfunction occurred more slowly. There was some evidence from case 3 that lithium might even have had a beneficial effect on the patient's organic dysfunction.

Although it is usually assumed that mania appears early in life, its appearance later in life is probably not rare, and thus its association with brain syndromes may be common. On the other hand, lithium itself has been implicated in subclinical EEG abnormalities (6) and may contribute directly to organic brain syndromes (7). Although one cannot generalize on the basis of evidence from just 3 cases, our data support the safety and effectiveness of lithium for controlling manic or hypomanic symptoms without adversely affecting associated organic brain dysfunction.

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The Treatment of Perceptual Disturbances in Schizophrenia with Naloxone Hydrochloride

BY ALBERT A. KURLAND, M.D., O. LEE MCCABE, PH.D., THOMAS E. HANLON, PH.D., AND DOROTHY SULLIVAN

The authors treated 12 schizophrenic patients who had overt hallucinatory symptoms with intravenously administered naloxone hydrochloride, a narcotic antagonist purported to have antihallucinatory properties. They found no evidence of the effectiveness of naloxone in preventing hallucinations over that of placebo when administered in a randomized, double-blind fashion.

THE SEARCH FOR COMPOUNDS that might alleviate the hallucinatory symptoms of schizophrenia has recently focused on naloxone hydrochloride, a narcotic antagonist used in the treatment of opiate overdose. The use of naloxone as an antihallucinatory agent is an outgrowth of the recent investigational interest in endorphins (endogenous morphinelike substances) as neurotransmitters or neuromodulators (1–4) and represents a specific attempt to test the hypothesis that endorphins might be implicated in much of the aberrant behavior associated with schizophrenia.

Supporting evidence that the endorphins may have significance beyond their obvious implications for pain perception and narcotic addiction is rapidly accumulating. The Swedish investigators Terenius and associates reported preliminary evidence of elevated endorphin levels in the spinal fluid of schizophrenic subjects (5). Bloom and associates found that certain endorphins produce anomolous behavioral effects when injected into the CSF of rats (6). In particular, they found that β-endorphin induces a state of marked and prolonged muscular rigidity resembling catatonic schizophrenia that is reversed within seconds following the injection of naloxone.

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The potential role of the opiate antagonists in the treatment of the perceptual/cognitive disturbances of schizophrenia remains to be elucidated. The rationale for the use of naloxone in counteracting the activity of endogenous opiates is predicated on the fact that because of its high stereospecific affinity for the opiate receptor sites in the CNS, naloxone displaces previously administered (exogenous) opiates from their receptor sites. The probability that such pure antagonists as naloxone and its congener, naltrexone, have relevance in the treatment of schizophreniform behavior is at least partially supported by the observation that the undesirable narcotomimetic and psychotomimetic side effects routinely observed in heroin addicts during the early (induction) phase of treatment with cyclazocine, an agonist-antagonist, are easily reversed with naloxone (7, 8). Of more direct import is the preliminary work of Gunne and associates (9), who administered intravenous naloxone to 6 hallucinating schizophrenic patients in a single-blind pilot study and observed that perceptual disturbances were attenuated or abolished for a period of several hours in 4 of the 6 patients. Moreover, the experimental treatment of all 6 patients was administered without discontinuing ongoing neuroleptic medication.

The above neurochemical evidence that the endorphins may play a significant role in the modulation of normal and abnormal human behavior, combined with reports of the regulatory role narcotic antagonists might serve in the treatment of such disorders, prompted the present pilot investigation of the effects of naloxone on the hallucinatory behavior of schizophrenic patients.

METHOD

Our study sample was composed of 12 chronic schizophrenic patients (7 men and 5 women) in whom perceptual distortions were a paramount feature of the illness. Ranging in age from 25 to 50 (mean=41.5), the subjects were patients of Spring Grove Hospital Center in Baltimore, Md. They had all manifested psychotic symptoms for at least three years. They were free of any serious physical disorders and had no recent history of alcoholism, drug addiction, or drug allergies.

The experimental treatment schedule, implemented in a double-blind randomized design, spanned two consecutive days; two intravenous preparations were

administered each day. Five of the subjects received injections of naloxone[1] on day 1 and injections of placebo on day 2; 7 received placebo injections on day 1 and naloxone injections on day 2. The determination of active drug versus placebo administration days was made by random assignment within sex; each subject served as his or her own control. For each day, the first injection was 1 cc and the second was 3 cc, administered a half-hour apart. For the active preparation, the respective dosages involved were 0.4 mg and 1.2 mg. As in the earlier work of Gunne and associates (9), the prescription of ongoing neuroleptic medication was uninterrupted during the course of experimental treatment.

A rating form was designed to assess pretreatment and posttreatment levels of hallucinatory behavior. Assessments of frequency of hallucinations were based on the personal expression or overt manifestation of distortions in the following perceptual modalities: auditory, visual, tactile, olfactory, gustatory, and somatic/kinesthetic. Our attempt to quantify changes in these modalities, although readily accomplished in some verbal and cooperative patients, had to be pursued by periodic questioning in patients who volunteered little information spontaneously. Despite this difference in responsiveness, we obtained the impression that what was subjectively reported by the patients was fairly reliable.

Before we prescribed the experimental treatment, each patient and his or her representative family member signed a form documenting that the patient's consent to participate in the study was voluntary and informed, in accordance with the basic elements of informed consent specified by the U.S. Department of Health, Education, and Welfare (10).

RESULTS

Along with summary data on selected patient characteristics, table 1 shows the presence or absence of hallucinatory symptoms of any kind before and at the end of both naloxone and placebo treatments. The lack of a differentially positive naloxone response in terms of the presence or absence of symptoms is evident. Of 8 patients who had hallucinatory symptoms before naloxone treatment, only 2 showed a complete disappearance of symptoms following subsequent naloxone injections. Correspondingly, of the 12 patients who had hallucinatory symptoms before placebo administration, 5 showed no symptoms following treatment.

Eight patients manifested hallucinatory symptoms before both naloxone and placebo administrations. The symptoms of 5 of these (patients 1, 8, 9, 11, and 12) persisted under both naloxone and placebo conditions, and those of 2 showed reductions of approximately equal magnitude in both instances. The hallucinatory symptoms of the remaining patient who had hallucinatory symptoms before both treatment conditions (patient 3) showed no change after naloxone administration but disappeared after placebo administration.

Auditory hallucinations were the most prominent of the perceptual disturbances; these were observed in 8 patients before naloxone treatment and in 9 patients before placebo administration. The mean frequency ratings for these patients were 4.1 and 4.2, respectively (4=very often). Following treatment, the respective mean frequency ratings were 3.1 and 3.0 (3=fairly often), indicating some improvement but a lack of differential drug response. Six patients who had auditory hallucinations before both naloxone and placebo treatment sessions responded in a consistent manner on the two occasions; 2 of the 6 patients responded favorably, and the remaining 4 showed no change.

Although they occurred too infrequently for definitive analysis, ratings of the hallucinatory symptoms associated with other perceptual modalities revealed no differential tendencies as far as treatment intervention was concerned. As for side effects, the use of naloxone with acutely disturbed schizophrenic patients concurrently receiving neuroleptic drug treatment (see table 1) resulted in no observable clinical complications.

DISCUSSION

The fact that we failed to confirm the hypothesized antihallucinatory effect of naloxone in this pilot study is subject to several interpretations. Either there is, in fact, no difference between the antihallucinatory effect of naloxone and placebo, or there are alternative explanations for the findings.

Although failure to corroborate the positive findings of Gunne and associates (9) appears most explicable in terms of application of more rigorous testing conditions, especially in view of the fact that in our study higher dosages of naloxone were used, the conflicting results might possibly be due to differences in the types of patients treated in the respective studies. Presumably, phenomenologically similar hallucinatory symptoms can be manifestations of several types of schizophrenic disorders, varying in etiological bases from maladaptive reactions to endogenous conditions. If patients with elevated endorphin levels constitute one segment of a study population, a differential selection of patients with respect to this dimension could lead to inconsistent findings when the treatment in question is specific to elevated endorphin levels.

Our finding that naloxone is ineffective in the treatment of the hallucinatory symptoms of schizophrenic patients underscores the complexity of the problem

[1]The drug used in the study, marketed as Narcan, was formulated in an injectable aqueous solution: each ml contained 0.4 mg of naloxone hydrochloride, 8.6 mg of sodium chloride, 2.0 mg of methylparaben and propylparaben in a ratio of 9:1, and water for injection as needed to 1.0 ml. Identical-appearing formulations except for the absence of naloxone were available for placebo administration.

TABLE 1
Selected Data on Patient Characteristics, Gross Hallucinatory Activity, and Experimental Conditions (N=12)

Patient	Sex	Age	Type of Schizophrenia	Concurrent Medication	Type of Hallucinations Under Experimental Conditions			
					Before Naloxone	After Naloxone	Before Placebo	After Placebo
1	Female	50	Chronic undifferentiated	Mesoridazine, chloral hydrate	Auditory, visual	Auditory, visual	Visual	Visual
2	Female	44	Chronic undifferentiated	Trihexyphenidyl HCl, loxapine succinate, chlorpromazine	None	Tactile	Tactile	None
3	Female	46	Chronic undifferentiated	Mesoridazine, trihexyphenidyl HCl	Auditory, visual	Visual	Visual	None
4	Female	48	Chronic undifferentiated	Chlorpromazine, trihexyphenidyl HCl	None	Visual	Auditory	None
5	Female	31	Paranoid	Trifluoperazine hydrochloride, trihexyphenidyl HCl	Auditory	None	Auditory	None
6	Male	48	Chronic undifferentiated	Haloperidol, benztropine mesylate, diphenhydramine hydrochloride	None	Auditory	Auditory	Auditory
7	Male	47	Chronic undifferentiated	Haloperidol, trihexyphenidyl HCl	None	Auditory	Auditory	Auditory
8	Male	40	Chronic undifferentiated	Chlorpromazine	Auditory	Auditory	Auditory	Auditory
9	Male	31	Paranoid	Haloperidol, phenytoin sodium	Auditory, tactile	Auditory, tactile	Auditory, tactile	Auditory, tactile
10	Male	45	Chronic undifferentiated	Thioridazine, fluphenazine decanoate	Auditory, olfactory	None	Auditory	None
11	Male	43	Chronic undifferentiated	Chlorpromazine	Auditory, olfactory	Auditory, olfactory	Auditory, olfactory	Auditory, olfactory
12	Male	25	Paranoid	Chlorpromazine	Auditory, visual	Auditory, visual	Auditory, visual	Auditory, visual

and the need for additional investigative efforts directed toward a systematic study of the interrelationships among type of psychotic disturbance involved, extent of CSF endorphin elevation, route and dosage of antagonist administration, and specific characteristics of the antagonist employed.

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Increased Sympathetic Nervous System Activity in Alcoholic Patients Treated with Disulfiram

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The authors measured plasma levels of norepinephrine (NE) and dopamine β-hydroxylase (DBH), pulse rates, and blood pressures of 81 hospitalized alcoholic patients. Treatment with 500 mg/day of disulfiram (but not 250 mg/day or placebo) resulted in small but significant increases in plasma NE and in blood pressure. The 500-mg dose did not appreciably inhibit DBH. Patients receiving high doses of disulfiram should have their blood pressure monitored and their dose decreased to 250 mg/day when possible.

DISULFIRAM (ANTABUSE) has been used in the treatment of alcoholism for three decades (1). The drug inhibits liver aldehyde dehydrogenase, which leads to high circulating levels of acetaldehyde when alcohol is ingested and subsequently oxidized to acetaldehyde by alcohol dehydrogenase (2). The resulting "acetaldehyde syndrome" may be related to sympathetic nervous system (SNS) discharge (3) and is characterized by vasodilation, orthostatic hypotension, tachycardia, a throbbing headache, vomiting, and, infrequently, death (4). Disulfiram alone has side effects of fatigue, tremor, reduced sexual potency, headache, and dizziness (5), and these effects can be mediated by the SNS.

Norepinephrine (NE) is the transmitter of the SNS. Dopamine β-hydroxylase (DBH), the enzyme responsible for the formation of NE from dopamine (6), contains copper ions (7) and is inhibited by disulfiram and its metabolite, diethyldithiocarbamate (8). Both compounds chelate copper ions essential to the catalytic activity of DBH. In experimental animals large doses of disulfiram inhibit DBH (9, 10) and reduce tissue levels of NE (11), but it is unlikely that the smaller doses used in patients effectively inhibit NE synthesis since DBH is present in great excess in mammals (12).

DBH is a component of NE storage vesicles and is released along with NE from the adrenal medulla (13) and sympathetic nerve endings (14, 15). The enzyme is present in human plasma (16), but its levels vary widely among individuals and provide a poor index of acute changes in sympathetic neuronal activity (17, 18). Plasma levels of NE appear to provide a more reliable biochemical index of SNS activity (19). Two prior studies investigating the effect of disulfiram on plasma NE levels used the fluorometric methods then available to estimate NE levels (20, 21). Those estimates compare poorly with more recent measures of NE levels in human plasma (19). Accordingly, we have examined SNS activity in alcoholic patients given the commonly prescribed doses of disulfiram as compared with alcoholics given placebo.

METHOD

Eighty-one alcoholic men admitted to the Alcohol Rehabilitation Unit of the National Naval Medical Center, Bethesda, Md., gave their written, informed consent for participation in the study. These patients, who drank excessive amounts of alcohol either habitually or episodically, lived in the hospital during the study and were closely monitored by one of us (L.F.M.) to exclude alcohol consumption. Patients were also excluded from the study if significant secondary medical complications or severe alcohol withdrawal symptoms supervened. The mean (±SE) age of patients included in the study was 33±1 year. After the patients had abstained from alcohol and medications for at least 4 days, sympathetic neuronal response during supine rest and to postural change was examined. Subjects were asked to remain supine and the needle of a "heparin lock" was inserted into an anticubital vein. Pulse rate was monitored by radial palpation at 5-minute intervals and blood pressure was measured by auscultation 15 minutes after venipuncture. When the patient appeared to be relaxed and had a stable pulse rate, but no sooner than 15 minutes after venipuncture, a basal 18-ml blood sample was drawn for analysis of NE and DBH. Pulse rate and blood pressure were measured and a second blood sample was obtained after each patient had stood for exactly 5 min-

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utes. This technique has been shown to give an accurate and reliable evaluation of acute SNS function (19).

Fifty-four patients who fulfilled the above criteria and remained in the hospital were then arbitrarily divided into 3 groups: group 1, 19 patients who were given 500 mg/day of disulfiram orally; group 2, 16 patients who received 250 mg/day in the form of compressed tablets (Antabuse); and group 3, 19 patients who received a placebo. After 3 weeks, responses to postural change were examined again. Eight subjects from group 2 were retested after continuing to take 250 mg/day for a total of 6 weeks. The freshly drawn blood samples were immediately diluted with an anticoagulant, 10% by volume of cold acid-citrate-dextrose solution (ACD), and centrifuged at 4° C; the plasma was then stored at −70° C until it was assayed for NE by the technique of Lake and associates (19). This method employs the radioenzymatic conversion of NE to ^{3}H-epinephrine and has a sensitivity (twice blank) of about 20 pg/ml of plasma.

DBH activity was measured in these plasma samples by the radioenzymatic method of Molinoff and associates (22) using 4 μl of plasma and phenylethylamine as substrate in the presence of 3.5 μM $CuSO_4$ at pH 5.5 in a total volume of 300 μl. DBH activity is expressed in units where 1 unit is equivalent to the formation of 1 nmol of phenylethanolamine from phenylethylamine per milliliter of plasma per hour.

RESULTS

Mean plasma levels (±SE) of NE and DBH for the 81 male patients during the first 4 days after hospitalization and after the withdrawal of alcohol but before the administration of disulfiram are presented in table 1. Levels in plasma of the neurotransmitter, NE, did not differ from 106 healthy control volunteers of the same mean age.

The change in SNS activity pre- to posttreatment with disulfiram for 54 alcoholic patients is presented in table 2. After 19 patients were treated with 500 mg/day of disulfiram for 3 weeks, the plasma levels of NE were higher than those measured before drug treatment, whether subjects were reclining or standing. Both systolic and diastolic blood pressures were elevated significantly above pretreatment values (but not to hypertensive levels) in reclining patients who received the higher dose of disulfiram. There were no significant changes in levels of DBH, in heart rate, or in blood pressure while patients were standing. Hospitalization can lower blood pressure in some hypertensive patients, and the alcoholic patients given placebo tended to have decreases in pulse rate, blood pressure, and NE levels. Patients receiving 500 mg/day of disulfiram, when compared with subjects receiving placebo, had significant increases in NE levels and systolic blood pressure while lying and standing and in pulse rate while standing.

TABLE 1
SNS Activity in 81 Unmedicated Alcoholic Patients

Patient Position	NE (pg/ml)	DBH (units)*	Pulse Rate (beats/minute)	Blood Pressure (torr)
Basal	266±13	519±42	72.2±1.2	124±2 / 79±1
Standing	572±27		92.3±1.6	119±2 / 84±2

*One unit equals 1 nmol of phenylethylamine converted to phenylethanolamine per milliliter per hour.

Patients who received 250 mg/day of disulfiram had no change in plasma NE or in blood pressure after 3 or 6 weeks of drug treatment, and did not differ from those receiving placebo. The only significant change after 3 weeks in patients receiving placebo was a decrease in standing pulse rate.

The fact that the addition of $CuSO_4$ gave a 100% increase in DBH activity in the plasma of patients both on and off disulfiram indicates that the addition of the metal ion did not mask the inhibitory effects of disulfiram on DBH activity. Patients receiving disulfiram did not have altered plasma DBH activity.

DISCUSSION

Administration of either 250 or 500 mg/day of disulfiram does not appear to alter levels of plasma DBH in reclining or standing patients, and no evidence of any significant effect on DBH activity was found. This may be attributed to the relatively small doses of disulfiram used compared with the doses (400 mg/kg) used in experimental animals to inhibit DBH. The observation that patients receiving disulfiram did not have decreased DBH levels does not rule out the possibility of some inhibition of activity, because the assay for DBH activity uses a 1:75 dilution of plasma, which would dilute any disulfiram present. The significant increase in plasma NE levels after patients were treated with 500 mg/day of disulfiram indicates that the overall level of DBH enzymatic conversion of dopamine to NE in patients receiving disulfiram is not impaired.

The higher dose of disulfiram produces a significant increase in plasma levels of NE, whether compared to pretreatment levels of the same patients or to levels after 3 weeks of placebo. This may be the result of disulfiram inhibition of aldehyde dehydrogenase and subsequent accumulation of aldehydes capable of releasing NE from sympathetic neurones (23). Disulfiram also inhibits the Mg^{++}/ATP-dependent uptake of monoamines by chromaffin granules (24), which may result in increased amounts of catecholamine reaching the plasma. The disulfiram-associated increase in plasma NE was accompanied by an increase in the blood pressure of recumbent patients. Because sympa-

TABLE 2
SNS Changes from Pre- to Posttreatment with Disulfiram in 54 Alcoholic Patients

Item	Disulfiram Dose (mg)	NE (pg/ml)		DBH (% change)		Pulse Rate (beats/minute)		Blood Pressure (torr)	
		Basal	Stand	Basal	Stand	Basal	Stand	Basal	Stand
Group 1 (N=19)	500	+107±25[a,b]	+118±53[c,d]	+0±23	+2±27	+2.7±2.1	+1.5±3.0[d]	+6.6±3.0[c,e] / +8.6±3.0	+8.0±4.9[d] / −0.3±3.9
Group 2 (N=16)	250 for 3 weeks	+57±56	−7±87	+2±16	+1±17	+0.4±2.7	+0.0±3.3	−4.8±4.1 / −1.2±2.6	−3.1±4.2 / −1.4±4.7
Group 2 (N=8)	250 for 6 weeks	+17±52	+131±96	−9±18	+10±26	+4.0±5.6	−3.3±4.1	+1.4±6.8 / −5.1±6.5	+6.3±8.9 / −2.4±7.2
Group 3 (N=19)	Placebo	+4±23	−43±41	−13±20	−11±19	−3.4±2.0	−7.2±2.3[a]	−4.0±2.4 / +2.2±2.7	−6.6±3.9 / +1.7±2.9

[a] Different from predisulfiram ($p<.001$, paired t test).
[b] Different from group 3 ($p<.005$, two-tailed t test).
[c] Different from predisulfiram ($p<.05$, paired t test).
[d] Different from group 3 ($p<.05$, two-tailed t test).
[e] Different from group 3 ($p<.01$, two-tailed t test).

thetic nervous system activity beyond basal output is not usually needed to maintain blood pressure in recumbency (25), increased discharge of NE in recumbent subjects should have pressor effects.

The present data extend and substantiate our earlier pilot study (26) by finding an increase in plasma levels of NE after 3 weeks of 500 mg/day of disulfiram. With significantly more subjects in the present study, both basal and standing levels of NE were elevated after 3 weeks. That DBH does not change but blood pressure increases concomitantly with the NE elevation has been brought to light in this extended study.

Although the changes in plasma NE and blood pressure were relatively minor in this group of alcoholic patients, the results suggest that large doses of disulfiram should be used with caution in hypertensive subjects, that blood pressures be monitored in normotensive subjects receiving 500 mg/day of this drug, and that the dose of disulfiram be reduced to 250 mg/day when possible.

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Screening for Alcoholism Among College Students

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When the Michigan Alcoholism Screening Test (MAST) was administered to 245 students on two midwestern college campuses, 29% of the students on the small, private college campus and 19% on the large state university campus scored more than 4 points, indicating possible alcoholism. The authors hope that this study will stimulate further research on alcoholism among college students.

COLLEGE CAMPUSES traditionally are sources of willing subjects for psychological experiments. However, campuses are difficult places in which to study the prevalence of mental disorders. Administrators are frequently frightened by the potential political ramifications of such studies, e.g., a cutting of funds by legislators or donors if the rates of some illnesses are found to be high. Some faculty and students, attracted to arguments that posit mental illness to be a figment of the imagination or the creation of a medical guild conspiracy, are hostile to psychiatric research. Frequently the issue of invasion of privacy is raised. Thus the prevalence of mental disorders among college students has been more a matter of conjecture than study.

Unlike schizophrenia or neurosis, alcoholism is a relatively neutral concept. Even the most hard-nosed scientist or supposedly enlightened skeptic can usually agree, when presented with strong clinical evidence, that a person is an alcoholic. Because alcoholism can be operationally defined, e.g., the criteria of the National Council on Alcoholism, we anticipated cooperation from faculty and students in our study.

We approached several instructors on two different midwestern campuses. One was a small, private, rural liberal arts college; the other was a large state university. We explained our project: the administration of the Michigan Alcoholism Screening Test (MAST) to a sample of students. The instructors agreed, and we visited several classes to explain our project to the students and invite their cooperation. We administered the MAST to several large groups and explained each question. The information was gathered anonymously; i.e., no student signed his or her name to the material.

THE MAST

The MAST is an easy-to-administer, 25-item yes-no instrument. The questions deal with behavior and not with either quantity of alcohol consumed or hypothetical psychological constructs. A score of more than 4 indicates possible alcoholism.

The test was devised by Selzer (1) in 1971. He gave the test in Michigan to five different groups (526 persons) and validated its accuracy by examining records of medical, legal, and social agencies. The majority of those on whom the test was initially validated were middle-aged white men. In 1972 Moore (2) administered the test to 400 general psychiatric inpatients; 30% scored more than 4 points. The correlation between the attending psychiatrists' global ratings and the MAST was 78%. Favazza and Pires (3) administered the MAST to 258 young, active duty Navy enlisted men; 96.7% of those hospitalized on an alcoholism rehabilitation ward scored more than 4 points, as did 30.5% of medical inpatients, 33.2% of orthopedic

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inpatients, and 22.5% of hospital corpsmen. These figures closely coincide with those of the in-depth Department of Defense study which found that 31% (Army) and 22% (Navy) of all enlisted men were heavy or "binge" drinkers (4). In 1975 Selzer and associates (5) gave the MAST as a self-administered questionnaire to four different groups (501 persons) and concluded that "a self-administered MAST questionnaire has substantial reliability and validity with the scores relatively unaffected by age and the denial of socially undesirable characteristics." A recent article on the effectiveness of the MAST when it was given routinely to 252 ambulatory patients attending a medical clinic (6) reported that 42% of all patients scored in the alcoholic range, with a 10% incidence of false-negative MAST scores.

For the purpose of our study, we administered the MAST anonymously to 245 students on the two midwestern college campuses in 1976.

RESULTS

Table 1 lists the results for 169 students in an undergraduate class in child psychology on the large state university campus (campus A). The ages of the students ranged from 17 to 27 years, with a mean of 19.9 and a median of 19.

Table 2 lists the results for 76 students in undergraduate classes in psychology, urban sociology, techniques in counseling, and introduction to social research on a small, rural, private college campus (campus B). The ages of the students ranged from 18 to 35 years, with a mean of 20.6 and a median of 20.

TABLE 1
Distribution of MAST Scores on a Large State University Campus (Campus A)

Scores	Males (N=50)	Females (N=119)	Total (N=169)	
			N	%
0–3	31	87	118	69
4	5	14	19	11
5	2	8	10	5.9
6–10	6	9	15	8.9
11–15	4	1	5	3.0
16–20	0	0	0	0
21–25	2	0	2	1.2
26–30	0	0	0	0

TABLE 2
Distribution of MAST Scores on a Small Rural Private College Campus (Campus B)

Scores	Males (N=38)	Females (N=38)	Total (N=76)	
			N	%
0–3	17	31	48	63.0
4	4	2	6	7.9
5	3	3	6	7.9
6–10	9	1	10	13.0
11–15	2	0	2	2.6
16–20	1	1	2	2.6
21–25	1	0	1	1.3
26–30	1	0	1	1.3

DISCUSSION

In a recent National Institute on Alcohol Abuse and Alcoholism newsletter (January 30, 1976) an article appeared about a 1970–1973 study done by Jessor and Jessor at the University of Colorado. The actual study will not be released by the authors until its publication, but according to the newsletter article the authors determined that by the end of the study more than a third of male students and slightly less than a third of female students could be classified as problem drinkers. A problem drinker was defined as a person who had had at least two drinking-related problems in the previous year. The problems included frequent intoxication (five or more times), social complications such as censure from family and friends, difficulties with school work, trouble with the law, or driving after having had "a good bit" to drink.

The alleged high rate of problem drinking in the Jessors' study is roughly comparable with the rates found in our study, i.e., 29% of the student sample on the private, rural campus and 19% of the student sample on the large state university campus. Our figures cannot be validated because we did not clinically interview our subjects. It is interesting to note that on both campuses health professionals actively involved with students thought our figures either "about right" or "a little low."

Even though the MAST is a screening and not a diagnostic instrument, we decided to reassess our data. Four questions had the highest rates of response:

1. Have you ever awakened the morning after some drinking the night before and found that you could not remember a part of the evening before? (39% campus A, 47% campus B).
2. Does your spouse (or parents) ever worry or complain about your drinking? (16% campus A, 18% campus B).
3. Do you ever feel bad about your drinking? (21% campus A, 29% campus B)
4. Do you ever drink before noon? (21% campus A, 29% campus B).

We then calculated the percentages of students who would still score more than 4 points when these four questions were deleted from the questionnaire singly and in combination (see table 3).

Even with all questions deleted, the percentage of students scoring in the alcoholic range is high (9% campus A, 14% campus B). Each of the four questions contains the word "ever." On a screening instrument "ever" is more clear than "frequently" but may in-

TABLE 3
Alcoholic Scores with Popular Questions Deleted

Questions Deleted	Students Still Scoring More Than 4 Points (%)	
	Campus A	Campus B
1	12	18
1,2	12	16
1,3	11	16
1,4	11	18
1,2,3	11	14
1,3,4	11	16
1,2,3,4	9	14
2	17	26
2,3	15	26
2,4	15	20
2,3,4	12	20
3	17	28
3,4	15	24
4	17	25

flate the final scores. It should be noted, however, that questions 2, 3, and 4 count for only 1 point, and question 1 counts for only 2 points.

Of special interest is the high proportion of female college students in our sample; 70% of our sample on campus A was female, as was 50% on campus B. On campus A 15% of the females and 28% of the males scored in the alcoholic range. On campus B 13% of the females and 47% of the males scored in the alcoholic range. In almost all alcoholism studies the rate for males is higher than that for females. Ours was no exception, although the rate for females in our study was much higher than we would have predicted.

We hope that our study will stimulate further research on alcoholism among college students. The MAST is easy to administer, well accepted by subjects, and appears to be a promising instrument. Its final usefulness will depend on validation studies of multiple populations.

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Informed Consent and Confidentiality: Proposed New Approaches in Illinois

BY RONALD SHLENSKY, M.D., J.D.

The author examines the basic background and definitions of the issues of consent and, more particularly, informed consent to relinquishing the privacy of mental health records. He sets these issues in the context of some of their specific inherent problems and describes possible solutions suggested by the State of Illinois Governor's Commission To Revise the Mental Health Code.

PATIENTS ARE OFTEN ASKED to relinquish their privacy with regard to their medical records. For example, a third-party payer might insist on scrutinizing a hospital record before remitting payment; a patient might be asked by his or her physician to arrange access to another doctor's records; or a health agency might require review of records to provide meaningful service.

Those of us who keep or control medical records have a statutory and civil duty to protect privacy.

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Therefore, we may not give access to these records to anyone without the patient's consent. Consent denotes a concurrence of wills (1). Thus, although every consent involves a submission, a mere submission does not necessarily involve consent. (2). Unfortunately, the current approach to our patients' privacy often results in submission rather than consent when access to medical records is sought. The reason for this injustice is that the concept of informed consent has not caught up to this area of patient services as it has to many others.

BACKGROUND

Volenti non fit injuria is a fundamental principle of the Common Law: to one who is willing no wrong is done. Thus, the very definition of a tort (civil crime) involves the absence of consent. A battery, for example, is the unpermitted touching of another. Permission or consent might be implied: an individual jostled slightly in a crowd may not ordinarily bring suit against the jostler. A patient who has consented to surgery cannot sue his or her surgeon for battery—unless the limits of the consent are exceeded.

Consent also requires some knowledge. Since 1905 the courts have increasingly focused on this matter of knowledge (3), and the concept of informed consent has thus evolved. A surgeon who operates without having explained the risks may be found negligent. At this point I would ask, If one consents to allowing access to one's medical records without having knowledge of the content of those records, is such consent informed?

PROBLEMS

The valid giving of consent requires "sufficient mentality to make an intelligent choice to do something proposed by another" (4). Thus, capacity is a consideration. The demented, the severely psychotic, the mentally retarded, and perhaps even the simply overwhelmed are at once protected and limited by the law. They cannot be informed because of their impairment; therefore, they cannot give a valid consent. The test formula generally applied is "reasonableness": minor dementia such as a mild organic brain syndrome will not operate to invalidate consent as long as there is a reasonable awareness of the issues, consequences, and risks. When a conservator has been appointed, his or her consent is required.

Conservatorship or guardianship (in Illinois the latter term is applied with regard to children), incidentally, suspends an adult's powers to create legal relations with others. Once found incompetent, an individual may not direct the disposal or use of his or her property, enter valid contracts, marry, change domiciles, or choose agents, such as doctors, lawyers, or guardian. The legally incompetent individual will probably be unable to make a valid will and may be denied the right to vote. Often some slight mental deterioration can lead to this state of affairs. Despite the severe incursions on freedom this legal process entails, it is nonadversary in character. This process is justified by its proponents as favoring family interests above those of the individual (5). Of course, once a conservator has been appointed, informed consent must be obtained from that party.

The question of how much to inform is a vexing one for physicians. On the one hand it may be negligent or at least cruel to frighten a patient away from treatment. On the other hand, if the patient develops a complication that was not specifically mentioned, there is the possibility of a malpractice suit. Further complicating this question are numerous studies demonstrating that patients informed by their doctors of potential complications recall only 10% of what they have been told (6). Again, "reasonableness" seems to be the test.

With regard to disclosure of medical records, there seems to be little reason for giving anything less than full information to the patient. However, when a physician believes that information might be harmful to a patient, the best approach would be to so inform the patient or family but to grant access if there is insistence.

Minors present special problems (7). The law has carved out protective disabilities that prevent minors from consenting to medical treatment, except under special circumstances. Illinois allows treatment for venereal disease and drug-related problems without parental consent as early as age 12 (8). We must recognize that parents and offspring might actually be adversaries. A teenager obtaining psychotherapy might have good reason to object to parental access to his or her records. According to a decision in Massachusetts, "The limits on the legal capacity of minors to act on their own behalf were not established to defeat the enforcement of their rights" (9). In some instances the courts have found that a minor approaching majority may give a valid consent if he or she fully appreciates the nature and consequences of his or her acts (10).

SOLUTIONS

With regard to mental health records, the State of Illinois is acting to resolve some of the dilemmas raised here. The Governor's Commission To Revise the Illinois Mental Health Code has drafted legislation that directly addresses the problems of confidentiality of mental health records. The proposed solutions to these problems include the following:

1. Privacy protection is afforded to a client of anyone not prohibited by law from representing himself or herself as a therapist. Currently, inconsistent distinctions are made among the various professions; in some instances there is no statutory privacy (11).
2. A person may see his or her own record. Of

course, this is already the case for mental health records in Illinois and, for all practical purposes, for health records in general.

The person must be 12 years old; up to age 18 the parents may also have access. A parent of a patient between 12 and 18 years old may not have access if the patient objects or if the therapist believes there are compelling reasons to deny parental access. In such a case, parents may petition a court for access.

If a patient disputes information in the record, a written statement by the patient may be entered into the record. The patient may request that any part of the record believed to be misleading or incorrect be modified; a court may compel such modification if justified.

3. No disclosure of the record to others may be made in the absence of informed consent.

This consent may be given by any person 18 years old or older, by the parent or guardian of a person under age 12, and by both the patient and parent or guardian of patients between ages 12 and 18. If a therapist believes disclosure of the record of a person between 12 and 18 years old is in the person's best interest and the parent or guardian consents, disclosure may proceed in the absence of the person's consent.

The consent must be in writing and specific as to who receives the information, the purpose of the disclosure, the nature of the information, and the consequences of refusal to consent. Blanket consents and redisclosures are prohibited. Provision is made for limited disclosure without consent when it is impossible to obtain consent due to incapacity and necessary to obtain it to receive benefits.

A therapist may disclose information in the absence of consent for purposes of supervision, record keeping, peer review, and to an attorney consulted regarding legal rights and duties owed the patient.

In any civil, criminal, administrative, or legislative proceeding, a patient may invoke privilege and refuse or prevent disclosure of records or communications except in the following instances: 1) when the patient introduces his or her mental condition as an issue, 2) when the mental examination has been ordered by the court, 3) in a proceeding brought by the patient against the therapist, 4) if the therapist determines that involuntary hospitalization is necessary to prevent physical harm, 5) when facilitation of guardianship is necessary, 6) when the communications were made in the course of treatment ordered to render the patient fit to stand trial and only with respect to that issue, and 7) when required by the Abused and Neglected Child Reporting Act.

In addition, nothing in the proposed legislation may preclude a therapist from making such limited disclosure as he or she deems essential to prevent imminent, serious physical harm. Caveat: one must have capacity to validly consent.

Essentially, the drafters of this proposed legislation felt that one's record is an extension of one's self and thus deserves protection and consideration. No doubt, the conveyance of private facts to others can exercise a profound influence on the person exposed. It should be noted that Illinois will probably also have a more sophisticated approach to the problem of conservatorship. A plan is under way to implement an approach based on partial incompetency, with limits on the loss of freedoms.

CONCLUSIONS

As individual rights receive more attention, privacy deserves greater protection. To give privacy up is a serious matter. An individual must know full well what he or she is doing when he or she acts. Both common and statutory laws support this trend.

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On Teaching Psychotherapy in a Community Mental Health Center

BY JEFFREY D. NASON, M.D.

The author discusses some of the unique problems related to teaching psychotherapy in a community mental health center. These problems arise principally from the diversity of philosophies and backgrounds of the health center staff and the lack of an overriding institutional tradition of psychotherapy. They often result in increased anxiety among trainees and require special attention from the teacher of psychotherapy in this setting.

THE CURRENT PROLIFERATION of community mental health centers and their increasing integration into mental health training programs brings into focus some difficult and intriguing problems in the teaching of psychotherapy. There are obvious administrative problems involved in the integration of community mental health center staff and trainees and in the balancing of responsibilities for service and training; these will not be the concern of this paper. What I wish to consider are some of the theoretical and practical problems that arise from trying to teach psychotherapy in the special circumstances of the contemporary community mental health center.

THE PROBLEM OF DIVERSITY

Psychotherapy has evolved rapidly and tumultuously in both concept and practice. We are all aware of the profound chaos and confusion of contemporary psychotherapeutic thought. Leston Havens brilliantly outlined the development of the major psychiatric schools in this century (1). In tracing the flow of changing psychotherapeutic assumptions, he showed how each assumption has become the basis of a qualitative change in psychotherapeutic practice. As Havens asserted, these changes are often unacknowledged, so that what we do becomes increasingly remote from what we say we do. Psychoanalysts practice existential therapy while talking hydraulic theory, and Sullivanians (and perhaps all of us) unconsciously become experts at behavior modification. Not only are these diverse assumptions unacknowledged, Havens argued, but they are often not understood, so that thoughtful students are confused and others become mindlessly eclectic or religiously sectarian. This situation recalls Yeats' fear that "The best lack all conviction, while the worst/Are full of passionate intensity" (2).

Granting that there are profound theoretical confusions in all of our schools, academies, and institutes of training in psychotherapy, we must still consider the special problems that have come with the emergence of the community mental health center as a locus of training. The working staff of the community mental health center—the team (including trainees)—tends to be at once both smaller and therefore more tightly integrated and substantially more heterogeneous in background, training, and therapeutic philosophy than the staff in larger, more traditional training institutions. In the traditional teaching hospital or private clinic, trainees and staff can be selected according to how they fit into the spirit or style of the particular institution. The resulting mutual intellectual support and validation, the pressures toward orthodoxy, or the presence of inspirational teachers can protect and nourish an institutional tradition involving a particular approach to psychotherapy.

The community mental health center, according to my experience, tends to attract a diverse group of therapists and would-be therapists from all parts of the intellectual, temperamental, and philosophical spectrum. There has been a substantial and, in terms of the acquisition of psychotherapeutic skills, often very useful blurring of professional disciplinary roles, but I do not think that this is the major cause of the kind of diversity with which I am concerned here. I think that the pressures on the community mental health center to prove itself, to justify budgetary allotments by demonstrating that its service, namely psychotherapy, really works, have created a climate that has encouraged diversity by fostering a pragmatic approach to symptom distress. The motto might be, "If it works, do it," rather than the more traditional, "We do it because it's the right way." As fruitful as such diversity and pragmatism have been, however, the price that is paid is often the loss of a traditional and institutional consensus on the "right" approach to a particular clinical problem. The need for clinical judgment and the chance of creative solutions are perhaps greatest in this situation, but it is also an anxiety-provoking situation for the trainee and the experienced clinician alike.

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Unfortunately, as a result of this anxiety therapists sometimes become paradoxically more rigid, positions become fixed, and diversity becomes factionalism rather than encouraging open-mindedness.

COMPETING CLINICAL OPTIONS

One of the most exciting developments of the last 20 years has been the burgeoning of interest in family therapy. This area provides an example of the competing clinical options evident in the community mental health center. Because of the new perspective of family therapy, traditional approaches to common problems must be rethought. For example, play therapy for the child and a "child guidance" approach for his or her parents (usually the mother) when a child is symptomatic or troublesome to others or individual therapy for an adult whose problems have to do with difficulty in separating from his or her original family or in living with his or her current one have been standard approaches. These must now vie for acceptance with the newer modes of family therapy.

This new perspective can and often does add a dimension to our clinical vision that allows us to choose our therapeutic approach more thoughtfully. However, the conceptual framework for much current family therapy is quite different from that of psychoanalytically or psychodynamically derived individual therapeutic models. The concept of an interactional system and the necessity of placing individual symptoms and psychopathology within a supra-individual matrix of regulation and meaning are essentially new ideas. They can be extremely useful if they help us view our patients more holistically and if they provide another option for therapeutic approach. However, as a competing therapy, often based on very different assumptions as to what is wrong as well as how to make things right, family therapy can add to the already abundant uncertainties and anxiety of the experienced clinician and, more particularly, of the trainee.

A common clinical vignette may illustrate this point. A young woman in her 20s and working comes to the clinic complaining of depression or discouragement because she has been unsuccessful in two attempts to leave home. Each time, after taking an apartment with some friends, she has become acutely anxious and has quickly returned home to a mother to whom she has a close but chronically hostile attachment, an alcoholic father, and a younger brother who has had intermittent psychotic episodes requiring hospitalization. The family therapist, with undeniable justification, says, "It's a family problem. They cannot separate from one another. Have them all come in and we will work with them together." The individual therapist says, "But she's an adult and her problems are internalized. We should work with her alone to help her to resolve her conflicts and go on with her life. Bringing in her family might just make things worse."

To examine the clinical considerations in this question is not the intent of this paper, but I think the example evokes a sense of what the trainee must feel when confronted with such profound differences in clinical judgment and in assumptions about the nature of what is therapeutic among his or her teachers. Because the community mental health center, unlike the traditional training institution, usually has no unifying tradition of psychotherapy, the trainee often feels buffeted by the rapidly shifting winds of therapeutic fashion. On the other hand, trainees might become disciples of a charismatic teacher or one available by the accident of proximity.

THE CLIMATE OF OPENNESS

The many problems of teaching psychotherapy in the situation outlined above are obvious. One task of enormous importance is to help the student remain a student and not become a disciple. To help students develop their own vision of human nature and of the experience of psychotherapeutic change means to a large extent sharing our vision, complete with distortions and biases, with them—to let them see through our eyes. This must be done in a way that leaves them free to be open to their own experience, to view all theory thoughtfully and critically, and to learn from others who see things very differently from the way we do. These problems, which are universal in teaching, are particularly acute in the community mental health center because of the special anxiety the trainee often feels from the lack of a unifying philosophy of what is to be his or her life's work. Here the task of the teacher is to help the trainee tolerate these anxieties without premature closure of emotional and intellectual growth.

Karl Mannheim, in his appeal for a "sociology of knowledge" (3), asked that we attend to the social and institutional climate that permits or inhibits certain kinds of human awareness and understanding—that allows us some perceptions of human experience and denies us others. The community mental health center, itself a product of rapid social and institutional change, presents special problems in maintaining the quality of that climate.

SPECIAL PROBLEMS OF TEACHING

As clinical director of a community mental health clinic with an active multidisciplinary training program and a highly capable but relatively heterogeneous staff, I have found three problems of teaching to be particularly vexing and challenging. The first is that of language. We are a veritable tower of Babel. The staff talk many different conceptual languages; the task of the teacher is often to translate these. There are currently several different systematized languages in psychotherapy, ranging from psychoanalytic theory to behavior modification theory to the various family the-

ories. In many ways the biggest problem results from the hybridization (or bastardization) of theoretical terminology: we sometimes use the same words and phrases but do not always mean the same things by them. For example, it has lately become very fashionable for therapists of all schools to talk about a loss of ego boundaries. This phrase may communicate very different things to a psychoanalytic therapist, an interactionally oriented therapist, and a structural family therapist.

Obviously an attempt to minimize the use of jargon is in order, but I do not believe that all jargon can be eliminated without a concomitant loss of the sophistication of certain ideas and observations. There is often no better way to say something than the way we are used to saying it, and I think it is preferable to let people talk in their own way if they exert the intellectual discipline to be terminologically consistent and to help others understand what they have said.

The second problem is that of clinical evaluation. With staff and students from so many different backgrounds and orientations, it is perhaps not simplistic to ask what an evaluation is or why it is necessary. Listening to evaluations presented by workers with different orientations brings these questions to mind, and one must also ask what, if anything, these evaluations have in common. To refer again to the example of the young woman unable to leave her home, the family-oriented therapist might elaborate for us, in diagrammatic form, the assigned roles, alliances and boundaries, and power relationships within the family that have kept the patient in place. The psychoanalytically oriented therapist will be dissatisfied with such an evaluation because it does not tell us enough about the conflicts raging within the patient and particularly because it tells us so little of the developmental aspects of the patient's conflicts and of her resultant ego strengths and deficits. The empathically oriented, perhaps existential therapist will be dissatisfied with this account because it does not give us a sufficient sense of the patient's felt experience and the nature of her pain. Finally, the medically oriented therapist will be concerned that insufficient attention has been paid to the family history of mental illness and to the patient's current mental status to enable us to decide about diagnosis, prognosis, or the preferred mode of treatment.

Should we try to standardize a process so subtle and so complex as a clinical evaluation? Granting the creativity inherent in the individual viewpoints of different clinicians, are there not some things that we would want to know about everyone who is seen at our clinic? With these questions in mind I constructed an outline for clinical evaluations intended primarily to call attention to some of the critical areas that should be considered in every evaluation, regardless of the orientation or principal interest of the evaluator. The language selected was deliberately simple and nontechnical. It was structured around four questions: 1) Why is the patient here now? (assessment of the current situation and not just the chief complaint), 2) Who is the patient? (including relevant history and descriptive mental status), 3) How do we understand the patient and his or her current problem? (a conceptual question inviting divergent conceptual styles), and 4) What can we do for the patient? (a treatment plan). I think that these questions, which can be greatly elaborated and obviously must be modified for evaluations of families, point to simple but universally relevant things that we need to try to learn about new patients.

Finally, the most subtle and difficult area of teaching in a community mental health center involves the introduction of the trainee to the experience of psychotherapy itself. Here I must reveal my own assumption that the process of psychotherapy is primarily an affective experience involving two or more people. It is my assumption that the process of becoming a psychotherapist involves a growing knowledge and tolerance of one's own most painful feelings and a growing ability to make active and accurate empathic contact with the inner life of another. Teaching psychotherapy must therefore be largely by example, and I believe that a good deal of our success rests on the relationship between the supervisor and the trainee. I do not mean that we are to be therapists for our trainees but that empathic and sensitive attention to the experience of the trainee in learning to become a therapist in this particular time and place is essential to the success of our work.

CONCLUSIONS

The community mental health center, which grew out of the need for a greater and more equitable distribution of mental health care, has increasingly become a locus of training in psychotherapy. Because of the diversity and heterogeneity of its staff and its lack of an institutional tradition of psychotherapy, the normal anxiety of trainees learning to become psychotherapists is often greatly increased, presenting special problems for the teaching of psychotherapy. It is in this setting that much of the growth of young therapists as well as the future course of psychotherapeutic theory and practice may well be determined.

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Mental Health Consultation in a Law School Clinic

BY ROY B. LACOURSIERE, M.D.

The author describes mental health consultation with legal interns in a law school legal clinic. Unique characteristics of this setting are described, including that it is not primarily a mental health area and that the consultees are trainees. Various types of consultee-centered consultation and direct and indirect client-centered consultation are discussed, as are problems that must be avoided, e.g., an excessive psychological focus on the part of the consultant or consultee.

IN THE lively community mental health movement, consultation is a basic and mandated activity. We have learned a considerable amount about the application of consultation skills with such groups as teachers, public health nurses, psychiatric paraprofessionals, and police officers. This paper describes consultation to legal interns and some of the unique aspects of this work.

Mental health consultants working with legal interns, as herein described, function primarily by helping the interns learn how to better represent their clients rather than by teaching them to enhance the clients' mental health. Good legal representation can make a significant contribution to a client's mental health—for example, when an attorney functioning as a child's guardian *ad litem* does what is best for the child rather than what might be most legally expedient or lucrative. A client who is under the stress of legal difficulties can be helped by being carefully listened and attended to. Although this is not the typical use of mental health consultation, it falls within its broad definition (1).

DESCRIPTION OF THE PROGRAM

As part of the Washburn University of Topeka School of Law's clinical legal education program, two elective semesters are offered in the in-house law office, the Washburn Legal Clinic. Students can take one semester in the junior year as law clerks and a second semester in the senior year as legal interns. The clients are represented by the legal interns, with the assistance of law clerks, all under faculty-attorney supervision. The clinic staff represents about 1,400 indigent clients each year in cases that include driving while intoxicated, divorce, child custody, commitment for treatment of mental illness or alcoholism, juvenile delinquency, and misdemeanor proceedings.

There are several requirements for a mental health consultant in such a clinic. Adequate skill in consultation is the first. Familiarity with local mental health, medical, and legal facilities is necessary to understand various aspects of the cases and to help in appropriate referrals for treatment, evaluation, etc. (The consultants also work in other settings with patients who could qualify as legal clients for the clinic on both financial and legal grounds; and patient-clients are shared occasionally.) Also required is an ability to understand and appreciate the orientation of attorneys, which stresses advocacy and legal rights. This understanding can be facilitated by reading, taking law school courses, doing forensic and civil commitment evaluations, etc. Furthermore, the consultant needs to be comfortable in his or her own profession. For example, one's professional allegiance is challenged when colleagues are on the opposing side of legal cases, as in child abuse cases in which the child has been seen by a fellow mental health professional and the alleged abusive parent is the legal clinic's client.

There are other characteristics of this consultation work that are related to the fact that consultees are not yet skilled in their own field. Although this makes them more unsure about their own roles as attorneys and in some ways hesitant to reveal their uncertainty to a consultant, their clearly defined learning role as interns can also make them more amenable to new input. Also, there are some cases in which a law or legal procedure may be more familiar to the consultant than to the intern, particularly those related to mental illness. Such bits of legal information can be conveyed unassumingly by saying something like "Let's look at the statute to be sure."

The Consultations

For purposes of presentation, the various types of consultation in the legal clinic can be arranged to follow Caplan's format (2, 3), with recognition that some of the consultation is "medical" as opposed to "mental health." Consultation methods are eloquently dis-

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The author thanks the law school faculty, Dean Raymond Spring, Prof. Donald Rowland (Clinical Director), and Prof. Donald Frey for their help and encouragement, and the Council on Legal Education for Professional Responsibility, New York, N.Y., which partially funded the mental health consultants (Drs. Frey and Lacoursiere).

cussed in Caplan's work (2, 3) and in briefer format elsewhere (4). Consultation requires, among other things, listening at several levels to the verbal and nonverbal messages of the consultee and responding to the appropriate level or levels. The consultations themselves are usually short and "one-shot," but occasionally they have been fairly long and have extended over a few contacts. The important point in this regard is adaptation to the case and consultee as required. An attempt is made to follow the cases with the interns after the initial consultation. This allows for consolidation of the consultation and a check on its usefulness.

The most frequent type of consultation has been consultee-centered case consultation, in which the consultant meets with the intern and occasionally also the supervisor, but not with the client. The main purpose of such consultation is to help the consultees improve their functioning as attorneys-to-be and only indirectly to help the client. In spite of this, the main consultation work is done by inquiries and listening directed toward the client but always with the thought of how to help the intern with the difficulties he or she is having with the client. There is some arbitrariness in determining the subtypes of this category, one of which results from lack of understanding on the part of the consultee; e.g., difficulty in understanding the significance of certain behaviors he or she has observed. An example that is seen often is the initial angry client who wants a divorce but is slow to follow through or misses appointments as the intern prepares the case. Interns may need some help to see that in such a case a divorce may no longer be desired and that marriage counseling might be suggested.

Another subtype involves the need for consultation because of the consultee's lack of technical skill or knowledge. Once the interns have determined that psychiatric evaluation or treatment would be beneficial for the client and/or the cases, they may need assistance in deciding how and where to refer the client.

Some consultations come about because of the intern's lack of self-confidence. In such cases the intern has sufficient understanding and skill but is hesitant, often because of inexperience, to do what he or she sees as necessary. For example, the intern may lack the self-confidence to confront or ask for an explanation from a client who gives information that is inconsistent with what has been found from other sources, such as court records of previous convictions, or to recommend help for a client charged for the third time with driving while intoxicated.

In some cases the consultation focuses on the intern's lack of objectivity in the case, which may result from excessive "personal involvement" or irrational bias toward certain types of people or behavior. Technically, this is theme interference, wherein a personal bias or theme interferes with the application of one's skills, e.g., "Clients wouldn't ignore your advice if they had to pay," or "These kinds of blacks are always in trouble." This is probably the most difficult type of consultation. Such lack of objectivity may be unconscious, or if there is some awareness of a bias toward that particular aspect of the case, the intern sees it as "reasonable." In the mental health field, consultation on such cases may take a few 30- or 45-minute sessions; however, they can sometimes be done in shorter clinic sessions with only one or two meetings (5), and the law school clinic setting often makes this mandatory. In dealing with theme interference, the intern's lack of objectivity or bias is rarely confronted directly, since confrontation would usually merely make him or her defensive. Rather, the client is focused on in such a way as to try to bring about basic changes in the orientation of the intern, e.g., by asking what this black client was doing before the current legal difficulties, how other members of his family are functioning, and perhaps questions about the previous defense if there were earlier legal difficulties.

In some consultations the main focus is on the client and various aspects of the case; helping the consultee develop as an attorney is a secondary focus. Such consultations are called client-centered, and can be either indirect or direct. In indirect client-centered consultations various aspects of the case file are reviewed and discussed with the intern. Occasionally such consultations involve medical rather than mental health concerns; e.g., are certain medical reports adequate grounds for a Social Security disability claim?

In a very few cases, the client is seen with the intern (direct client-centered consultation) after the case file has been reviewed. This is particularly likely when psychological reports or recommendations have seemed inadequate or contrary to the intern's expectations or the intern needs help to decide if and where a particular client should be referred for psychiatric evaluation or treatment or what other course should be pursued. There have been several requests to see clients with whom interns said they had trouble communicating. Usually, with encouragement to help their lack of self-confidence, the interns can manage these situations quite well, and it is rarely necessary for the consultant to see the client.

The client contacts are not included in formal reports to be used in the case. The consultant is not in a position to submit neutral reports because he is an employee of the law school clinic. When such reports are required, they are obtained from the usual mental health or medical practitioners in the community. Also, having the consultant see clients directly can undercut the more difficult and more useful task of using consultation as much as possible to promote the professional development of the intern. The more the consultant performs tasks that the intern can and usually must learn to do, the less the intern's professional growth is encouraged.

The consultants are also involved at times in various discussions not covered by the above categories, both with the clinic administrative and supervising attorneys and with interns. Such discussions have covered many areas, including the morale of the intern

group and the mixed acceptance of the clinic program by some of the nonclinical law faculty and students. Although these miscellaneous areas are a small part of our consultation work, they can be technically described as program-centered administrative consultation and consultee-centered administrative consultation (2).

DISCUSSION

Mental health consultation in the legal clinic does not aim to help the consultant do mental health work with legal clients through the interns, nor does it turn the interns into mental health counselors. In fact, there are certain conflicts between an intern's "counselor" and "advocacy" roles, particularly in civil commitment procedures. There seem to be some interns who function as counselors (therapists) and others as advocates, sometimes almost in spite of their client's best interests. Some of these advocates may be hesitant to consult on cases with strong psychological components for fear of losing a client or case to the mental health (counseling) field (e.g., a divorce case to a marriage counselor). A third group of interns can more comfortably and appropriately shift between the counselor and advocacy roles as the client and case demand.

The consultant particularly needs to watch the mental health/legal boundaries when the clients have glaring and distressing psychiatric problems. For the consultant in such cases (e.g., disturbed juveniles or individuals involved in civil commitment procedures) to focus excessively on the psychopathology or let the intern do so often distracts both of them from handling the legal problem at hand in an optimum way. Under such circumstances the intern may be most helpful with the client's psychiatric difficulties by delving no further than to ask the client whether he or she is getting help for the emotional problems. Of course, there are times when psychiatric problems and the quality of psychiatric treatment are of central importance and need careful scrutiny by the intern and consultant. One such case led to litigation, which was eventually dropped, against a mental hospital and at least two of its psychiatrists. About a third of the cases for which consultation is sought are subsequently referred for psychiatric evaluation or treatment. Many of these are juvenile or child custody cases in which psychiatric evaluation or treatment is often desired by the court or intern for optimum resolution of the legal situation.

I have been favorably impressed with the quality of representation by most of the legal interns; mental patients seem to receive equal or better advocacy from legal interns compared with private attorneys. Perhaps reciprocally, the consultants have been fairly well received by the interns and the clinic faculty; about three-fourths of the interns avail themselves of consultation services. Questionnaires the interns complete to evaluate their experience in the clinic generally indicate that the consultations have been considered quite helpful.

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Voodoo in Africa and the United States

BY KENNETH M. GOLDEN, M.S.W.

The author states that the practice of hexing is not just a nearly extinct phenomenon of isolated rural communities of the deep south of the United States but is evident in large urban areas in the northeast and west. He describes the psychological mechanisms of voodoo as practiced in West Africa to provide insight into similar practices in the United States.

THERE HAVE BEEN scattered reports of suspected illness and death resulting from voodoo practices in the United States (1–4). The following case is illustrative:

Case 1. A 33-year-old black man from a rural area near Little Rock, Ark., was admitted to the neurology service of the University of Arkansas for Medical Sciences. The patient had been having seizures recently and had become increasingly irritable and withdrawn from his family. When he could no longer be detained safely on the neurology service, he was transferred to the psychiatric ward, where he became increasingly more agitated, confused, and almost delirious. He became very fearful whenever people approached him, and he began to hallucinate. He finally slowed down after being given 1000 mg of chlorpromazine (Thorazine), but the necessity for bed restraint remained. All neurological findings, including a brain scan, proved normal.

After two weeks of hospitalization the patient suffered a cardiac arrest. All efforts to revive him failed. An autopsy provided no reason for the death. After he died, the patient's wife told staff members that her husband had been seeing a "two-headed," an older woman considered by the community to be a witch who cast spells and healed people. The widow stated that her husband had angered the two-headed and that she had caused his death.

"Conjure doctors," "the two-headed," "root doctors," and "hoodoo men" are believed to be able to trick or hex a person, causing sickness, insanity, and death; they are also held to be able to cure a person who has been hexed (5–7). These agents use herbs, roots, stones, powders, insects, charms, and incantations. I have heard reports of a woman who lives in a federally funded housing project in North Little Rock, Ark., who is widely regarded as capable of casting spells that can cause marital discord, illness, sterility, madness, and even death. She has a collection of voodoo literature, sets of voodoo images, and quantities of potions in her apartment.

Hexing practices are no longer a phenomenon only of rural isolated communities in the deep south. Physicians have provided evidence that voodoo and hexing practices exist as far north as Connecticut (2–4). Complex systems of voodoo practice have also been described in heavily populated urban areas (8). Similar belief systems have been shown to exist among many subcultures and ethnic groups throughout the United States, such as in the Mexican-American communities in the southwest (9–11).

Voodoo and similar cult practices are still in evidence in some areas of Africa. The practice of voodoo in the United States is a blend of African voodoo and European witchcraft beliefs. The practices that evolved in the United States were in reaction to the environment here (12). Although voodoo in the United States differs from the African practice, an understanding of the function, importance, and evolution of voodoo in its original environment may provide insight into its use and capabilities in the United States.

AFRICAN CURSE DEATH

As a Peace Corps volunteer teacher I spent two years in West Africa. There I lived in an area where the voodoo cult originated (7) and where cursing and hexing were actively practiced (13). *Vodu* in the Ewe dialect of the West African village I lived in means "one to be feared." The vodu cult was only one of a number of similarly powerful cults. (The practice of vodu was transported by Ewe slaves to Haiti and Louisiana during the early part of the eighteenth century. The primary diety of the vodu cult, the legba, appeared throughout my West African village in the form of moon-faced idols made of sun-hardened clay. The legba is still a prominent diety in Haiti.)

The village is only 8° away from the equator; the villagers barely eke a subsistence out of this scorching, barren plains area. Although the village appears to be sleepy, the lives of the villagers are strictly dictated and controlled from birth to death by the traditions of the cults. Disobedience of tribal custom is punished by fines, disgrace, banishment, or, when the infraction is particularly serious, by curse death, which means certain death to the victim.

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My landlady was fatally affected by such a curse. She was a prosperous marketwoman in her mid 30s who often traveled to neighboring Togo and through the southeast corner of Ghana to market her goods. For a year or so she had been suffering from severe and acute attacks of abdominal pains. She had had exploratory surgery performed by European doctors on three occasions with no positive result. Toward the end of my Peace Corps tour I noticed that she was losing weight and saw her less and less often in the marketplace. When she died, there was, uncharacteristically, no funeral celebration for her; she was buried on the outskirts of the cemetery. When I asked a friend of mine why, I was told that she had been cursed by the yehwe, one of the major cults in the village, because she had been an adulteress.

For the curse to be successful, the victim has to be made aware that he or she has been cursed. Priests learn through divination ceremonies that gods or ancestors are angered due to the transgressions of a certain villager. It is not hard to imagine how in a small village the priests could obtain this knowledge in less than otherworldly ways. The priest's divination soon becomes common knowledge. The slightest suggestion is sufficient to cause tremendous fear in the victim. Tribal laws are rigid, and all members know them. The transgressor knows that there is nothing he or she can do to reverse or negate the curse. Death comes slowly but surely over a period of months.

When the curse becomes known, the victim's family and friends as well as the entire community withdraw their support. The victim becomes an outsider to the few cohesive and organized activities of the village. He or she is thus no longer protected from the evil wishes of ancestors and witches.

Feeling hopeless and helpless, the victim withdraws, thus furthering his or her isolation. Eating and drinking habits become irregular, and the victim settles into an increasingly lethargic state. Although the threat to life is not acute, the emotional strain of feeling hopeless is evident over an extended period of time. The victim fatigues easily in order to conserve the energy needed to protect threatened resources from the emotionally overstressful situation (14). The victim remains in a state of chronic fatigue and melancholia, and, with no interest in living, he or she simply dies.

Unlike the curse death in this village, curse deaths in other parts of Africa have been reported to occur immediately after the curse is placed (15, 16). Often only moments after having been pointed out in a tribal gathering, the victim drops dead. These instances often occur in lengthy tribal ceremonies called specifically for the purpose of discovering a transgressor; the victim's anxiety, fear, and guilt are greatly heightened as the ceremony proceeds. When cursed with all the drama of the ceremony, the victim dies suddenly. Many physicians have speculated on the physiological basis of such curse death as well as other types of death caused by emotionally stressful situations (15–22).

In my West African village there are individuals known as *jujumen* who also have the ability to cause harm to others through magic. Unlike the priests, jujumen are not part of the legitimate social system. If a person wishes evil on an enemy, he or she can persuade a jujuman with money and goods to hex the victim. The victim is informed of the hex through a sign, such as powder on a doorsill or owls flying over the house at night. The hex is believed to cause insanity, sterility, infertility, and sickness. Unlike the curse, it can be negated and even reversed by a more powerful jujuman.

DISCUSSION

A number of prerequisites to the success of a curse or hex appear wherever in the world they are found: the victim must believe in the power of the person who administers the curse and must either know or at least suspect that a hex has been placed. The resistance to Western medical techniques both here and abroad might depend on the strength of the victim's belief. In the village I lived in, belief in the power to hex and curse is wholehearted. In areas where the belief is weaker, the victim seems more available to treatment (1). The use of projection and denial by the victim seems to occur when curses and hexes are effective (23, 24). Overdependency and a feeling of powerlessness also occur.

The practice of some churches in the United States presents a similar example of communal belief: priests and elders in these churches often provide total care in the areas of physical and emotional problems (25–28), and exorcisms are not uncommon (29, 30). The communal rejection experienced by the victim of the African curse has its parallel in the European and American practices of shunning, ostracizing, railing, and excommunicating.

However, a religious, tribal, or community belief system is not essential for a curse to be effective. Overdependency on parents rather than on a priest seems to be all that was necessary in certain cases reported in the United States (31, 32). Further, psychophysiological forms of giving up are often seen in hospitals. Patients who suffer a loss of body image due to surgery or an accident or those told of their imminent death have been known to react by withdrawing, eating and drinking poorly, and socially isolating themselves; at times these reactions result in premature death (33). Anorexia nervosa and marasmus may be similar giving up mechanisms (34).

Overwhelming feelings of fright, fear, hopelessness, and helplessness, not unlike those felt by the cursed African, have been known to cause death in the United States when people have been confronted with the demise of a loved one, a business loss, or a dangerous situation (18, 35, 36). "Anniversary" and other forms of sudden and unexplainable deaths might be psychologically and physiologically related (37–39).

The implications of these phenomena might be far-reaching. Further systematic and rigorous research seems necessary and indicated. Research can provide the mental health professional with greater understanding of the powerful influence of culture and society on the individual and the intricate relationship of mind and body.

Patients are often reluctant to tell their doctors about hexing practices for fear of being belittled or not understood. Physicians might be unaware of the existence and importance of these beliefs to their patients. With the continued disaffection and isolation of various minority and ethnic groups in the United States, the trend toward reliance by large segments of the population on indigenous practitioners might be on the increase. An awareness on the part of physicians of the existence of hexing practices within their communities may lead to more reported cases and the development of effective treatment techniques to deal with the psychological resistances and physiological difficulties of patients who have been cursed.

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John Stuart Mill on the Liberty of the Mentally Ill: A Historical Note

BY JOHN MONAHAN, PH.D.

The author discusses the quote from Mill's On Liberty *that is often cited by libertarians in opposition to involuntary commitment of the mentally ill. This quote has been taken out of context; other statements in the document indicate that Mill excluded from his libertarian credo those "without the ordinary amount of understanding," i.e., those people who would now be considered mentally ill.*

IN THE continuing debate on the involuntary commitment of persons diagnosed as mentally ill and believed to be suicidal, gravely disabled, or otherwise a danger to themselves, one passage has been quoted repeatedly by libertarians to provide historical and philosophical support for their position. It is the credo of John Stuart Mill in *On Liberty*:

> the only purpose for which power can be rightfully exercised over any member of a civilized community, against his will, is to prevent harm to others. His own good, either physical or moral, is not a sufficient warrant. He cannot rightfully be compelled to do or forbear because it will be better for him to do so, because it will make him happier, because, in the opinions of others, to do so would be wise, or even right. (1, p. 22)

This eloquent statement of the libertarian tradition in its most pure and classic form is cited in virtually every legal, psychiatric, and psychological treatise on the right of the mentally ill to decline intervention that others believe would benefit them (e.g., references 2–4). The statement is interpreted as indicating that to be a true libertarian, one must eschew all involuntary treatment of the mentally ill that is justified by a *parens patriae* concern for their welfare.

The purpose of this communication is to suggest that a fuller reading of Mill's work would lead to the contrary conclusion that he specifically excluded those who would now be called "mentally ill" from the scope of his argument. One is, of course, free to hold that Mill was in error when he declined to apply his libertarian thesis to the mentally ill, much as we today would object to his exclusion of "barbarians" (see below). Yet it should be recognized that Mill himself found nothing inconsistent in applying libertarianism only to those lacking a mental disorder. Quoting Mill in support of the abolition of civil commitment is done out of context and has the effect of distorting the historical record.

In the paragraph following the one typically cited, Mill notes the first exception to his libertarian thesis that only harm involving others is subject to state action:

> It is, perhaps, hardly necessary to say that this doctrine is meant to apply only to human beings *in the maturity of their faculties*. We are not speaking of children, or of young persons below the age which the law may fix as that of manhood or womanhood. Those who are still *in a state to require being taken care of by others* must be protected against their own actions as well as against external injury. (1, p. 22, emphasis added here and in following quotes).

In its most narrow reading, this exemption applies only to children, but it may also be read as applying to others who lack "the maturity of their faculties" and are "in a state to require being taken care of by others." That Mill intended the broader reading can be judged from his next sentences, which constitute a startling example of the colonial racism of the mid-nineteenth century:

> For the same reason, we may leave out of consideration those backward states of society in which the race itself may be considered in its nonage. . . . Despotism is a legitimate mode of government in dealing with barbarians, provided the end be their improvement, and the means justified by actually effecting that end. Liberty, as a principle, has no application to any state of things anterior to the time when mankind have become *capable of being improved by free and equal discussion*. Until then, there is nothing for them but implicit obedience to an Akbar or a Charlemagne, if they are so fortunate as to find one. (1, p. 23)

Later in *On Liberty*, Mill makes the exemption of individuals who would now be regarded as mentally ill more explicit.

> As soon as any part of a person's conduct affects prejudicially the interest of others, society has jurisdiction

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over it, and the question whether the general welfare will or will not be promoted by interfering with it, becomes open to discussion. But there is no room for entertaining any such question when a person's conduct affects the interests of no person's conduct besides himself, or needs not affect them unless they like (all persons concerned being of full age, and *the ordinary amount of understanding*). (1, p. 135)

Finally, toward the end of his treatise, Mill directly addressed the issue of persons who engage in behavior "dangerous to self," clearly holding that the psychologically disordered constitute an exception to his general rule of nonintervention:

If either a public officer or anyone else saw a person attempting to cross a bridge which had been ascertained to be unsafe, and there was no time to warn him of his danger, they might seize him and turn him back, without any real infringement of his liberty; for liberty consists of doing what one desires, and he does not desire to fall into the river. Nevertheless, when there is not a certainty, but only a danger of mischief, no one but the person himself can judge of the sufficiency of the motive which may prompt him to incur the risk; in this case, therefore, (unless he is a child, *or delirious or in some state of excitement or absorption incompatible with the full use of the reflecting faculty*), he ought, I conceive, to be only warned of the danger; not forcibly prevented from exposing himself to it. (1, p. 172)

The full record, therefore, indicates that Mill was quite aware of limits to his libertarian philosophy. He chose to exclude from its range of application those not "in the maturity of their faculties," those "in a state to require being taken care of by others," those without "the ordinary amount of understanding," those not "capable of being improved by free and equal discussion," and those whose behavior was "incompatible with the full use of the reflecting faculty"—in short, those who today would be regarded as mentally ill. Use of the partial citation to the contrary is historically inaccurate.

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CLINICAL AND RESEARCH REPORTS

This section contains reports of new clinical or laboratory observations, single cases of unusual interest or importance, descriptions of pilot studies, suggestions for future research, etc. Reports should be submitted in duplicate, typed double-spaced throughout, and may contain 1,000 words, no more than 1 table, and a maximum of 10 pertinent references. Figures cannot be used. Reports will be reviewed by at least one referee and the Editor; the author will receive a postcard informing him/her of the disposition of the submission. Reports will be edited for clarity and conformance with Journal *style.*

An Oral Opiate Antagonist in Chronic Schizophrenia: A Pilot Study

BY DAVID H. MIELKE, M.D., AND DONALD M. GALLANT, M.D.

Several endogenous peptides with biological properties resembling opiates have been isolated from brain. These compounds, chemically classified as endorphins and enkephalins, have been reported to affect a number of behavioral measures in animals, which suggests that their regulation could play an etiologic role in mental illness (1). Beta-endorphins may be the most potent of these compounds: intracisternal injection in rats has induced a rigid immobility (catatonic state) lasting for hours (1). "Wet-dog shaking," similar to that seen with opiate withdrawal, was also observed following lateral ventricular injection of these peptides. The β-endorphin-induced catatonic state and the shaking induced by alpha-, beta-, and gamma-endorphin and met^5-enkephalin were reversed by the administration of naloxone (1).

Jacquet and Marks (2) found that microinjection of β-endorphin in rat periaqueductal grey matter resulted in profound sedation and catalepsy, which led them to suggest that this peptide is an important neuromodulator of the CNS, exerting effects similar to those produced by neuroleptics.

Studies in human subjects have been encouraging. Gunne and associates (3) observed an immediate hallucinolytic effect of naloxone, a narcotic antagonist, in two chronic schizophrenic patients. In a small group of chronic schizophrenic and manic-depressive patients, the levels of two endorphins were measured in serial samples from the cerebrospinal fluid (CSF) before and after treatment with a neuroleptic agent (4). The authors interpreted their results as indicating that endorphins may reach abnormally high levels in patients with chronic psychoses because those patients who responded to treatment showed a decrease in CSF endorphin levels.

Naltrexone is a long-acting potent narcotic antagonist that is effective when administered orally (5, 6). It is approximately 17 times as active as nalorphine and 2 to 3 times as active as naloxone in precipitating abstinence (5). In doses of 50 mg/day, naltrexone exerts a degree of blockade similar to that obtained with 4 mg of oral cyclazocine and has minimal agonistic effects (6). No significant adverse reactions have been observed in clinical studies (7). Forty-eight-hour blockade against 25 mg of heroin has been observed with administration of 120 to 200 mg of naltrexone (8).

Method

The purpose of this study was to evaluate the efficacy and safety of naltrexone HCl in five schizophrenic inpatients, each having at least seven positive items of the 12-point diagnostic scale used by the World Health Organization (WHO). According to the WHO criteria, a minimum requirement of 7 positive items should offer no more than a 1% chance of diagnostic error (9). Subjects were one man and four women, aged 34–47, who had been hospitalized for 5–26 years. All patients displayed florid symptoms of psychosis with active hallucinations at the outset of the study. All patients were free of active medication for 2 weeks before administration of naltrexone. The initial dosage was 50 mg each morning, and the daily dosage was increased by 50 mg per week to a maximum of 250 mg/day in divided doses. The effects of naltrexone were evaluated by the Clinical Global Impression scale (CGI), the Brief Psychiatric Rating Scale (BPRS), and

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Naltrexone was supplied by Endo Laboratories, Garden City, N.Y.

the Nurses' Observation Scale of Inpatient Evaluation (NOSIE). Efficacy measures were conducted at baseline and every other week throughout the 6-week evaluation. The laboratory procedures included complete blood count, urinalysis, BUN, SGPT, and alkaline phosphatase and were performed at baseline and weekly throughout the study period. An ECG tracing was obtained each week.

Results

The findings of this exploratory uncontrolled clinical trial suggest that naltrexone, in doses as large as 250 mg/day, is ineffective in alleviating schizophrenic symptoms. Two subjects were dropped when they were receiving 50 mg twice daily because of severe exacerbation of the psychotic process. Three patients were given 250 mg/day in divided doses for 9 days without evidence of symptom remission. The two subjects who were dropped were diagnosed as having chronic undifferentiated schizophrenia. The diagnoses for the remaining three subjects, who were given higher doses, were paranoid schizophrenia (N=1) and chronic undifferentiated schizophrenia (N=2). The CGI, BPRS and NOSIE reflected the lack of clinical improvement.

All patients remained free of side effects. Laboratory findings showed no significant deviations from pretreatment levels and weekly ECGs maintained baseline configurations.

Comment

Although no definitive conclusions can be drawn from the results of a small uncontrolled trial, there appears to be little doubt that naltrexone, in daily doses of 250 mg, is an ineffective treatment for schizophrenic symptoms. One would have to consider the possibilities that naltrexone does not antagonize CNS endorphin activity in schizophrenic patients or that abnormal amounts of endorphins do not have a significant primary role in the production of schizophrenia.

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Rapid Treatment of Delirium in Critically Ill Patients

BY DAVID P. MOORE, M.D.

Delirium in the critically ill patient may be life-threatening. Delirious patients may endanger themselves by pulling out tubes and arterial lines or misaligning fractures. Therefore, rapid symptomatic treatment of the delirium is indicated. The following case report describes the rapid treatment of a delirious patient with a regimen used in rapid tranquilization of patients with functional psychoses.

Case Report

The patient, a 34-year-old man, was admitted to the hospital after an auto accident. He sustained second-degree burns over the face, neck, and shoulders, pulmonary contusion, and multiple fractures. A laparotomy revealed a ruptured spleen, and a splenectomy was performed. The patient was placed on controlled ventilation and given morphine and diazepam as needed for pain during burn care. During the first 7 days in the intensive care unit the patient was cooperative, followed directions, and communicated with staff by nodding his head "yes" or "no." On the 8th day he became agitated, struck a nurse, and pulled out his nasogastric tube. Physical and laboratory examinations failed to reveal anything that could account for the change in the patient's clinical state, and diagnosis of delirium of unknown etiology was made. Rapid treatment was started with haloperidol, 10 mg I.M. every hour, with an endpoint of satisfactory remission or limiting side effects.

After he had received 30 mg of haloperidol, there was sufficient remission to allow resumption of routine burn care. There was no evidence of sedation, hypo-

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tension, or extrapyramidal side effects. A regular regimen of haloperidol was started at a dosage of 15 mg b.i.d. through the nasogastric tube. An order was also given for supplementary doses of 5 mg of haloperidol I.M. every hour as needed for motor agitation and uncooperativeness. Over the next 4 days the supplementary dosage required gradually decreased, and on the 14th day the patient required no supplementary haloperidol. Over the next 5 days the regular daily dosage was tapered, and the haloperidol was discontinued entirely on the 19th day. The patient's subsequent hospital course was unremarkable, and the delirium did not recur.

Discussion

Delirium has been characterized as a syndrome of cerebral insufficiency (1). Optimum management of delirium includes diagnosis and treatment of the underlying cause of the cerebral insufficiency and symptomatic treatment of the delirium itself. Symptomatic treatment may involve environmental manipulation and drug treatment. Environmental measures that are often effective in treating delirium include a continuing relationship with a physician (2), measures to permit adequate sleep (3), placement in a room with windows (4), and provision of clocks, calendars, and night-lights. However, these measures may not produce a sufficiently rapid response.

When rapid attenuation of the delirium is required in order to prevent life-threatening behavior, drugs may be useful. Diazepam, chlorpromazine, and haloperidol have been used in the treatment of delirium (5), but to my knowledge there have been no studies comparing their effectiveness in delirium. By analogy with the functional psychoses, I hypothesized that antipsychotics would be effective in the treatment of delirium.

Rapid treatment of functional psychoses with both haloperidol and chlorpromazine has been effective (6). In one study chlorpromazine was found to be more likely to produce convulsions and severe hypotension than was haloperidol (6). Thus haloperidol would seem to be preferable to chlorpromazine because it is less likely to cause life-threatening side effects. Rapid attenuation of psychotic symptoms can be achieved by giving haloperidol, 5 mg/hour I.M. (6–8). Oral administration is unsatisfactory for rapid treatment because peak plasma levels are not attained for 2–6 hours (9).

Acting on the hypothesis that haloperidol would be as effective in the rapid treatment of delirium as it is in functional psychoses, I have treated three critically ill delirious patients with haloperidol, including the patient described in this case report. Each patient was given 5–10 mg of haloperidol I.M. every hour until either remission or limiting side effects occurred. No patient required more than 30 mg before remission occurred. After the patients responded to the rapid treatment, a regular dosage was started in order to maintain the remission until the underlying cause of the cerebral insufficiency was resolved.

There are no experimentally verified guidelines for deriving a regular dosage from the total dosage used during rapid treatment. I started patients on a total regular dosage equal to the amount used during the rapid treatment. This regimen provided some control of the delirium without causing untoward effects. In order to provide further control as needed, I also ordered supplementary doses of haloperidol, 5 mg I.M. every hour as needed. Supplementary doses were generally required daily for several days to maintain control. Fewer supplementary doses were needed over succeeding days as the underlying cause of the cerebral insufficiency gradually resolved. After any 24-hour period in which no supplementary doses were required, the total regular daily dosage was reduced by one-third. This reduction was continued, providing the delirium did not recur, until the patient was no longer receiving haloperidol. This regimen was successful in alleviating the delirium in all three patients. There were no limiting side effects.

My experience with these three patients indicates that delirious patients may be treated effectively, safely, and rapidly with haloperidol. Controlled studies are needed to confirm this hypothesis. In the meantime, I suggest that consideration be given to the use of haloperidol in treating the critically ill patient with a life-threatening delirium. The number of deaths secondary to self-extubation and other life-threatening behavior might be reduced in this way, and delirious patients might become more cooperative in their treatment.

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Suicide in Adolescence

BY PAUL C. HOLINGER, M.D.

Suicide is the second or third leading cause of death in adolescence in many Western countries (1, 2); only accidents and homicides are responsible for more deaths in that age group in the United States (1). However, there are serious methodological difficulties involved in researching and writing about adolescent suicide (3). The observation regarding the lack of case studies and presuicidal data throughout the literature on adolescent suicide (4) is understandable in light of these problems. Notable exceptions include reports by Mason (5) and Sabbath (6).

This paper reports an unusual case of double adolescent suicide in which two brothers killed themselves by hanging in separate incidents within a month. The suicides occurred in institutions that are usually protective—a jail and a psychiatric hospital.

One must consider the multiplicity and interweaving of factors associated with any suicidal outcome. This case, however, highlights three particular issues with corresponding treatment implications: the impact of a suicide on another suicidal adolescent and his family (7), the inadequacy of mobility of investments of the suicidal adolescent (3), and the role of hostility from significant others (6, 8).

Case Report

Ted, a 16-year-old boy, was admitted to the adolescent unit for depression, narcotic use, and stealing. These symptoms had been present since age 10 but had increased markedly and had been accompanied by suicide attempts and psychiatric hospitalization after his girlfriend left him about 1 year before this admission.

Ted's birth and his mother's pregnancy were considered normal. He had three older sisters, two older brothers (6 and 3 years older, the latter named John, after his father), and a younger brother. His parents stated that Ted was different from and slower than the others, noting that he had repeated first grade. The patient characterized his mother as anxious and ungiving and his father, a blue-collar worker, as warmer but unavailable. Ted stated his parents especially valued academic achievement, which prevented him from getting much feeling of worth from them. He attempted to invest himself in his sisters and brothers, particularly John, and his first memory involved playing Lincoln Logs with John and "having a great time." Ted had always met peers as "Johnnie's younger brother" and saw himself in that way. He felt that John had "everything one needed to be popular: broads, drugs, music, looks, and money."

Ted was hospitalized for 5 months. There was no evidence of thought disorder or psychosis, and he initially displayed a quality of bravado. He and his family, particularly the parents, engaged in intensive treatment aimed at establishing a therapeutic alliance and dealing with his massive depression and feelings of dependency, rejection, inadequacy, and rage. After he was discharged, Ted did well for 3 months, at which time a recurrence of the admitting symptoms prompted a search for rehospitalization.

During this search, John was jailed overnight for drug possession, and it was thought that he may have ingested other drugs, including barbiturates, to prevent the police from finding them. That night, John committed suicide in jail by hanging himself.

Initially, Ted appeared to have integrated around the suicide. He saw himself as having a mission, i.e., helping his parents through the crisis, and he functioned well. However, the initial integration slowly became suspect, particularly in the face of the parents' continued glorification of John. Ted described a "weird" feeling being in John's room, took over the use of his car, and occasionally wore his clothes. Six weeks after his brother's death, Ted was jailed for a minor traffic violation. His mother physically fought with Ted when she was paying his bail, reportedly screaming "I wish you had died instead of your brother Johnnie," and "God took the wrong one." The next day, self-destructive gestures with a rope and an overdose precipitated Ted's emergency admission to another psychiatric hospital. He soon began to seem less depressed and to talk about future plans. However, 5 days after his admission Ted slipped away when the staff's attention was diverted and was found hanging by his belt in the shower room. He was in coma and died 24 hours later.

Thereafter, therapeutic efforts were aimed at preventing further deterioration within the rest of the family, and the parents started supportive treatment. Ted's mother was especially guilty about the relief she felt with Ted gone and her physical safety and that of her possessions assured.

Discussion

Although one must keep in mind the multidetermined nature of suicide, three specific issues in this case warrant particular attention. First, with respect to the impact of the suicide the phenomenon of two adolescent brothers committing suicide within a month by the same method is important. Ted's integration around his idealized brother's death is noteworthy. This integration seemed to develop through

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Ted's increasing sense of mission in regard to his parents and through increasing identification with his dead and glorified brother. He tried to take his brother's place and identity in the family and grasped for some sense of self-worth. Treatment implications include the need for awareness of the potentially critical nature of such an integration around a sibling's suicide and for the availability of treatment for the parents as well as the surviving adolescent.

Second, Haim (3) noted that adolescents usually can perform the work of mourning that results from narcissistic wounds, damage to the ego ideal, and object losses. However, suicidal adolescents have inadequacy of mobility of investments, i.e., inability to disinvest the disappointing or lost object. Despite the pain, they maintain their investments, repeat their behavior, and brood on their disappointment (3). Ted manifested a marked inability to disinvest his parents because of the nature of his attachments to them and the unavailability of different channels of investment. His other major investment became his brother John and the delinquency subculture that John valued. Throughout treatment there was some evidence of investment in and idealization of the therapist and staff. Treatment implications include the necessity of mobilizing, and being able to tolerate, idealization by such patients.

Third, the role of hostility from significant others has been studied less than the patients' hostility turned inward. Sabbath (6) proposed the expendable child concept, which presumed a parental wish, conscious or unconscious, interpreted by the child as a desire to be rid of him. Gould (9) contended the core factor in the formation of a "suicidal personality" in adolescents was the felt loss of love. Rosenbaum and Richman (8) found more extreme hostility and less support in families of suicidal patients than nonsuicidal patients. Ted's parents saw him as "different," if not unwanted, and the reported eruption of overt hostility was striking. Sabbath (6) extended the expendable child concept to that of the expendable patient in terms of staff-patient or doctor-patient relationships, and Havens (10) described unconscious desires on the part of the staff to get rid of a hospitalized adult patient who later committed suicide. Such reactions, best understood as countertransference phenomena, were present in the therapist and staff in the case described here. Treatment implications include recognizing, understanding, and working through such transference and countertransference reactions, in addition to helping the family and patient to understand the role of expressed hostility.

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The Capgras Syndrome as an Adaptational Maneuver in Old Age

BY ALVIN I. GOLDFARB, M.D., AND MARCELLA BAKUR WEINER, ED.D.

Capgras and Reboul-Lachaux in 1923 first described a syndrome of failure to recognize a familiar person coupled with the belief that he or she was a "double" who was similar to the original person but an impostor (1). The syndrome has been described in persons with paranoid and manic-depressive psychoses and other conditions; it is usually attributed to ambivalent emotional attitudes toward the person whose real identity is denied.

The Capgras syndrome has caught the attention of many authors (2–4). It was believed that the patient's feelings of strangeness (anxiety) about the familiar others led to this state of nonrecognition. Depersonalization and unreality factors have been proposed as explanations for the syndrome (5).

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Unconscious libidinal drives also have been suggested as the underlying force in that the syndrome may be a "defense reaction" against the unleashing of forbidden impulses (1). The other person is denied, negated, or rejected through the use of defenses such as projection and identification. Three factors characterize this conceptualization: 1) the impostor is a person of importance to the deluded one, 2) the pretender is not equal to the impersonated other, and 3) attempts to deal with the anxiety seem to lead to the delusion. Although some of the afflicted persons were "confused" and "disoriented in time," most of the patients described by the authors cited seem to have been cognitively intact. Observers noted that often the perceived "double" resembled the misidentified person in the past. The "illusion of doubles" is thus rooted in a "change of consciousness marked by a double attitude toward the present and past experience" (4).

In our experience, the Capgras syndrome occurs with frequency in association with those difficulties of memory and learning classified as organic brain syndrome (OBS) or dementia. The illusion of doubles has been cited as "an example of delusional misidentification," a state of nonrecognition or inability to properly identify a person well-known and personally related to the afflicted individual. This double is always an important person, and the patient does not misidentify others. The double is usually accused of not having the virtues the patient would like the "original" to possess. The dynamics thus appear to be an adaptive solution by way of communicating that the accused person is in need of change, i.e., should be different than he or she is (6). The following cases present some examples of the syndrome.

Case Reports

Case 1. A man of 78, Mr. A, was found wandering in the streets. Taken to the police station, he said that he was looking for his wife. When his wife was telephoned and spoke to him, he said, "That is not the wife I'm looking for." Apparently, he acknowledged that he had a real wife whom he had left to find another who would be more gratifying. Although he did not call his wife an impostor, she was nevertheless "not his wife." His behavior may be interpreted as a coping attempt. Such an attempt at mastery is a search for aid, a search for a beneficent relationship, whether or not it was experienced in the past; it is an indication of the need for a dependent relationship (7).

Case 2. Ms. B, a 78-year-old woman, accused her husband of being an impostor. She stated that she did not want him in the house. Whenever he left the house (e.g., to empty the garbage), she locked the door and would not allow him back in. In order to gain access to the house, he had to call their son or neighbors or plead with his wife to let him in. He was constantly the target of her abuse and accusations. She also called her son to complain about the interloper, the impostor, and could not be convinced of his identity.

He had been and remained a demanding and intemperate person unable to put up with his wife's many shortcomings, which had increased with her progressive memory loss. He could not understand her current difficulties as being related to this irreversible memory loss. She, in turn, bitterly resented his critical attitude. In the past, she had always both respected and adulated him, restraining any criticism or opposition out of fear.

In treating the couple, it was pointed out to the husband that his wife was reacting to his seeming criticism of her by putting blame onto him, saying, in effect, "You aren't the husband that I had, the one who understood me. Therefore, you are a stranger to me." It was explained that she could not help forgetting things but that he could not be blamed if he occasionally lost his temper, and he was told that it would be helpful for her if he were nonconfrontational and supportive. The son received similar explanations.

Discussion

The Capgras syndrome, as described in the cases cited, appears to be a common elaboration of a desire to recapture a dependent relationship with a close associate that has been lost either because there has been a change in one's needs or in the character of the previously supportive person. It is understandable as an attempt to find personal assistance, emotional support, and reassurance on the part of one who feels frightened and weak from another who is delegated to behave in a supportive way. The behavior of the aged person displaying the Capgras syndrome may be regarded as motivated and determined by the patient's current emotional and physical status, his or her life situation, personal expectations, and past patterns of adjustment. The motive is to reconstitute the environment by replacing the current, nongiving, nonacceptable one with a nurturant, giving, and acceptable one. Such behaviors can be understood as adaptive attempts. Thus, the Capgras syndrome is a clear-cut and easily understood elaboration of the individual's attempt to force, to gain, to manipulate, and to get from another person that which he believes is coming to him. It is therefore not necessarily an indication of idiosyncratic personality difficulties in the aged person or of specific psychotic states such as paranoid or manic-depressive psychoses, as many have suggested (2–4). Rather, this syndrome may occur (as it does in older persons) whenever there is a decrease in discriminatory capacities (7).

Conclusions

The perception of imaginary differences and imaginary resemblances with respect to persons well-known to an elderly individual is frequent and can be understood as based on subjective distress, unhappiness with an existing relationship, desire to change, and attempts to signal the need to alter transactions and to find what is now lacking in the people around them. Therapeutic intervention based on an understanding of

this phenomenon can lead to reduced intensity of symptoms in the patient and comfort to both him and his family.

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Lithium in the Treatment of a Child Abuser

BY BARRY M. PANTER, M.D.

In this article I will report the use of a new treatment approach, lithium, in the case of a woman who abused her children. The decision to use lithium was based on several factors. The child abuse and other destructive acts occurred while the woman was in a rage, and Sheard (1) has found lithium to be effective in the control of rage reactions. Rifkin and associates (2) reported that lithium helps to dampen affect in patients with the diagnosis of emotionally unstable character disorder; my patient described changes in mood consistent with cyclothymic personality or mild manic-depressive illness. Phenothiazines, antidepressants, and minor tranquilizers had not been helpful. The patient's downward spiral into destructive impulse and action produced a sense of desperation in everyone involved.

At the time lithium therapy was started with this patient, there were no reports of this use of lithium in the literature. Recently Kerr (3) reported a similar case in which aggressive outbursts and child battering ceased when his patient was stabilized on lithium.

Case Report

Ms. A was 30 years old when she was referred for treatment because of frequent verbal and physical family fights, which were often followed by Ms. A's sullen withdrawal, drinking and driving, and abuse of her children, Annie, age 4, and Joey, age 7. The need for intervention was recognized by Mr. and Ms. A.

Her destructive behavior erupted after the family moved close to Ms. A's parents, which reactivated repressed conflicts and contributed to Ms. A's psychological and behavioral decompensation.

Ms. A is the older of two girls. Her sister was the "good," favored child, and she was the "bad," neglected one. Her mother was promiscuous and habituated to pills for pain and insomnia. Ms. A felt that her mother used her like an object. Her father was withdrawn and not interested in her. Ms. A recalled that when her feelings were hurt she became angry and cruel; she pulled wings off butterflies and smashed the heads of cats. When she was 9 her father began to punish her by pulling her pants down and spanking her. She was excited by this and looked forward to the spankings, feeling guilty about the shared activity with her father and relieved by the pain.

During the years between ages 10 and 18 she was sexually involved with a male friend of the family who was her father's age. He was also her mother's lover. Her mother knew of and encouraged the sexual relationship: "If my mother couldn't be with him, she would send me in her place." Ms. A felt excited by the sex, grateful for the male attention, and guilty. Her sense of herself as the bad child was greatly reenforced.

At 19 she was hospitalized for several months with the diagnosis of schizophrenia. Soon after her discharge she married Mr. A, a successful sales representative for a large retail corporation who is often away from home because of business. When he is home, Ms. A resents him because "He tries to run my life. Everything has to be the way he wants it. He treats me like I'm his slave."

Ms. A had many psychiatric symptoms prior to the move close to her parents. She abused both children, especially her son. They occasionally needed X rays and medical attention for contusions and bloody noses. There were no fractures or hospitalizations. The abuse occurred during "fits of anger." She said, "I meant to stop him from doing something bad, and then I couldn't stop myself from hitting him. I became confused." She drank to "get away from the things that bugged me and to calm myself." She had several phobias that occasionally prevented her from leaving the house. After the family move, the symptoms became worse. She felt her mother treated her unfairly. Her husband was "home and controlling me." Additional symptoms appeared—she hallucinated cameras and eyes watching her and heard female voices telling

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The author would like to thank Dr. Theodore Van Putten for his valuable assistance with this case.

her she was bad and should kill herself. She withdrew into fantasy for days at a time. She enjoyed and was reluctant to leave her self-destructive fantasy world because "At least I control what happens there."

Course of Treatment

The first goal of treatment was to protect Ms. A and her children. Repeated hospitalizations for overdoses and assaultive behavior in the past had done this but little more. There was no clear improvement in her behavior or psychotic thinking on daily doses of 1000 mg of chlorpromazine, 60 mg of trifluoperazine HCl, 800 mg of thioridazine, 40 mg of haloperidol, or 200 mg of doxepin HCl. Extrapyramidal side effects, sedation, and dryness of the mouth were present but minimal. These drugs did not improve the target symptoms of dangerous behavior and psychotic thinking.

The hospitalizations, which varied from several days to 5 months in duration, followed abuse of the children, drinking, or self-destructive acts. The self-destructive acts never consciously distressed Ms. A. She felt that she had gotten something bad out of herself, and was relaxed and calm afterward.

During a later hospitalization, lithium was prescribed, and her rage, assaultiveness, and self-destructive behavior diminished greatly. (The effect of lithium on her behavior is shown in table 1.) She has struck her children twice since she started lithium treatment, but these episodes were, by her description, qualitatively and quantitatively different from those which had occurred before. She was able to gain control of herself quickly and did not hurt the children.

Her lithium level is maintained at 1.3–1.5 mEq/liter on a dose of 1800 mg/day. She has been hospitalized twice in the 2 years since she started on lithium, first after an overdose of analgesics and sleeping pills (serum lithium=0.8 mEq/liter) and then after she reported having taken steps in a plan to kill her parents (serum lithium=0.7 mEq/liter). Episodes of psychotic thinking still occur, but she is not impelled to act on these thoughts as she was before.

Ms. A listed the following effects of lithium:

> I am more amenable to suggestion and make positive efforts to change the situation. When I'm off the lithium I find it easier to stay depressed. My suicidal feelings are present but I don't feel the push within me to actually do it. I get angry but I don't lose control of myself. I have not become enraged.

Discussion

The psychodynamics of much of what was happening to Ms. A were clear early in the course of her treatment. Her ability to test reality was intact most of the time. Under stress she regressed to a psychotic state in which primary process was dominant. She was unable to control her behavior and often acted on her psychotic thoughts. Her sense of self was of being incomplete, vulnerable, bad, and threatened, which led to great anxiety. She needed the presence of and fusion with the significant other—mother, husband, or child—to relieve the anxiety of separateness. However, she felt that fusion meant being controlled and used, which enraged her and led to the aggressive outbursts. During the psychotic regressions she often was unable to distinguish between herself and others. She projected the "badness" into her children and then attacked it. When enraged she was not able to distinguish clearly who was being beaten. These violent acts of child beating, wrist slashing, and window smashing represent a fusion of sexual and aggressive drives and a fusion of self and other. The violent act gratifies the fused drives and, insofar as it is self-destructive, atones for the gratification at the same time. The diagnoses of explosive personality and habitual excessive drinking describe Ms. A's past behavior. The most useful psychodynamic diagnosis is Kernberg's borderline personality organization (4).

This understanding of what was happening was not useful to her initially. The tenuous control that her fragile ego could exert on her behavior was overthrown by the onslaught of her rage. Each outburst was followed by guilt-ridden withdrawal and psychologic decompensation. Lithium decreased the intensity of these emotions and she no longer was impelled to act on them. Her ability to control her behavior has enabled her observing ego to develop. As she has gained understanding of herself and her relationships, her anxiety has diminished. She relates to others with less distortion of reality and consequently with less aggression. Since she has been taking lithium, she has been able to provide a safe and adequate environment for her children and her husband.

TABLE 1
History of Destructive Behavior Two Years Before and Two Years After Lithium Therapy

Behavior	Before Lithium	After Initiation of Lithium Therapy
Striking children*	2–3/week	2 (total)
Hospitalizations	11	2
Total hospital days	357	11
Overdoses	10	1
Breaking windows	15	0
Slashing wrists	10	0
Drinking*	daily	rarely
Episodes of drunkenness*	3–4/month	1 (total)
Episodes of rage*	4–6/week	0

*Estimates based on patient's and husband's reports.

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Is There a Role for Medical Ethics in Postgraduate Psychiatry Courses?

BY MICHAEL JELLINEK, M.D., AND DEAN PARMELEE, M.D.

Medical ethics is receiving increasing public and professional attention. Discussions of ethical issues are now a part of many medical school curricula and residency training programs. However, in informal review of postgraduate psychiatry courses, we found that ethical issues were given little or no attention. Is there a lack of relevance or interest in ethical issues among practicing psychiatrists attending postgraduate courses? Are there technical difficulties in constructively sharing ethical perspectives?

We decided to explore these questions at the Harvard Medical School-Massachusetts General Hospital postgraduate course in psychiatry. The three goals of the study were to determine the level of interest in ethical issues, to define specific topic interest, and to evaluate several educational techniques.

Method

All participants in the postgraduate course were given a 1-page questionnaire entitled "Ethics Seminar," stating the general goals of the seminar and a listing of major topics and subtopics (see table 1). Participants were requested to indicate areas of interest and suggest further topics.

The seminar lasted for 1 hour and was recorded. The format for the seminar included a brief introduction and presentation of case studies (1, 2), both chosen on the basis of questionnaire responses.

Results

Of the 85 participants in the postgraduate course who received the questionnaire, 43 returned them; 25 stated that they planned to attend the seminar but only 13 did so. Table 1 gives the number and distribution of responses by topic and subtopic. There was no significant difference in areas of interest between those who attended the seminar and those who did not. "Confidentiality" and "emergency room issues" were the two topics of major interest; subtopics generating the most responses were "third-party payers," "informed consent in private practice," and "forced administration of medication." On the average, each respondent indicated five subtopics of interest. In addition, participants wrote in the following subtopics: management of physician's misconduct, rights of minors and parents, and premature/inappropriate discharge of patients. Participants related anecdotal data from their clinical work, sharing experiences that were common for the psychiatric practitioner. Legal mandates often dominated ethical decision making, and a variety of other factors, such as family wishes, patient's future employability, and specific psychiatric pathology, were secondary. Consideration of abstract ethical concepts and precedents played a minor role.

TABLE 1
Ethical Issues Questionnaire

Topics/Subtopics	Responses	
	Number	Percent
Confidentiality	64	29
Third-party payers	28	13
Inpatient records	18	8
Outpatient records	18	8
Informed consent	35	16
Research	7	3
Private practice	28	13
Contract with patients	37	18
Physician/patient responsibilities	21	10
Implications for patient trust	16	8
Emergency room issues	82	37
Who do you serve?	12	3
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The discussion concerning confidentiality focused on one specific question: How much and what kind of information should be released at a patient's request? If one releases "too much," the patient's future employability or insurability may be jeopardized; if "too little" is released, the third-party payers may not reimburse and the patient's future physician may not have adequate information. In other words, should the physician do what he feels is in the patient's best *medical* interest, or should the physician include social, economic, and legal factors in decision making?

The discussion of forced administration of medication raised many questions. Does the psychotic patient have the right to refuse medication, or is it the physician's responsibility to do what is in the patient's best (medical) interest? What is the physician's responsibility to society or to other patients on a ward if a patient is dangerous? For the most part, participants felt that such questions were interesting but that their decisions in daily practice were made in the context of the individual case and with the feeling that the legal system should make the final determination.

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Discussion

Approximately one-half of participants in this postgraduate course expressed an interest in medical ethical issues. Especially prominent were the areas of confidentiality, the role of third-party payers in health care delivery, and forced administration of medication. Thirteen physicians felt sufficiently motivated to attend the elective seminar, which was held at night and was in competition with other traditional seminars held simultaneously.

The seminar/discussion format is a useful medium for practitioners to explore ethical perspectives and decision-making processes. The discussion group quickly developed a sense of warmth, reflecting both the level of tension and confusion as well as the benefits of sharing common clinical dilemmas.

We saw two problems in the seminar as an educational method: 1) The sharing of anecdotal material did not easily give way to consideration of more abstract ethical concepts. 2) Participants needed more information and guidelines to separate legal, economic, social, and ethical perspectives in problem solving—case studies alone were not helpful. It is possible that a formal lecture in advance of the discussion group would create a more suitable framework for these considerations.

In conclusion we feel that the complex nature of ethical problems, the level of practitioner's interest, and the benefits of the discussion group strongly suggest that ethical issues be included in postgraduate psychiatry courses.

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Paroxysmal Kinesigenic Choreoathetosis or Hysteria?

BY DAVID A. WALLER, M.D.

Paroxysmal kinesigenic choreoathetosis (PKC) is a distinct neurologic entity characterized by recurrent, brief, stereotyped attacks of involuntary bizarre posturing and writhing (1). Attacks are induced by movement after a period of immobility and are sometimes so violent that the patient is hurled to the floor. There is no loss of consciousness, amnesia, or EEG abnormality before, during, or after an attack, and examination between attacks reveals no neurologic findings (2). A physician unfamiliar with this entity is likely to consider the phenomenon hysterical and to treat the patient exclusively with psychological approaches, thereby denying him or her the demonstrated benefits of medication (1, 2).

Case Report

A 22-year-old woman college student came to the neurology clinic of a large general hospital for definitive diagnosis and management of a "seizure problem" she had had since age 8. She stated that a neurologist in her hometown had diagnosed her as having "paroxysmal choreoathetosis" and had treated her with diphenylhydantoin. The number of seizures had increased recently. She also had had psychiatric treatment for a variety of problems seen as either provoking or stemming from her seizures. While a history was being elicited, she doubled over and her right hand and leg writhed for about 5 seconds. There was no loss of consciousness. She then sat up and continued to speak. She described an aura of "feeling like her leg was going to sleep" before severe attacks. Neurologic examination was unremarkable, as were skull X rays, computerized tomography, and waking and sleeping EEGs. Although she claimed to be taking diphenylhydantoin as prescribed, the serum phenytoin level was trace. Mental status examination was unremarkable other than mild blocking that was not associated with psychotic symptoms. As she was leaving the office at the end of a follow-up visit, she suddenly dropped to the floor and started to writhe. When asked to stand up, she arose and walked from the clinic. The diagnosis of hysteria was made, and she was referred to the psychiatry service.

During psychiatric evaluation she had several attacks that seemed to confirm the diagnosis of hysteria, in view of the absence of change in consciousness, postictal amnesia or confusion, or abnormal EEG findings, and the presence of psychological conflicts. Review of the literature on paroxysmal choreoathetosis, however, pointed to this as the correct diagnosis for several reasons: the stereotyped nature of the attacks, precipitated by movement and corresponding exactly

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to Stevens' sequential photographs of attacks (2); movements beginning in the distal portions of the limbs and progressing to violent thrashing that lasted 15–30 seconds; onset during childhood; family history of a sister with an apparently mild form of the disorder (paresthesias that rarely progressed to movements); history of an initially good response to diphenylhydantoin; and, finally, the characteristic absence of other neurologic findings. She was therefore referred for further evaluation by a neurologist who specialized in movement disorders.

The neurologist confirmed the diagnosis of PKC as definitely as was possible in the absence of any known definitive laboratory data and placed the patient on 400 mg/day of diphenylhydantoin. Follow-up in both psychiatry and neurology clinics was advised. In a subsequent psychiatric visit, the patient stated that since she had resumed diphenylhydantoin the number of attacks had decreased strikingly from three or more severe attacks a day to three severe episodes in the 3 months she had been on the medication. Guilt-stricken, she confessed that before her initial visit to the neurology clinic she had not been taking her medication as prescribed. She did not know why. Further psychiatric treatment was advised to deal with her difficulty in accepting help for her disorder, but she was lost to follow-up shortly thereafter.

Discussion

In an overview of the diagnosis of hysteria, Chodoff noted that the term "hysterical," as applied to physical symptoms, initially meant "of nonorganic origin" (3). The term then took on added meanings, and more recent articles on hysterical convulsions have suggested designating them "nonepileptic seizures" or "pseudoseizures" (4, 5) and have provided criteria for suspecting such psychogenic convulsions (4, 5). A diagnostic problem arises in that PKC is descriptively a type of pseudoseizure, which is nonetheless of presumed organic rather than psychological etiology. In a report of cases in Taiwanese immigrants, Jung and associates (6) summarized the evidence for and against classifying PKC as a seizure disorder and concluded that accurate classification is impossible with present information. The effectiveness of various anticonvulsant medications (7) argues in favor of an epileptic phenomenon, while the demonstration that L-dopa is effective (8) supports the theory that it is an extrapyramidal disorder. Two cases that came to autopsy did not show definite structural alterations in the brain (1, 2). Depending on one's confidence in the classic descriptors of PKC, the differential diagnosis between it and hysterical conversion is almost impossible unless there is a history of onset in childhood. Cases of hysterical seizures generally do not begin before puberty (4, 5), but the onset of PKC is usually between the ages of 6 and 15 years (9). By this criterion the case I have described is one of paroxysmal kinesigenic choreoathetosis. PKC should be distinguished from the dystonic form of paroxysmal choreoathetosis, a less common disorder first defined by Mount and Reback (10). Paroxysmal dystonic choreoathetosis usually begins in infancy, and individual attacks may last several hours and are not induced by movement (9).

Although cases in only one member of a family have been documented, PKC often appears in a familial pattern compatible with that of an autosomal dominant trait with low penetrance (6). There is a tendency toward spontaneous remission over time. The apparent organic nature and pharmacologic response of PKC do not diminish the importance of psychological aspects. Emotional stress seems to act as an added precipitant to attacks (1), and patients who are motivated can learn techniques by which they can consciously suppress an imminent attack (6). Kertesz described several patients with PKC as anxious or depressed; one committed suicide (1). In the present case, once the number of attacks was reduced pharmacologically, emotional aspects caused the patient to reject needed treatment.

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Atrial Myxoma Masquerade

BY RALPH N. WHARTON, M.D.

In the general hospital, the consulting psychiatrist is often called on when no "organic pathology" can be found. After extensive medical assessment by a variety of specialists a patient may be advised as a last resort to seek psychiatric help for a panoply of symptoms of an evanescent and sometimes apparently hypochondriacal nature. The true hypochondriac is encountered not infrequently, but so are patients who present with a variety of diffuse aches and pains and weight loss that are related to an underlying tumor which does not become clinically apparent for months. In general, there is an intertwining of both psychological and organic pathology at the time of referral.

There are always new diagnostic entities and novel situations that test the mettle of consultants, demanding the highest clinical acumen. Long-standing psychopathology of substantive proportions often complicates a difficult new situation. When a variety of opinions has been given by physicians in several settings, the patient's conflict may grow and the initial psychological problems may be compounded. In this paper, I will describe the case of a patient who represents a unique combination of all of these problems.

Case Report

A 50-year-old married, childless woman lawyer was admitted to the Columbia-Presbyterian Medical Center in New York City 10 days after the onset of *left-sided* weakness and aphasia that had cleared in a suburban hospital in 2 to 3 hours. An echocardiogram at that facility showed possible evidence of an atrial myxoma.

History. The patient's father and mother-in-law had both died after cerebrovascular accidents in the summer of 1972 following long illnesses. Although she was upset emotionally then and thereafter, the patient did not consult a physician until July 1974.

At that time she complained of an episode of sweating, nausea, and transient blurring of vision that lasted about 1 hour. Her pulse was somewhat rapid (80 beats per minute) and she had a mild, faint tremor. On her second visit to her personal physician she was told she had mild hyperthyroidism and was treated for 3 months with methimazole through October 1, 1974.

In late October she had a second episode of tremor, dizziness, and *left-sided* weakness, as well as insomnia that persisted for several weeks. She was seen briefly at another hospital and given 10 mg of diazepam for sleep. No physical abnormalities were found at the time of that examination. Subsequently she became afraid to go anywhere without her husband, although she was repeatedly reassured and told that nothing was medically wrong. In February 1975, she had a hysterectomy in a suburban hospital for "fibroids." In May 1975, after an episode of weakness, tremor, and dizziness, she was admitted to another hospital and told she had had a slight stroke. She had lost 9.1 kg in 6 months and developed transient *right-sided* weakness on the day of admission. After discharge, she again became anxious and consulted another New York City neurologist, who could find no evidence of a stroke. She also consulted an internist who could find no evidence of thyroid, heart, or vascular disease.

In July 1975, on referral by her initial physician, she consulted a psychiatrist who prescribed diazepam and amitriptyline. Her three sessions with him were stormy. She reported being told she was the angriest woman the psychiatrist had ever seen, and stopped treatment after the evaluation sessions.

Her internist, who continued to see her every 6 months, referred her to me for evaluation in late September 1975. When I first saw her, the patient was obviously depressed, anxious, and angry. Mental status exam did not reveal any thought disorder or impaired judgment. There were no difficulties in orientation, counting, or calculation. The patient's identification with the members of her family who had suffered strokes was obvious and seemed to account for part of her overreaction to the physical symptoms. Her anger at her physicians and confusion about her illness prevailed. I saw her twice weekly at first and then biweekly for 40 sessions over the next 15 months before the diagnosis of atrial myxoma was finally made. The psychological issues of import included her past conflict with her father over her authorship of his will, conflict with her husband over their joint law practice, and finally by a move to a new home in July 1976.

During the period of psychotherapy, the patient regained 4.54 kg and returned to work for 2- and 3-month periods before her symptoms in December. Her appetite, sleep, and mood had generally improved, but the nocturnal palpitations and "anxiety" persisted. She was reassured by her internist on three different occasions that she did not have significant cerebrovascular disease or notable heart disease. During my summer vacation, the patient had a moderate recurrence of insomnia and again consulted the internist.

Hospital admission. During a Christmas party and after a few drinks, the patient's husband frantically called me, saying his wife had had a stroke and was being admitted to a hospital in New Jersey. I visited the hospital and examined the patient. It was obvious that all her symptoms had cleared again within hours. Also, there was apparently no new psychic stress. Another medical conflict ensued: the physicians insisted

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that she have prompt bilateral arteriograms and heparin; the patient refused and I supported her decision. A plan was arranged for her to have no invasive tests and to be transferred to New York City. In the waiting period, an additional cardiac consultation was arranged. At that time, the suggestion of a myxoma was considered and an echocardiogram was done.

Angiocardiography at Columbia-Presbyterian Medical Center was confirmatory. The patient underwent surgery on January 6, 1977, and a 3×4×2 cm pedunculated atrial myxoma arising from a 1½ cm base just at the lower end of the left side of the foramen ovale was removed; a ball valve-like mechanism was apparent. Pathological examination showed a classical atrial myxoma; 44 minutes of cardiopulmonary bypass were required for excision.

Discussion

Atrial myxoma is the most common (50%) intracavitary cardiac tumor (1). The first successful angiographic diagnosis of myxoma was made in 1951 (2). By 1965, there were 350 recorded cases in the literature, ranging from infants to the elderly; currently there are well over 1,000. Approximately 75% were seen as occurring in the left atrium (3) and represented 35%–50% of primary cardiac tumors. Most occurred in patients between the ages of 30 and 50. The effects of the tumors (4) may be due to 1) primarily mechanical obstructive symptoms that may be intermittent, 2) embolic phenomena, or 3) nonspecific symptoms such as weight loss.

This patient presented all of these manifestations. Syncope, when the patient is upright, is common with intracavitary cardiac tumors, as are dyspnea and atypical chest pains. This patient's difficulties resulted in nocturnal palpitations due to mechanical factors associated with her tumor as well as to emotional concomitants. Even after surgery, some mild nocturnal anxiety persists. However, the palpitations that resulted from obstructive effects of her tumor are no longer present. The patient no longer demands her husband's constant presence. The coincidence of psychiatric factors in this case obviously complicated the diagnosis.

The patient continued psychotherapy and antidepressant medication after surgery. Her early responsiveness to the psychotherapy made the diagnosis more difficult early in treatment. The delay in diagnosis was due in part to the intermittent nature of the symptoms and the difficulty in hearing the cardiac sounds; the crucial diagnostic clue was the tumor on a pedicle creating a unique noise when the patient was in a particular position. The changing side of the weakness (first left, then right) was an additional missed clue misinterpreted as psychological.

I was perplexed by the persistent palpitations and the physical symptoms that resulted in the periodic visits to the internist, who had never seen a case of myxoma. Echocardiography might have been used sooner had the course or physical findings been different.

It is important for consultants to consider atrial myxoma in patients with transient syncopal attacks, episodic weakness, and/or atypical nocturnal palpitations. Echocardiography (5, 6) adds special crucial evidence in the 30–50 age group in the presence of puzzling, transient, ischemic-like attacks.

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Patient Responsibility and the AMA Discharge: A One-Year Follow-Up Study

BY DAVID J. WITHERSTY, M.D.

Outcome studies of discharges against medical advice (AMA) have described posthospital adjustment as being the same for AMA patients and non-AMA patients (1, 2). A recent study of AMA discharges using a matched non-AMA control group found little difference in posthospital adjustment and suggested that AMA discharges might be therapeutic (3).

The Behavioral Medicine Inpatient Service at the West Virginia University Hospital (described elsewhere, see references 4–6) offers a unique opportunity to study the AMA discharge. The service is totally open, with no seclusion rooms or locked doors, and admission is voluntary. Patients are expected to take responsibility for making decisions regarding admission and discharge. Furthermore, they are told that they are expected to take responsibility for making

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changes in their maladaptive behavioral patterns.

Under such conditions, one would expect AMA discharges to be high. This is the case—approximately 20% of the patients leave against medical advice. Although results of two one-year follow-up studies suggest that it is therapeutic to place primary responsibility for admission and discharge decisions as well as behavioral change with the patient (5, 6), one must question what happens to those patients who elect to leave the unit against medical advice.

The remainder of this article will present data that were obtained in a one-year follow up study of AMA patients compared with data obtained in previous studies of non-AMA patients.

During the period between March 1975 and August 1975, 480 patients were admitted to the inpatient service: 99 patients left against medical advice during that time. The age of AMA patients ranged from 15 to 68 years (mean=32.5). The average number of days on the unit was 4.7, compared with an average of 14 days for other patients. Of the 99 AMA patients, 38 had been hospitalized on the inpatient service of the West Virginia University Hospital before the present admission. Thirty patients had been hospitalized previously on psychiatric units at other hospitals and so had no previous hospitalizations elsewhere (data on admissions to other facilities were unavailable for 9 patients).

Prior to discharge 71 of the 99 patients had attended at least one session of group therapy, the primary treatment modality on the unit, and 23 had not (attendance data were unavailable for 5 patients).

It is the policy of the inpatient service to use multiple diagnostic labels for the purpose of charting. Diagnostic labels for the 99 AMA patients included anxiety or depressive neurosis (N=65), psychotic disorders (N=17), alcohol or drug abuse (N=38), personality disorders (N=10), transient situational adjustment reactions (N=10), psychophysiological disorders (N=2), organic brain syndrome (N=2), and mental retardation (N=5). These diagnoses are comparable to those of our usual patient population, with the exception of a higher number of alcohol and drug abusers.

Data were collected from 33 female and 28 male AMA patients contacted by telephone 1 year after their discharges. There was a recidivism rate of 52.5% during the year after discharge. Previous studies had revealed recidivism rates of 20.9% and 26% for non-AMA patients (5, 6). Of those AMA patients who were rehospitalized, 73.5% returned to our service and 26.5% were admitted to psychiatric units in other hospitals.

Patients were asked if they were currently seeing someone for help on an outpatient basis; 21% refused to answer or were uncertain. Of those who answered the question, 62.5% were obtaining outpatient help. The two previous studies of non-AMA patients found rates of 54.8% and 61% for outpatient follow-up care (5, 6).

To determine the attitude of AMA patients toward our inpatient service one year after discharge, we asked whether they would be willing to return to the unit if it became necessary to do so: 52.5% answered yes, 14.8% said no, and 32.7% were uncertain.

Almost three-fourths (73%) of the patients thought that their condition had improved since discharge, 1% stated that they had remained the same, and 26% said that their condition had worsened.

The one-year follow-up data on AMA discharges seem to suggest that if patients are given the responsibility to choose the time of discharge, approximately 20% will elect to leave against the advice of their physician. We believed that hospitalization was initially indicated for all of these patients, and it was gratifying that over 50% of the AMA patients sought rehospitalization and that 62.5% of those who responded were engaged in outpatient follow-up care. It is interesting that of those patients who were rehospitalized during the year, 73.5% returned on a voluntary basis to the West Virginia University Hospital inpatient unit.

Our data do not support previously published claims that individuals discharged against medical advice have a posthospital adjustment similar to non-AMA discharges in that our AMA patients had a much higher recidivism rate. Outpatient follow-up treatment was approximately the same for our non-AMA patients and AMA patients.

We found a higher proportion of drug and alcohol abuse in our AMA patients compared with non-AMA patients, a finding that is consistent with previous reports (7, 8).

This study did not answer—and was not intended to answer—the question of whether AMA discharges are therapeutic. However, the data do raise several questions. Is the rather high AMA discharge rate a patient's way of determining whether or not he or she *is* in control of the treatment program? Do patients have to test our system to see if we are really sincere when we advise them that they have the responsibility for admission and discharge decisions as well as for making changes in their own behavior? One could propose this explanation in view of the fact that almost three-fourths of the AMA patients who were rehospitalized chose to return to our unit. One must also consider the possibility that the AMA discharge is an expression of anger and hostility, or even ambivalence, as some authors have suggested (8).

Many of the patients who leave our service against medical advice do eventually obtain psychiatric help either as inpatients or outpatients. Furthermore, it has been our impression, supported by the two previous one-year follow-up studies (5, 6), that giving patients the opportunity to play a significant part in admission and discharge decisions helps to establish a therapeutic inpatient milieu. Further evaluation of the effect of AMA discharges is needed.

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Degraded Protein-Containing Food and Monoamine Oxidase Inhibitors

BY JULIAN LIEB, M.B., B.CH.

Although it is recognized that patients on monoamine oxidase inhibitors (MAOIs) should have some dietary restrictions, there is as yet no generally accepted diet. Reports by Horwitz and associates (1) in 1964 and Blackwell and associates (2) in 1967 that correlated hypertensive reactions in patients on MAOIs with the ingestion of tyramine-containing foods led to the development of MAOI diets that restrict these foods to a greater or lesser extent.

As early as 1909 Barger and Walpole (3), in a classical paper, reported the identification of isoamylamine as a "pressor constituent of putrid meat." In a recent report, Stewart (4) suggested that any protein-containing food that has undergone degradation could present a hazard to patients on MAOIs. This probably results from the fact that tyramine is produced by decarboxylation from tyrosine, which is derived from protein by proteolysis or hydrolysis.

I have recently observed 3 patients with hypertensive reactions in which the offending agent appears to have been denatured protein rather than the specific items that usually appear on MAOI diet lists.

Case Reports

Patient 1, a 48-year-old woman, was treated with 30 mg/day of tranylcypromine for a refractory bipolar depression. She reported a severe headache approximately 2 hours after eating a sandwich consisting of rye bread, butter, lettuce, and tuna fish from a tin that had been opened for 2 days and had not been refrigerated immediately after opening. Her blood pressure rose from a pretreatment level of 120/78 to 150/100 and subsided spontaneously over a period of 90 minutes. This patient experienced a second reaction some weeks later after drinking a cup of coffee with cream. Her blood pressure rose to 170/120, but she became normotensive within 30 minutes of oral administration of 50 mg of thioridazine.

Patient 2 is a 42-year-old schoolteacher who was treated with 60 mg/day of tranylcypromine for an atypical depression with prominent features of adult brain dysfunction. She reported a severe headache 1 hour after eating a cake-covered ice cream bar. The ice cream had been removed from the school refrigerator a few days previously and then returned to the freezer about an hour later. The patient stated that the inside of the freezer had a "peculiar, rancid smell." Her blood pressure rose to 160/115 from a pretreatment reading of 115/78 and normalized within 60 minutes without active treatment.

Patient 3 is a 57-year-old dentist who was treated with 30 mg/day tranylcypromine for a refractory bipolar depression. One hour after a lunch of beef chow mein he experienced a severe throbbing headache and had a blood pressure reading of 150/85 a half-hour later (pretreatment blood pressure was 110/70). He became normotensive within an hour and a half without active treatment. The chow mein consisted of bean sprouts, water chestnuts, ground beef, and soy sauce. The ground beef was prepared from a roast that had been cooked 3 days earlier.

Discussion

In case 1, the first reaction was probably due to previously opened canned fish and the second reaction to cream.[1] The reaction of patient 2 seems to have been due to rancid ice cream. In the third case the offending agent was either the beef or the soy sauce. Stewart (4)

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[1]There was a report in 1959 of one patient who suffered "jitteriness" after drinking coffee while on an MAOI (5), but in view of the widespread consumption of coffee, it is highly unlikely that coffee is an offending agent.

reported that one sample of soy sauce was found to contain only 1.76 μg/g of tyramine and this figure, together with the paucity of reports of reactions to soy sauce, implies that an association is unlikely (4, 6). Stewart thinks that degraded protein in the meat seems a more likely explanation.

The four reactions reported in this paper lend support to Stewart's conclusion that the foods that have been reported to cause hypertensive reactions may have in common the degradation of a protein constituent, and that only fresh food or freshly prepared frozen or canned food should be eaten by patients on MAOIs (4).

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Phenothiazine-Induced Priapism: A Case Report

BY JEROME I. GOTTLIEB, M.D., AND THOMAS LUSTBERG, M.D.

Priapism is defined as "the persistent abnormal erection of the penis, which usually occurs without sexual desire" (1). The condition is usually quite painful and often requires emergency urological procedures, such as draining and the use of shunts, to prevent permanent physiological impotence (2). It has been suggested that priapism can occur as a result of phenothiazine therapy (3). Although priapism secondary to the use of several different phenothiazines has been reported in the urological literature (3), we are aware of no such reports in the psychiatric literature.

The following case supports the concept of priapism as a complication of phenothiazine therapy. It is also the first report we know of that correlates priapism and mesoridazine therapy. In the discussion that follows, we will explore the physiological mechanism of phenothiazine-induced priapism.

Case Report

Mr. A, a 28-year-old chronic schizophrenic, reported a history of normal sexual functioning before July 1976. He had been treated intermittently over an 8-year period with chlorpromazine, 400–1000 mg q.d., with no known drug complications. In July 1976, the patient lost his job and moved to another city. He was taken off chlorpromazine and treated with mesoridazine, 400 mg q.d., for 3 months.

Three weeks after he started the mesoridazine, the patient had his first episode of priapism, which occurred after intercourse and resolved spontaneously within 12 hours. Following this incident, he reported having mild difficulties maintaining an erection, although he experienced no problems with ejaculation or orgasm.

On the day he was admitted to the hospital, the patient had taken approximately 1 g of mesoridazine. Several hours later he had had intercourse and had developed a painful and persistent erection, which eventually necessitated an emergency intracorporal shunt procedure. The operation was performed, but the patient became impotent, which led to a later psychiatric hospitalization for depression. (He was taken off mesoridazine.) At that time, he verbalized concerns about his impotence, loss of masculinity, and rejection by his girlfriend.

Mr. A had a history of multiple psychiatric hospitalizations for treatment of chronic schizophrenia and depression. He also had frequent concerns about his masculinity. In addition, he had a long history of drug abuse, including frequent use of cocaine, heroin, and amphetamines. He denied penile injections. His medical history is unremarkable, and laboratory studies revealed no evidence of hemoglobinopathies, platelet malfunction, or blood dyscrasias.

Discussion

The major tranquilizers have been implicated in several different drug complications related to sexual function. Theories involving various neurogenic pathways have been promulgated to explain impotence associated with antipsychotic medications. Before reviewing these, we will summarize current theories of the physiology of male sexual activity.

Psychic or local stimuli send impulses to the upper portion of the lumbar cord, where reflexes are set up. Parasympathetic outflow from S2-S4 then causes re-

When this work was done, Dr. Gottlieb was Chief Resident, Department of Psychiatry, Georgetown University Hospital, where Dr. Lustberg is Resident. Dr. Gottlieb is now in private practice at 8316 Arlington Blvd., Suite 516, Fairfax, Va. 22030; he is also Staff Psychiatrist at Fairfax Hospital and at the Woodburn Community Mental Health Center, Annandale, Va.

laxation of arterioles to the corpora of the penis. The cavernosus bodies are engorged under pressure, resulting in an erection. Sensory impulses from the glans penis then travel back to the sacral cord to the integrating center in the upper portion of the lumbar cord. These impulses subsequently trigger a sympathetic discharge that causes secretions from the seminal vesicles, ejaculatory ducts, and prostate. Somatic nerves from S2-S4 stimulate spasmodic contraction of muscles surrounding the urethra and pelvic floor, resulting in ejaculation. By stimulating contraction of the arterioles and relaxation of the venules of the penis, sympathetic nerves are responsible for detumescence (3, 4).

Phenothiazines have strong peripheral anticholinergic and adrenergic blockade properties. Failure to obtain or maintain an erection (under parasympathetic control) may result from the former. It has been postulated that alpha-adrenergic blockade may be responsible for retrograde ejaculation (5).

In priapism, two mechanisms may operate. First, by preventing the firing of sympathetic discharge, the phenothiazine-related alpha-adrenergic blockade may interfere with detumescence. Second, phenothiazines may have a contributing CNS effect (3). Hinman (6) postulated, for example, that prolonged stimulation of psychic centers may lead to continual venous stasis, which would increase the viscosity of the blood and result in subsequent edema of the trabeculae. Priapism would then occur and would be perpetuated by stimulation of a positive feedback system.

Thioridazine has been associated with sexual dysfunction (7), and chlorpromazine has been indicated as a possible etiological agent in priapism (9). In the case we have presented, the ingestion of 1 g of mesoridazine, a besylate salt of a thioridazine metabolite, was related temporally to the development of priapism. The possible mechanisms we have proposed warrant further investigation.

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LETTERS TO THE EDITOR

This section contains reactions to Journal *articles, statements of opinion, comments on Association activities, requests for information, etc. Letters should be submitted in duplicate to the Editor, who makes all decisions regarding publication. Letters must be typed double-spaced throughout and should not contain more than 500 words and 5 pertinent references. Criticisms of published articles will automatically be sent to the author(s) for response. Letters will be edited for clarity and conformance with* Journal *style. We regret that we cannot inform writers of the disposition of letters or return those which are not printed.*

Ethics in Alcoholism Research

SIR: I would like to express the considerable uneasiness I felt after reading "The Effect of Alcohol and Smoking on Testosterone Function and Aggression in Chronic Alcoholics," by Harold Persky, Ph.D., and associates (June 1977 issue).

The study they reported is of some slight interest, but the manner in which it was conducted is profoundly disturbing. I cannot help but bristle at the notion that a number of men who were addicted to alcohol were supplied with virtually unlimited quantities of that drug by physicians working in a public hospital with the support of the National Institute of Mental Health and the National Institutes of Health. There is nothing in the article to indicate that any attempt was made to direct these men toward help with their addiction. Granted that this was a research study, did the physicians involved in it make any effort to improve the health of their subjects? All of the subjects underwent a one-week period of enforced sobriety and abstinence from cigarettes. Were all of these men, regardless of their research or control group assignment, given the opportunity to continue that sobriety and abstinence *after* one week? I would contend that each of them should have been actively encouraged to do so.

I know this piece of research must have gotten past the appropriate "human rights in research" committee, but the article does not make it clear how that could have happened.

I hope that there are some explanations available. Otherwise, one must wonder what has happened to *primum non nocere*.

MARTIN GOLDBERG, M.D.
Philadelphia, Pa.

Dr. Persky and Associates Reply

SIR: We are clearly concerned about the welfare of alcoholic patients. In fact, two of us spend most of our time managing large treatment programs for addicted individuals. Furthermore, as noted in the paper, all of the volunteers were carefully screened, and those whose participation might involve medical or psychiatric risk were excluded. Excluded individuals were offered treatment and, of course, those accepted for the study were strongly urged to enter treatment after completion of the study. Treatment was both medical (for example, detection of unexpected diabetes or hypertension) and psychiatric. The patients were quite appreciative, and several maintained contact with the clinicians long after the conclusion of the study.

The study was not "gotten past" a human subjects committee. It was carefully reviewed by a committee at our university and by an independent committee at the hospital housing the research center. Both committees made helpful suggestions concerning the consent forms and determined that participation was of potential benefit to the subjects as well as to persons in general afflicted with this condition.

Dr. Goldberg seems to be upset primarily by the prospect of giving a drug to a subject under controlled conditions. This procedure is the only way that actual drug effects can be determined and has been generally accepted by the scientific and clinical community, as evidenced by the numerous publications describing studies of this nature over the past decade. We strongly feel that clinicians who are truly concerned about their patients will want to obtain valid information about the substances patients ingest. Information based on subjective impressions and historical recollection can be quite misleading. It is for this reason that studies of the present type are essential if we are to advance our understanding and treatment of addictive diseases.

HAROLD PERSKY, PH.D.
CHARLES P. O'BRIEN, M.D., PH.D.
ERIC FINE, M.D.
Philadelphia, Pa.

More "Secondary Mania"?

SIR: I was interested in "Secondary Mania? A Case Report" by Kenneth N. Wiesert, M.D., and Hugh C. Hendrie, M.D., Ch.B. (Clinical and Research Report, August 1977 issue) because I have recently struggled with a similar case.

A 40-year-old married office worker was admitted to a community mental hospital two weeks after the birth of her first grandchild. The patient was referred from an emergency room where an interstitial pneumonia had been diagnosed. She was hyperactive, irritable, aggressive, and argumentative, with pressured speech. She said her husband was trying to poison her. She appeared drowsy at times, but this seemed secondary to her report that she had not slept for 3 days.

Her family said she had had two prior episodes of retarded depression, which had cleared without hospitalization. They said the patient also had episodes of hyperactivity and insomnia, during which she became restless and cleaned and

washed constantly. Her father had been hospitalized repeatedly for depression, and a sister and niece had been treated for retarded depressions.

After admission, the patient experienced the somatic delusion that she was in labor. However, delusions and insomnia cleared after 48 hours of treatment with lithium carbonate and haloperidol. She remained hyperactive, and was discharged with a diagnosis of bipolar affective disorder. The manic episode was felt to have been precipitated by a "grandmother reaction."

After her discharge, the following information was obtained. The patient had a severe akathisia secondary to haloperidol that complicated assessment of activity level. Two days before her admission, she had started a course of tetracycline. She had never taken this drug before, and it was discontinued on hospitalization. Before her admission and during the first few days of hospitalization, she experienced vivid waking dreams. In the hospital, she had been afraid to disclose this symptom. The previous "depressions" had coincided with two episodes of interstitial pneumonia.

She was withdrawn from psychotropics and continues to be followed by an internist. One year later, she manifests an obsessive-compulsive personality but functions well and has shown no mood swings.

It is difficult to say whether this is "secondary mania" or simply a difficult diagnostic situation. Two senior psychiatrists were convinced that this was a manic patient. Affective disorder, independent of a toxic precipitant, may eventually surface.

JEAN GOODWIN, M.D., M.P.H.
Albuquerque, N.M.

Structural Change in Tardive Dyskinesia

SIR: In regard to the "Special Section-Tardive Dyskinesia" (July 1977 issue) it seems to me there is a rather plausible explanation for the consistent finding that apomorphine does not worsen tardive dyskinesia, but amphetamine does. (I refer to the papers by Robert C. Smith, M.D., Ph.D., and associates; John W. Crayton, M.D., and associates; and Bernard J. Carroll, M.B., B.S., Ph.D., and associates.)

Since tardive dyskinesia occurs primarily with long-term antipsychotic drug use, it seems likely that it involves a structural change in the postsynaptic receptor, a change that does not reverse fully when the antipsychotic agent is withdrawn. This is supported by the fact that tardive dyskinesia occurs more often in elderly patients, since aging in general leads to decreased tissue plasticity and resiliency. This hypothesized structural change apparently makes the receptor more sensitive to dopamine (which is released by amphetamine) but less sensitive to apomorphine. It may originally be a compensatory change, i.e., an attempt by the neuron to circumvent the dopamine-blocking action of neuroleptics. It is not surprising that a structural change could affect which chemical moities would interact with and stimulate a receptor.

This theory of a structural change in the receptor causing tardive dyskinesia can be further evaluated by biochemical studies involving such dopamine agonists as piribedil and bromocriptine, but it can be verified only by histological techniques. One would speculate that any such structural change would occur in some but not all dopaminergic neurons, since dopamine receptors in different parts of the brain appear to be affected differently by long-term neuroleptic use (1–3). The occurrence of a structural change in the receptor seems to me a more likely explanation of why tardive dyskinesia does not worsen with apomorphine than does the explanation involving presynaptic receptors.

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JEFFREY MATTES, M.D.
Glen Oaks, N.Y.

More on the Use of Manganese in Dyskinesia

SIR: We noted with interest the work of Richard A. Kunin, M.D., in the use of manganese in tardive dyskinesia (letter to the editor, January 1976 issue). We have had six patients with tardive dyskinesia who responded extremely well to 20 mg of manganese chelate t.i.d. The treatment seemed to work best in those who manifested fistulations of the tongue, with improvement of symptoms in 24 hours and remission within several days. Two of the patients stopped their medication either on their own or by error, and the dyskinesia symptoms recurred. Both patients again had remission of symptoms within several days after restarting the medication. Manganese chelate was also used with six other patients who had various extrapyramidal symptoms but no fistulations of the tongue, but none showed any improvement in the symptoms.

JOSEPH P. NORRIS, M.D.
ROBERT E. SAMS, M.D.
Parkersburg, W.Va.

Ganser-like Signs in Carbon Monoxide Encephalopathy

SIR: The manifold array and fluctuating course of neuropsychiatric symptoms in carbon monoxide poisoning have been well described. Ginsburg and Romano (1) warned that "In particular the rapid changing of apraxias, agnosias, astasia-abasia, and the amnestic-confabulatory state often contributes to a false diagnosis of hysteria." Garland and Pearce (2) noted that "transient affective disorders of a depressive type were seen in the early stages." Many cases of carbon monoxide poisoning are results of suicide attempts, so misdiagnosis of depression may occur. Treatment with ECT may be harmful when there is brain damage by carbon monoxide.

We wish to report another potential diagnostic pitfall in cases of carbon monoxide intoxication: misinterpretation of signs resembling those of the Ganser state, classically described in prisoners awaiting trial (see reference 3 for a full description).

Our patient, a 20-year-old man awaiting trial, suffered smoke inhalation in a fire at the jail where he was being held.

Psychiatric consultation was requested 5 days after the fire because of his continued abnormalities of mental status. His gaze was vacant, his attention wandered, and speech was slurred. He answered only simple questions. He could identify a pen, watch, and key, count from 1 to 10 forward and backward, feed himself, and follow simple commands (e.g., to touch his hand to his nose). He stated he was in a hospital.

Seven days after the fire he called a pen a pencil and could not identify a watch or key. He counted from 1 to 10 but could not count backward, did not know where he was, and was unable to feed himself. Two days later he was unable to identify a pen or watch. He called a key a motor (it was a car key). He could write his name, but could not copy a circle or say what it was. He walked with agility and could follow directions as to when to return and where to sit. When asked whether he was right or left-handed, he answered, "both" (he is right-handed).

At that time, he gradually developed choreoathetoid movements and pseudobulbar speech. An EEG showed nonspecific generalized disorganization and slowing, and a CAT scan showed focal decrease in density in the region of the basal ganglia bilaterally. It is worth noting that as these signs developed, his perceptual and cognitive abilities began to improve. He is now gradually improving in all areas.

The variability in this man's mental status, with agnosia, apraxia, and "near-miss" answers, led us to consider the diagnosis of a Ganser state. However, as his course unfolded, evidence of his severe, multifocal brain injury became apparent. In cases of carbon monoxide encephalopathy, extreme caution is necessary before consideration is given to any alternative or additional diagnoses.

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JOSEPH MCEVOY M.D.
THOMAS CAMPBELL, M.D.
Nashville, Tenn.

Death with Dignity and Survivor Guilt

SIR: I would like to present a clinical vignette that may illustrate the trauma which can result from the decision to permit discontinuation of extraordinary life-saving measures for one's parent.

A 40-year-old woman was recently brought to the emergency room for treatment of a self-inflicted gunshot wound to the right axilla. The bullet grazed the area of the brachial plexus, resulting in a paresis of the right upper extremity.

The patient reported that she was unaware of any precipitating factors that might have led to her decision to shoot herself. She seemed to be an articulate, intelligent woman and held a responsible position with a large department store. She stated that she had noted increasing feelings of depression for several months.

The interview revealed that in May 1977, the patient had been summoned to an intensive care unit in West Virginia, where her elderly mother had been sustained on a life support appartus for the preceding 9 days. The facts of her mother's case were clear, and attempts at resuscitation were futile. After consideration of the evidence, the patient signed a document authorizing the hospital staff to discontinue use of the life-support equipment.

Although great strides have been made in obtaining death with dignity for the hopelessly ill, the assumption is often made that the ancient struggle against the wish for the parent's death has dissolved. The burden may be even greater for the obsessional patient who has masked his or her affect behind an overdetermined rational exterior. We must realize the potential for guilt in a person who has acted out his most feared impulse: homicide.

ALAN E. RAINESS, M.D.
Silver Spring, Md.

More on Drug-Induced Blurred Vision

SIR: I concur with the observations about blurred vision and its treatment with pilocarpine made by J. Gary Carter, M.D., and associates (letter to the editor, August 1977 issue). I have encountered this problem in a small number of patients I have treated over the years with phenothiazines, tricyclic antidepressants, MAO inhibitors, and butryophenones. This complication has occurred in about 10%–20% of 321 patients with Gilles de la Tourette's syndrome who were treated with varying dosages of haloperidol. The effect is dosage-related but may occur with small dosages. Although the problem usually disappears within 6 to 12 months, it is very distressing to patients who are students, accountants, jewelers, etc.

An alternative to the use of daily pilocarpine that has been effective for most of our patients is the prescription of inexpensive and temporary corrective lenses that can be discarded when the complication disappears.

ARTHUR K. SHAPIRO, M.D.
New York, N.Y.

Another Approach to Self-Induced Depersonalization

SIR: The potential ill-effects of popular techniques for inducing altered states of consciousness (ASCs) have been recognized longer than was implied by Raymond B. Kennedy, Jr., M.D., in "Self-Induced Depersonalization Syndrome" (November 1976 issue). Dr. Kennedy was correct to complain of the limited interest the Western psychiatric literature has shown in these phenomena. However, the varieties of psychological disturbance that may be caused by naïve abuse of consciousness-altering techniques are more extensive than his title suggests.

A wide range of techniques (including both spiritual and "commercial" meditation, hypnosis, autogenic training, Zen, Yoga, and states induced by various drugs, prayer, fasting, reverie, and sensory deprivation) have been used to induce similar states, and all have the potential to produce negative as well as positive effects (1).

Zen recognizes such effects as *makyo* and *Zen-byo* (Zen-illness). Zen aims at a full enlightenment and awakening of the personality, but this state of *satori* is different and more humble and realistic than the delusory self-induced trance-states and depersonalization/derealization states known as *makyo*. Disciples are taught that they have to pass through a

sequence of major and minor *satoris*, each representing the integration of emerging subconscious material into the personality. Over-valuation of and attachment to a small *satori* is *makyo*.

Psychotic symptoms, megalomaniacal and omnipotent delusions, aggression, and even suicide are recognized as occasional complications of the *makyo* state in Eastern literature (1). A recent case report (2) described a middle-aged woman who developed a severe dysphoric episode after initiation into Transcendental Meditation. Takaishi and others (cited in reference 1) have described disturbances in Japanese high school students who had "played" with hypnosis, including difficulty awakening from trance, continuation of daydreaming, and unreality states.

Meditation involves a temporary inhibition of conscious ideation. This can allow the release of unconscious and otherwise repressed material that should be mildly observed and not identified with or cathected. The naïve and inadequately trained meditator may fail to distinguish between the consciousness/self who perceives and the conscious ideas that are perceived, mistaking a part of the experience for the actual goal of psychotherapy or meditation.

While meditation techniques can be of considerable personal and clinical value, their indiscriminate merchandising, which has been a notable feature of the "consciousness movement," has significant risks. There should be some selectivity with regard to those who are trained in such techniques, since an appropriate degree of ego strength is needed. Individuals with hysterical personalities may be more prone to develop a dissociative state in relation to an ASC, and those with borderline states may also find ASCs disturbing. Education about the goals and effects of the techniques and guidance with regard to the nature and management of such experiences as *makyo* would make the experience safer.

Dr. Kennedy described how reassurance transformed frightening depersonalization into ego-syntonic occurrences and proposed that patients in this condition should be taught to accept themselves and their depersonalization uncritically. It is important not to encourage cathexis of the experiences or overidentification with them. The Zen technique for dealing with *makyo* is to stay indifferent to the phenomena, recognizing them as products of the ego/self but merely observing them neutrally, not considering them as real. Other techniques such as concentration on breathing or posture have also been used, and simple autogenic and muscle-relaxation routines can be useful.

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MICHAEL A. SIMPSON, M.D.
London, England

Dr. Kennedy Replies

SIR: I did not mean to imply that such states as depersonalization are to be cathected, which seems to be Dr. Simpson's understanding of my article. He is quite right to emphasize that these phenomena should be transitory. In the cases reported, one person was simply instructed to discontinue meditation and the other was referred (not by myself) to the Indian Guru Swami Muktanada Paramahansa, a man with considerable knowledge of such situations.

Further, I did not mean to imply that my report of such situations was unique. The world literature on metaphysics and allied fields including meditation is replete with such topics, far too numerous to mention, many of which antedate the birth of Christ. The psychiatric literature is less well endowed, however, and one hopes the in-press reference cited by Dr. Simpson will add to the psychiatric literature on such subjects.

I wish to add one caveat in reference to Dr. Simpson's letter, however. He has used several terms that are not a part of the psychiatric terminology and that remain quite subjective and largely theoretical for me—and I suspect for most people who have done work in such areas. Such subjective terms as *satori* and *makyo* occupy an appropriate place in religious and metaphysical writings but do not seem to me to have a legitimate place in Western psychiatry, which is already plagued with the struggle to develop a scientific terminology that will allow us to approach a relative form of objectivity. A precise language is the basis for any reasonable scientific endeavor, and the Russian mystic and metaphysician Peter Ouspensky (1, 2) designated this as one of the cornerstones in any sound metaphysical system designed to study consciousness. Ouspensky also notes that such precise languages are precise only for the time and place of the particular system with which they are used and are not transposable. As scientists, we would do well to heed such advice lest we create (or add to) our own Tower of Babel.

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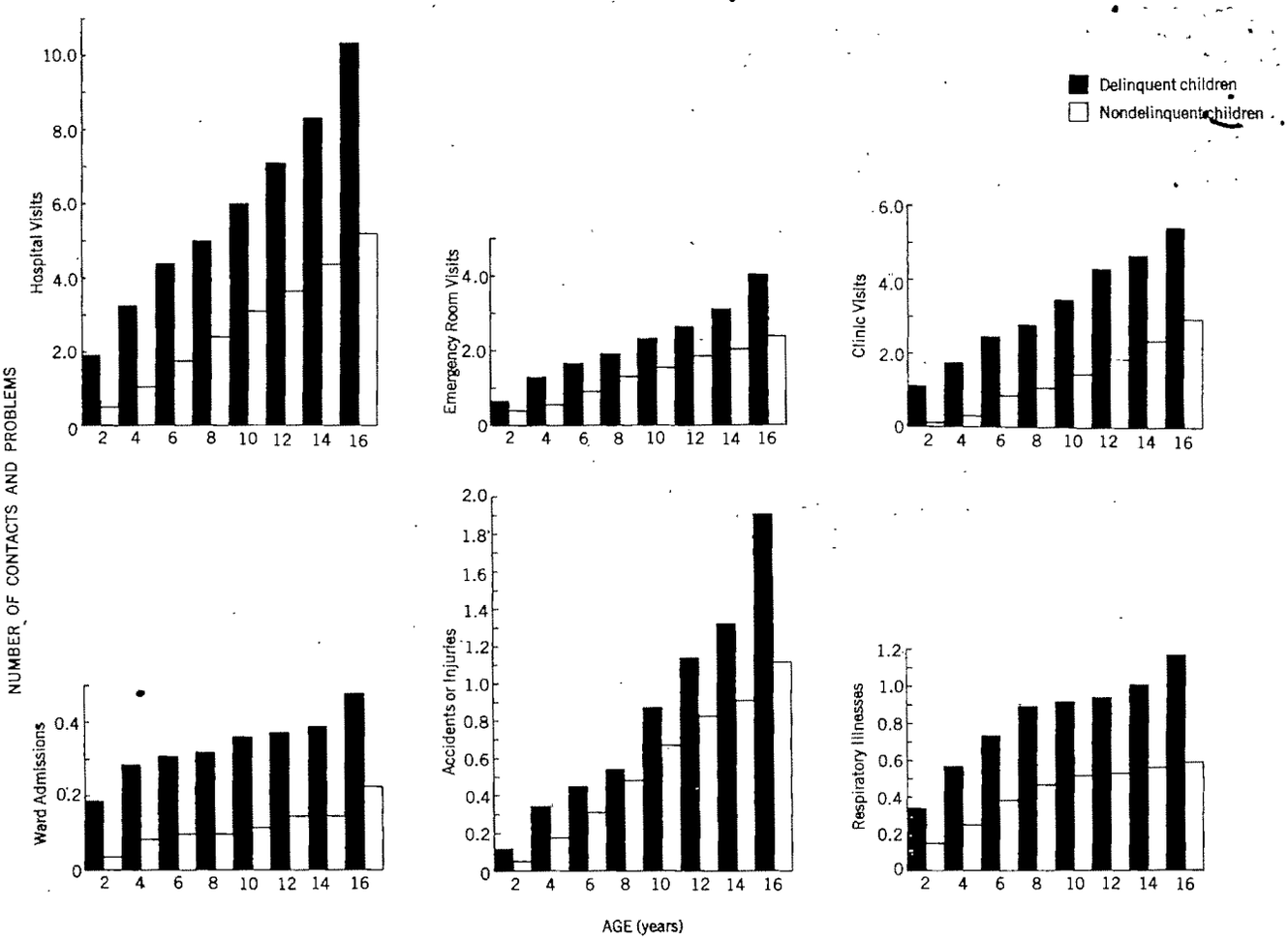
Psychiatrists in CMHCs

SIR: I have just completed a period as medical director of an urban community mental health center with a catchment area of 200,000 people (predominantly blue-collar workers and their families). I feel compelled to respond to the excellent article by James S. Eaton, Jr., M.D., and Leonard S. Goldstein, M.D., "Psychiatry in Crisis" (June 1977 issue).

I believe that one of the reasons why the number of psychiatrists serving in community mental health centers has decreased in recent years is that mental health administrators—most of them nonphysicians—seem to feel that nonphysician members of the mental health team can do the job of therapy just as well or better than psychiatrists and do it for less money. Therefore, from a cost-efficiency standpoint, it seems to such administrators more logical to hire less expensive mental health professionals rather than "high-priced" psychiatrists.

The potential fallacy of this kind of administrative thinking becomes apparent only in the long run (if ever), and through the results obtained, in terms of the importance of accurate

Figure 1
Cumulative Hospital Contacts and Medical Problems of Delinquent and Nondelinquent Children Throughout Childhood



BOOK REVIEWS

You Are Not Alone: Understanding and Dealing with Mental Illness. A Guide for Patients, Families, Doctors and Other Professionals, *by Clara Claiborne Park with Leon N. Shapiro, M.D. Boston, Mass., Atlantic-Little, Brown, 1976, 480 pp., $15.00.*

This sizable volume was four years in the making and was written to tell the readers what they need to know about mental illnesses and the problems of living that usually bring people to mental health services. Ms. Park is a dedicated woman who has known the frustrations of seeking help for an ill child and who is now intent on helping people who do not know what to do or where to go when a loved one becomes ill, and Dr. Shapiro is a knowledgeable psychiatrist. Ms. Park learned from "unexpected people in unexpected places what you can do especially when you realize that you are not alone"; therefore, this is the central theme of the book. She found that the mind has many mansions and that the way we see the world is not the only way it can be seen.

The authors are aware of and have looked into every controversy and every advance in the mental health field. Their stated intent is simply to present both sides of the disagreements about controversial subjects, to do this fairly, and to do it in sufficient detail. They introduce various specialists in the mental health field and describe the background, pedigree, and functions of each. They discuss the labeling of illnesses, medical terms, and the medical model. They list various opinions of mental illness and give lucid discussions of all types of illnesses that mental health workers encounter. These are informed, detailed discussions, as are the listings of the various forms of treatment—from the analytic systems of Freud, Jung, and Adler to the newer therapies, including even the radical therapies, which they say "challenge all forms of treatment and find everybody out of step but the patient." This suggests to them "not psychiatry but anti-psychiatry."

The authors explore dynamic therapies, behavior therapies, and group therapies. They posit the reassuring thought that most psychotherapies are more different in theory than they are in practice. "Dynamic or insight therapy," they note, "works to change behavior from the inside out and behavior therapy works from the outside in." They doubt that insight and understanding into the sources of psychological problems in themselves have the power to change things. Self-knowledge, they say, is no guarantee whatever of effective problem solving and may not even be necessary to it.

Wittingly or unwittingly, the authors lean toward the behavioral approach, noting that many of its patients are not of the YAVIS[1] variety; rather, they are often the direct opposite. They feel that because the system is based on fundamental principles of learning behavior it works for everyone—the chronically insane, the brain damaged, the neurotic, and the delinquent.

Somatic treatments are discussed fairly and intelligently—lithium, ECT, and orthomolecular therapy all come under the authors' ken. There is a broad discussion titled "Thinking About Causes" in which the authors range far and wide. They apparently have read everything; their footnotes are as voluminous as their bibliography.

This work reminds us plaintively of the pain caused by the assumption of family blame for a member's illness before all of the facts have been considered. However, the authors view the search for causes of mental upset as important for everyone and delve into these as well.

The second section of the book is concerned with the practical matter of what the community can offer. The search sometimes is disheartening, and the guiding theme the authors offer is never to accept no for an answer: "Go to the real people," they say, i.e., those who know where and how help can be found. They point out that under some circumstances the home can be a therapeutic milieu and families can be cotherapists. The authors bring home to us sharply the burden on the family of dealing with sick loved ones. Frequently the tolerance of the family is high because of interest and guilt feelings about the patient's illness.

Hospitals of various types, "The Law and Your Relative," and various areas of conflict are all sympathetically considered. Part three of the book is titled Outrage—And How to Use It. Much of it is concerned with the hunt for proper placement of children in need of help in the face of bureaucratic school officials. The authors also consider the stress of dealing with the elderly who are mentally ill. The essence of the final chapter, "The Lord Helps Those Who Help Themselves," is that one can get any kind of help needed if he or she is persistent.

If this review seems pedestrian, it should not be, for I enjoyed this as the most complete book of its kind that I have yet encountered. Mind you, there is much I disagree with in it, but that does not subtract from the fact that it is an excellent work. It is well written, moves along smoothly, and there are sprightly little quips in the margins. Appendix A suggests some helpful readings, and Appendix B gives some useful addresses, ranging from that of the American Psychoanalytic Association to that of the *Madness Network News*, with various stops in between. There is no doubt about the fact that all of this information will be of invaluable assistance to families of sick persons—and for doctors, too; I picked up a great deal of information from it myself.

[1]YAVIS is an acronym for ideal patients for insight therapy—Young, Attractive, Verbal, Intelligent, and Successful (1).

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F.J.B.

An Assessment of the Community Mental Health Movement, *edited by Walter E. Barton and Charlotte J. Sanborn. Lexington, Mass., Lexington Books (D.C. Heath and Co.), 1977, 191 pp., $18.50.*

In view of the attention now being paid to the community mental health centers program as a result of the need to recommend changes that might lead to Congressional action and the ongoing activity of the President's Commission on Mental Health, this collection of papers edited by Drs. Barton and Sanborn is timely and informative to those with interest in this area. The publication is the product of a three-day institute held at Dartmouth College in Hanover, N.H., in September 1975. Although editing has delayed its publication until 1977, this does not detract from the value of the papers presented in this volume.

The initial chapters by Drs. David Musto and Jack Ewalt are particularly helpful, not only because they provide a historical context for an assessment of the community mental health center program in its current operational form but also because they correct some of the current mistaken perceptions regarding the original intent of the first Joint Commission on Mental Illness and Health (1955–1960). These chapters also emphasize the importance of understanding the historical perspective before considering major changes in philosophy or programming. An excellent example is provided by Dr. Ewalt in his review of the actions of the first Commission regarding prevention services. With the current thrust toward examining the relationship between direct services and primary prevention services, with a possible interest in shifting the balance toward primary prevention, his words of caution are important.

The papers of Drs. Jack Zusman and Donald Langsley provide an excellent discussion of the philosophical basis of community and social psychiatry, a thorough review of the multiple components of the community mental health center program, and an excellent bibliography that will enable any interested reader to explore additional sources.

The three papers by Drs. Jack Wilder, George Dyck, and Elmer Gardner are concerned with a broad overview of different operational community mental health center programs. Drs. Wilder and Gardner give examples of urban community mental health center programs, and Dr. Dyck provides a description of rural community mental health center programs. Dr. Bernard Holland gives a brief but excellent assessment of the criticisms of community mental health centers. Each of these chapters is lucidly written and highlights the major issues.

Finally, the concluding chapters by Drs. Lucy Ozarin and Stanley Yolles review the available evaluation literature on community mental health center programs and make a few predictions regarding the future directions in which the community mental health center will have to proceed to gain stability.

My only criticism of the papers in this volume is that, although they provide an excellent description of the history of the community mental health movement and its current operational mechanisms, they offer very little concrete information that will assist interested individuals in defining workable solutions to address the problems presented. Nevertheless, this volume is an excellent addition to the existing literature on community mental health, primarily because of its scope and lucidity.

ALLAN BEIGEL, M.D.
Tucson, Ariz.

Psychological Problems of the Child and His Family, *edited by Paul D. Steinhauer and Quentin Rae-Grant. Toronto, Ont., Canada, MacMillan of Canada, 1977, 449 pp., $12.95.*

Those of us who have tried to teach growth and development and psychopathology of childhood and adolescence to medical students face the difficulty of trying to reach a large, diverse audience. Some students have a background in psychology; others have neglected the social and behavioral sciences while concentrating on gaining admission to medical school. Some could not care less about the subject; others, whose interest is awakened, demand more. Some understand the significance of psychological factors for medical practice; others are interested only in the "blood and guts" of medicine. The design of an appropriate textbook for this conglomerate audience has confounded all of us.

In *Psychological Problems of the Child and His Family*, our Canadian colleagues have added to the array of volumes in this field a compendium of discussions of the basic and significant areas produced by various authors. Each chapter constitutes a lecture in itself and is accompanied by a bibliography. The writing is interesting, simplified, and thorough. Although a reader may carp unnecessarily at the selection of references or the underemphasis on certain areas of development, he or she will find here a relatively brief text that often oversimplifies the complex but never insults the audience.

Texts written by a number of authors are often a patchwork with an unevenness of quality. The editors of this book have painstakingly reviewed each chapter so that the text reads as if it were done by one or a very few authors. It is free of jargon. There are many illustrative vignettes. Some chapters are, of course, better than others, but this is a minor criticism. Emphasis is placed on interpersonal aspects without neglect of intrapsychic and developmental aspects. No one attitude is overemphasized, and no one rides a particular hobbyhorse. For example, the admonitions regarding the limitations and misuse of drug therapy are appropriate. The role of the physician in family crisis and chronic care is well presented.

Anyone teaching a course in child psychiatry to medical students (there are precious few who do, since curriculum time is avidly guarded and the priority assigned is low) will find it useful to peruse this text. The authors state in the preface, "If it is to succeed in its task, it is not enough that it be read and memorized; it must be understood and even enjoyed." It has succeeded in its task. It is my hope that many of our students will sip its contents; it is a good vintage.

IRVING PHILIPS, M.D.
San Francisco, Calif.

Temperament and Development, *by Alexander Thomas, M.D., and Stella Chess, M.D. New York, N.Y., Brunner/Mazel, 1977, 265 pp., $13.50.*

Clearly, the brightest lights in terms of serious research in personality development are Thomas and Chess, who initiated the New York Longitudinal Study some 20 years ago. Their work has produced two widely acclaimed publications, *Behavioral Individuality in Early Childhood* (1) and *Temperament and Behavior Disorders in Children* (2). With this third publication one finds a fuller exposition and perhaps a greater emphasis on the amalgam of constitution and environment.

The three temperamental types teased out of their studies are familiar to all: the difficult child, who reacts strongly and negatively, is slow to adapt, and is irregular in habits; the easy child, every parent's fondest wish, who is very adaptable, positive in outlook, and very regular; and, lastly, the slow-to-warm-up child, who, although not negative, approaches new situations cautiously but in the end positively if not pushed too hard. The authors include short questionnaires on temperament for parents of infants and parents and teachers of children 3–7 years of age. Although such questionnaires are somewhat gimmicky, their purpose is important—to assist in identifying the child's basic temperament. Once temperament has been identified, the authors feel that there will be a degree of predictability in terms of the child's response to stressful demands in the environment.

The authors feel that their 20 years of investigation support their basic contention that it is possible to identify in infancy children who will be at risk in terms of their basic temperament and future behavioral disabilities. They include many practical suggestions for parents who have identified their child's temperament; e.g., a slow-to-warm-up child should be given extra time to adjust to such new situations as school and camp.

There is a most interesting appendix on conducting an interview on the child's everyday behavior. It is a personal communication to the authors from Dr. P. Graham, who with Rutler and George has also studied temperament as a predictor of behavior disorders in children. Their studies attempt to describe the child's personality dimensions as mood, intensity of emotional expression, activity, fastidiousness, malleability, regularity, and approach/withdrawal behavior.

Thomas and Chess conclude their book with a quotation from Sir Thomas Browne:

> It is the common wonder of all men, how among so many millions of faces, there should be none alike: now contrary, I wonder as much how there should be any. He that shall consider how many thousand several words have been carelessly and without study composed out of 24 letters; withal how many hundred lines there are to be drawn in the Fabrick of one man, shall find that this variety is necessary.

This is an important study; this is an important book.

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JOHN C. DUFFY, M.D.
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The Symbolic Life: Miscellaneous Writings. The Collected Works of C.G. Jung, Vol. 18. Bollingen Series XX, *by C.G. Jung; translated by R.F.C. Hull. Princeton, N.J., Princeton University Press, 1976, 852 pp., $22.50.*

We now have the final volume of Jung's *Collected Works*. All that remain are the two volumes of general bibliography and general index. No library of any significance should be without this 22-volume series.

Psychotherapists, analysts, scholars of psychoanalysis, and analytical psychologists may profitably turn to volume 18 in the series. It is difficult to review a collection of 160 disparate items written by Jung over a period of 60 years—a mélange of addresses, reviews, forewords, letters, questions and answers, comments to newspapers, comments to magazines, unpublished manuscripts, and other writings. The title of the book is misleading, but its subtitle is not. The title comes from Jung's statement, "You see, man is in need of a symbolic life—badly in need. . . . Where do we live symbolically? Nowhere, except where we participate in the ritual of life" (p. 273). This statement is from his 1939 address to the Guild of Pastoral Psychology in London.

The volume consists of 16 parts; the last 13 are titled according to the volume of the collected works to which they are, in effect, supplements. With this in mind, the reader can understand why things were divided up in such a way. Many of the contributions came to light only after the previous volumes had been published. The 16 sections are titled as follows: The Tavistock Lectures (1935), Symbols and the Interpretation of Dreams (1961), The Symbolic Life (1939), On Occultism (volume 1 of the *Collected Works*), The Psychogenesis of Mental Disease (volume 3), Freud and Psychoanalysis (volume 4), On Symbolism (volume 5), Two Essays on Analytical Psychology (volume 7), The Structure and Dynamics of the Psyche (volume 8), The Archetypes and the Collective Unconscious (volume 9), Civilization in Transition (volume 10), Psychology and Religion (volume 11), Alchemical Studies (volumes 12, 13, and 14), The Spirit in Man, Art and Literature (volume 15), The Practice of Psychotherapy (volume 16), and The Development of Personality (volume 17). Although these items would obviously interest, amaze, delight, or provoke individuals differently, I will highlight the items that impressed me.

My general reaction to this book is a reflection of a statement taken from Jung's foreword to Harding's book *Psychic Energy* and reprinted in this volume:

> The pioneer in a new field rarely has the good fortune to be able to draw valid conclusions from his total experience. The efforts and struggles, the doubts and uncertainties of his voyage of discovery have penetrated his marrow too deeply to allow him the perspective and clarity of vision needed for a comprehensive survey. Those of the second generation, who base their work on the groping experiments, the lucky hits, the circuitous approaches, the half truths and mistakes of the pioneer, are less burdened and can take more direct roads, envisage more distant goals. (p. 469)

The inclusion of Jung's prefaces to the books of his students and analysands in this final volume of his writings is doubly germane. These prefaces, reviews, and forewords reveal the insight of the pioneer into the products of the second generation and vice versa. The volume is a kind of springboard to us of the third generation, as Jung notes, "This simplification and clarification redound to the benefit of those of the third generation, who are thus equipped from the outset with an over-all chart."

The five Tavistock lectures, which Jung gave in 1935 at the age of 64, are informative, spirited talks presenting an overview of analytical or complex psychology, the basic ideas of the collective unconscious, association test experiments, typology, dream analysis, and transference. The question

and answer periods following each speech reveal an amplification of Jung's ideas as well as his personality. (The lectures have been published before [1]). Jung's detailed interpretation of one dream (lecture 5) is a precise example of the "Jungian" analyst at work.

The chapters in the section titled Symbols and the Interpretation of Dreams should especially be noted: "The Significance of Dreams," "The Function of the Unconscious," "The Language of Dreams," "The Archetype in Dream Symbolism," and "The Problem of Types in Dream Interpretation."

Since some people tend to confuse Jung with his subject, especially when he deals with occultism and the mystic quality of the archetypal experience, the section on occultism, which contains his original views on clairvoyance, magnetic phenomena, visions, spiritualism, apparitions, ghosts, and what these phenomena signify, gives us the chance to examine what Jung actually said and the context in which he said it.

The previously unpublished typescript of a 1916 lecture titled "Adaptation, Individuation, Collectivity," presented in Zurich, contains trenchant observations of the psychology and ethics of work and individuation.

In the 88-page section titled Civilization in Transition, Jung expresses his personal and symbolic ideas about the world in which he lived—comments on Switzerland, Germany, Europe, America, Communism, and National Socialism. Jung's feelings about civilization are particularly expressed in the memorable essays "Return to the Simple Life," "Marginalia on Contemporary Events," "Answers to 'Mishmar' on Adolph Hitler," and "Psychology and National Problems."

The section titled Psychology and Religion includes the noteworthy chapters "Jung and Religious Belief" and "On the Discourses of the Buddha." Jung's clinical, symbolic ideas will always annoy individuals who insist on intellectual answers and theories, who see them as poetic, mystic, and unscientific, but what is true? To the phenomenologist/empiricist Jung, psychic entities are truths. He asks, correctly, not are they true (we have long since passed that step with mythology), but who says they are true, in what psychological situation, and what is the symbolic meaning.

The Symbolic Life may turn out to be Jung's last symbolic gift to a world badly in need of the symbolic life:

> That gives peace, when people feel they are living the symbolic life, that they are actors in the divine drama. That gives the only meaning to human life; everything else is banal and you can dismiss it. A career, producing of children, are all *maya* compared with that one thing, that your life is meaningful. (p. 275)

In conclusion, I again quote Jung: "I can recommend [this book] to all those who know how to value things that break through the monotony of daily life with salutary effects [sometimes!] shaking our certitudes and lending wings to imagination" (p. 327).

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HARRY A. WILMER, M.D., PH.D.
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Primate Models of Human Neurogenic Disorders, *by V. G. Startsev; translated by Marienne Schweinler and Vadim Pahn; edited by Douglas M. Bowden. Hillsdale, N.J., Lawrence Erlbaum Associates (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1976, 191 pp., $19.95.*

The aims of this book are to present to the medical world the development of a different kind of experimental model of neuroses in monkeys and to acquaint American scientists with the work of Soviet primatologists. It deals both experimentally and theoretically with the question of symptom specificity, that is, why some individuals respond to psychological stresses with gastric disorders, others with sexual impotence, and still others with high blood pressure.

The book reports a series of studies by Russian workers involving the following conditions: 1) gastric achylia, 2) precancerous gastric lesions, 3) functional hyperkinesis and paralysis, 4) experimental neurogenic disorders of the sexual cycle, and 5) chronic hyperglycemia.

This is a very interesting book. I highly recommend it to interested American scientists as an overview of what the Russians are doing in the area of primate research, particularly as it involves modeling of human "neurogenic" disorders. The terminology throughout the book will be confusing to most American readers without a careful study of the editor's introduction; however, this introduction is well done and will clarify otherwise confusing terminology.

The basic model described in a number of sections throughout the book relates to immobilization stress, involving the concept that the physiological system likely to become chronically disturbed is the one that was particularly active when the animal was subjected to the nervous stress. This model is discussed in relation to the systems listed above. Many readers will be frustrated by the lack of detailed reporting of studies. Throughout the book, one finds jumps from the theoretical background of the study to the conclusions without adequate descriptions of the methodology and, especially, of the data.

The preface by David Graham is well written. There is increasing interest in primate models, and, although this book represents one approach among many, it is worth having because much of the work described is not easily available elsewhere.

WILLIAM T. MCKINNEY, JR., M.D.
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Physical and Sexual Abuse of Children: Causes and Treatment, *by David R. Walters. Bloomington, Ind., Indiana University Press, 1975, 188 pp., $7.95.*

With minimal attention to the gory characteristics of child abuse, the author, a psychologist, presents a timely overview of the subject for professionals involved in treatment of the problem. The difficulty inherent in specifying behaviors that constitute abuse remains unsolved. However, the author differentiates between the physical and sexual abuse of children in both etiology and treatment. Perhaps one of the most helpful sections of the book is Walters' typology of physically abusive adults. Among the 10 classifications of abusers are the parentally incompetent, the frustrated and displaced, the accidental or unknowing, the institutionally prescribed, and the subcultural.

According to the author, effective treatment of the child abuser is contingent on differential diagnosis as to type of

abuser. The immediate treatment goal for all types is cessation of the abuse by assisting the parents in the development of nonabusive child-rearing techniques. Our traditional view of the child abuser as mentally ill should be discarded, Walters says, because pathology per se is present in only a minority of cases. However, when the abuse is a symptom of mental illness, removal of the child from the home is more often indicated than in other types. Prognosis is poor for the mentally ill abuser because cessation of the abuse is dependent on resolution of the illness. Walters emphasizes that no therapeutic reasons justify risking the life or well-being of a child and that it is unethical to use a child as a clinical tool to aid the mentally ill abuser.

Sexual abuse of children typically involves an adult male and a female child. However, the sexual taboos of our society have severely hampered our understanding and treatment of the problem, and reporting of such cases is limited by the fact that the abuser is usually a friend or member of the child's family. Our society's approach to sexual abuse is unique in that the child frequently becomes the accused while the adult is viewed as the victim. The author believes that the dynamic underlying sexual involvement with one's own child is anger toward one's spouse. He considers effective treatment of the sexually abusive adult extremely difficult and recommends referral to a mental health professional.

Although some of the strategies outlined by Walters for the amelioration of child abuse seem rather obscure, his suggestions for treatment are noteworthy. Group therapy is recommended as a more effective and less costly treatment approach than individual therapy. Regardless of treatment modality, a nonjudgmental and nonauthoritarian attitude is required of the therapist if change is to occur. This book is highly recommended for professionals involved in the problem of child abuse.

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The Measurement of Personality, *edited by H. J. Eysenck, Ph.D., D.Sc. Baltimore, Md., University Park Press, 1976, 511 pp., $29.50*

Dr. Eysenck presents a trait-oriented conceptualization of personality and an essentially norm-centered strategy for comparing differences between people. There is nothing new about this; it is offered partially to strike a blow against person-oriented conceptualizations, which, along with being too "literary" for Dr. Eysenck, give evidence that people do not manifest traits consistently. Although one admires Dr. Eysenck's strong personal convictions about his work, his view of his scientific approach as strong, as compared with phenomenological approaches as weak, is an oversimplification that ignores the tremendous complexity of human nature. It is no more meaningful than calling Rousseau a man of large vision and Eysenck a man of small vision.

In *The Measurement of Personality* Dr. Eysenck divides 67 research articles into 10 sections. He prefaces each section with a commentary aimed at forging a rudimentary paradigm he believes is emerging for the field of personality. The linking commentary may be compared to a theoretical lightning-and-thunder bonding together of a string of molecules and stirring them with vitality. Dr. Eysenck restricts his conceptualization of personality to an elementary set of factors, such as traits, which he believes underlie individual differences. For a trait to warrant inclusion into his matrix it must be able to be integrated into general psychology (perception, learning, etc.), must be anchored biologically, and must be strongly determined by genetic causes.

According to Dr. Eysenck, "A proper system of description of human personality would resemble the Mendelieff table of the elements." He believes that at present only three elements outside the cognitive field present themselves as candidates: extroversion-introversion, neuroticism or emotionality as opposed to stability, and psychoticism. (I am reminded of the research and writings of Roland Fischer, who offered a cartography of inner space to match our detailed maps of outer space [1].) Dividing people into extroverts and introverts (using the Myers-Briggs Type Indicator) and comparing them along various dimensions of perception, the extrovert is less sensitive to taste and less drug-sensitive. He or she is described as requiring more sensation and given to thinking and judging, whereas the introvert is more intuitive and given to feeling and perceiving, is taste-sensitive, and requires a lower effective drug dosage.

The Measurement of Personality throws into bas-relief the physiological bases of Eysenck's theory of personality dimensions along measures of sensation, vigilance, perception, psychomotor behavior, learning and conditioning, memory and recall, cognition and creativity, and social and sexual behavior. He makes the point that individual psychological experience is tied in predictable ways to traits correlated to physiological factors. He presents a great deal of substantial evidence for this point. Nevertheless, he is inclined to fault descriptions of personal experience that do not address themselves to biological issues. Perhaps, as his evidence suggests, there are two kinds of people in the world along the extrovert (sensor, judge) and introvert (intuitor, perceiver) continuum that give rise to two difficult approaches to personality study. It seems that there are ways of accepting this phenomenon without thinking of one approach as hard and strong and the other as soft and weak.

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DAVID F. BERRY, PH.D.
West Hartford, Conn.

Suicidology: Contemporary Developments, *edited by Edwin S. Shneidman, Ph.D. New York, N.Y., Grune & Stratton (Harcourt Brace Jovanovich), 1976, 546 pp., $26.50.*

This book by a variety of knowledgeable authors covers many dimensions of suicide and related topics. The very structure of the book offers a unique touch in that each chapter is preceded by a historical note or excerpt. The first one, encompassing the sections on suicide from the *Encyclopedia Britannica* of 1910–1911 and that of 1973, includes Shneidman's own minimasterpiece.

Nonetheless, *Suicidology* is a book of mixed merit. Perhaps it represents a stage in evolution of the understanding of a subject. An early stage of applied knowledge is the collation of information and the creation of generalizations that act as guidelines. As sophistication increases and nuances become more appropriate, one sees the development of differentiation and the breakdown of generalization. Thus this

book demonstrates new perspectives while it mandates a questioning of traditional viewpoints.

The literary qualities of the book work to its disadvantage as a communications device. Many chapters are written in a heavy-handed style that requires the utmost in concentration; this is not a book to read during the 10-minute break. In some cases, statistics abound to befuddle the perusing reader; in others, one encounters numerous examples of specialized vocabulary and almost idiosyncratic verbalisms and neologisms.

With its defects, this book is much better than most edited books; it is replete with reams of information, controversy, and even philosophy (e.g., will suicide become the preferred way of death?). The chapter on Great Britain would imply that the Samaritan Suicide Prevention Movement has lowered the suicide rate there in contrast to its apparent results in the United States.

One encounters the effect of fluidity of time and social change in relation to suicide. According to some measurements, suicide has decreased over the decades. Young suicide shows an increase, as does black suicide; young blacks and whites in the New York area have about the same suicide rate. However, current sociological studies offer contradictory or limited interpretive opportunities.

Predictability remains an unachievable goal. Diggory states, "Our present knowledge is all hindsight; there is no method of predicting." Kiev offers a mass of clinical observations, particularly of the difficulties in handling impulsive people and those who deny the sick role; he states that the social world of the patient is more critical in the patient's subsequent clinical course than diagnosis or severity of the psychiatric condition. The discussions on self-mutilation and suicide and the analysis of suicide among the gifted are most interesting.

The content of this book is too extensive to summarize, and the danger of generalization when it is not justified is great. Those intrigued by the drama of suicide will find a journey through this topography of self-destruction worthwhile. The reader should be cautioned that it is not a light or pleasant journey and that he or she might well complete this laborious trip with the feeling of not being sure where he or she was or where he or she is going.

IRWIN N. PERR, M.D., J.D.
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The Art of Adoption: The "Hows" and "Whys" by an Adoption Worker Responsible for Over 900 Adoptions, *by Linda Cannon Burgess. Washington, D.C., Acropolis Books, 1976, 154 pp., $8.95.*

This book makes a significant contribution toward understanding the whole process of adoption. The author is knowledgeable in her field and writes of her experiences clearly and well, holding the reader's interest throughout.

She begins with a chapter on "The Social Worker and the Agency," stressing the prime responsibility of the agency in deciding what will happen to the unwanted child. She writes of adoptive parents and what their emotional problems might be. Her orientation is Freudian, but not markedly so. The chapters on biological mothers and fathers tell of the often poignant struggle in relinquishing a child for adoption. Her chapter on involved grandparents, adoptive or natural, is particularly interesting and rather surprising in that it reveals the amount of influence these relatives often bring to bear in cases of adoption.

Other chapters deal with the child, the questions that will inevitably be asked, and the outside influences that will affect the child's attitudes. Burgess discusses the problems of suspected rejection, genetic heritage, and court decisions. Opening Pandora's box, she brings up the issue of the adopted person seeking his or her natural parents. This is a current and rather controversial issue, and some adopted individuals are demanding that agency and court records be made available to them. The author leans toward the idea of giving the child early knowledge of the natural parents if feasible, but she points out that a great deal depends on the emotional maturity of the adoptive parents—some can tolerate it and some cannot.

This book is written primarily for adopted children and their parents, both natural and adoptive, but it would make good reading for psychiatrists, psychologists, and social workers. The generally accepted concept is that the child should be told of his or her adoption as early as possible, but the fact of his or her natural parents remains, only to rise later in life.

The author is pragmatic, aware of the many problems that can occur in adoption and how society views them. She writes without prejudice and presents an interesting and often moving account of her experiences over the years in this rather difficult field.

STUART M. FINCH, M.D.
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Bringing Up Children Overseas: A Guide for Families, *by Sidney Werkman, M.D. New York, N.Y., Basic Books, 1977, 215 pp., $9.95.*

This slim volume, written primarily for families who are about to move to a foreign country, is also a useful guide for professionals whose advice is so often sought. It is written in simple, commonsense, practical language, e.g., under the chapter title "The Lure of Living Overseas" are such headings as "romance and adventure," "a wish to discover a heritage," "escape," "the hope for eternal renewal," "the Maharaja syndrome," "the Nipa Hut syndrome," "the gold at the end of the rainbow syndrome," and "the 'eternal wanderer' syndrome."

Bringing Up Children Overseas is a guidebook that is developmentally oriented in its discussion of the conflicts children and youth have over moving and travel. The author emphasizes the family's need to anticipate disappointments and to reorder priorities and set goals in the new setting. He describes the very different and delicately complicated nature of relationships with foreigners, pointing out that the need to change expectations and behavior in adapting to a new culture is not theirs but ours.

Much of the book is reminiscent of the Peace Corps' discovery of and experience with culture shock. The author discusses the effects of a different culture on identity formation in depth, providing a particularly good and sensitive discussion of the old world of servants who are traditionally and customarily used as caretakers for children in other countries. He covers not only the problems associated with the involvement of these caretakers but also their eventual loss to the child. The chapter on education in other countries is particularly useful, as is the one outlining special problems of adolescents.

Throughout the book Dr. Werkman takes an optimistic point of view, minimizing rather than maximizing problems but exposing them realistically, offering practical tips, and emphasizing self-help. The chapter on specific fears of children and their meaning, including the author's advice as to what to do about them, is nicely paired with a chapter on common minor medical problems.

Those who have lived in another country will find that this book reads like a diary; those who are about to move to another country, whether for civilian or military purposes, will find it a gold mine of invaluable information on what to anticipate and how to cope. It is packed with practical suggestions ranging all the way from having the American youngster carry a pocket tape recorder with tapes of music with him or her to placing a bucket of manure on the porch to free the bedroom of flies during the night. The psychiatrist who is called on for advice will do well to have this book on his or her shelf.

JOHN F. MCDERMOTT, JR., M.D.
Honolulu, Hawaii

The Delinquent Way of Life: Third Report of the Cambridge Study in Delinquent Development, *by D.J. West and D.P. Farrington with the assistance of Gwen Gundry, B.J. Knight, and S.G. Osborn. London, England, Heinemann, 1977, 203 pp., £6.00.*

This third report of the Cambridge Study in Delinquent Development is a remarkable follow-up study of some 400 young men born in 1951–1954. The research subjects were chosen in 1961–1962 because they were attending six adjacent primary schools in a working-class area of London. This report is based primarily on interviews with 389 of the subjects in 1971–1973, plus a review of their official records of delinquency. One hundred one of the subjects had an official conviction for one or more offenses committed before the date of the interview.

Contrary to the authors' expectations, they found that convicted delinquent subjects were not typical representatives of the whole group. The delinquent subjects showed behavior traits strongly supporting the concept of a delinquent character. The subjects were more immoderate in their smoking, drinking, gambling, and sexual habits. They drove more recklessly and were more often spendthrift. They were not interested in reading or in further education. Their job records were less stable than those of the nondelinquent subjects. The delinquent subjects earned more money per week, but they were typically in jobs without prospects for advancement.

The authors found that many of the hard-core recidivist delinquents who were antisocial at age 18 had already been recognized as deviant when they were as young as age 8. It seems that delinquent character formation begins early. An inherited temperamental disposition might be a contributing factor, but lack of attention, affection, and consistent training from parents during the formative years is a more obvious and probably more important factor. In later years, according to the authors, "the acquisition of antisocial habits from already delinquent companions, and the stigmatizing effects of being identified with the problem group of truants, rebels, and convicted persons probably reinforce deviant life styles."

This is a monumental study with surprisingly complete follow-up. Statistical analysis is extensive and resourceful. It deals with extent of delinquency and catalogues a large number of predictors both in family background and in the early development of delinquents.

The authors are cautious about their suggestions for action. They suggest that it might be desirable to concentrate some of the scarce social resources to help the most needy, the educationally retarded offspring of large, poor, problem families. They suggest that a sample of vulnerable young males could be randomly allocated to several groups, one of which would be a control group left to the usual resources of existing welfare agencies and each of the others offered a different kind or combination of special help. Follow-up over a period of time should indicate the advantages of each method.

The authors consider delinquency in its quantitative aspects. They make no effort to differentiate qualitatively different subgroups of delinquents. They group only by extent of delinquency and/or recidivism.

RICHARD L. JENKINS, M.D.
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Psychosomatic Medicine: Current Trends and Clinical Applications, *edited by Z.J. Lipowski, M.D., Don R. Lipsitt, M.D., and Peter C. Whybrow, M.D. New York, N.Y., Oxford University Press, 1977, 617 pp., $19.50.*

This volume should have considerable general appeal to *Journal* readers. It consists of a collection of papers originally published in 1975 and 1976 by the *International Journal of Psychiatry in Medicine*. The papers as a group are comprehensive, current, and well organized. If the reader's goal is to read an up-to-date and sophisticated overview of the current trends and clinical applications of psychosomatic medicine, this text provides an elegant experience.

The contents consist of 50 papers, including a timely introduction and epilogue, organized into five parts, each dealing with a salient aspect of psychosomatic medicine. Part one provides perspective with 10 largely theoretical and historical papers under the heading Current Trends in Psychosomatic Theory: The Mind-Body Complex and the External Environment. Part two is titled Intermediate Mechanisms: From Psychological Response to Physiological Reaction. Three brilliant review papers are included here that address neurophysiological, neuroendocrine, and immune mechanisms. An entire volume could easily be given over to papers on mediating mechanisms; however, the editors restrict this rapidly evolving literature to the three major and relevant systems.

Part three, Current Trends and Methodological Approaches in Psychosomatic Research, is concerned with quantification and research methodology. This section is subdivided so that clinical, epidemiological, psychophysiological, and animal studies are presented separately. One quickly appreciates the magnitude of the existing research in psychosomatic medicine in spite of the many quantification obstacles.

Psychosomatic Approach in the Practice of Medicine, part four of the book, provides a modernized look at therapeutic and management issues. Advances in psychopharmacology, biofeedback, behavior modification, and diagnostic principles are pragmatically addressed. The importance of the doctor-patient relationship is appropriately emphasized as a salient contribution of psychosomatic medicine in the 1970s.

Lastly, part five deals with the most unripened aspect of

psychosomatic medicine, namely, teaching. Student and institutional resistance are generally acknowledged. Although the authors identify historical and methodological problems, the reader might be left with a feeling that the resolution of problems in the teaching of psychosomatic medicine is not on the near horizon.

It is difficult to find fault with such a comprehensive and scholarly volume. Admittedly, psychosomatic medicine is many things to many people; however, the clinician has long tended to consider psychosomatic medicine exclusively in terms of the patient who presents with somatic symptoms in the context of psychosocial stress. Patients with psychosocial manifestations of somatic pathophysiology probably deserve more attention. Those suffering from the psychosocial sequelae of dementia, complex-partial seizures, or pharmacologically induced behavior disorders are examples of this very large and often neglected group.

This book is reasonably priced and reflects the work of outstanding contributors in the field. Its organization and clinical relevance allow for easy reading, and its content provides an excellent reference source. It is a pleasure to recommend it to all *Journal* readers.

WILLIAM E. THORNTON, M.D.
Charleston, S.C.

Heroin Addiction: Theory, Research, and Treatment, *by Jerome J. Platt and Christina Labate. New York, N.Y., Wiley-Interscience (John Wiley & Sons), 1976, 398 pp., $19.95.*

This book was written, according to the authors, for two purposes. One was to provide a survey of knowledge about the subject of heroin addiction. The second was to provide a wider perspective than could be obtained from the selective sampling of the literature that characterizes most of us in our professional reading habits. To accomplish their purposes the authors reviewed more than 2,000 published references. Of these, 512 were selected for material presented in the book, and another 494 citations published since 1970 were selected as a supplementary reference list. Thus the book provides a useful selected bibliography on the subject of heroin addiction.

There are four main parts to the book, all of which contain useful reviews of the varied and frequently conflicting findings reported in the literature. Value judgments on the part of the authors are rare, and the useful perspective they provide is that there are no simple answers to this complex and puzzling problem.

Part one deals with the historicolegal context of heroin addiction on international, national, and state levels. The authors provide concise accounts of some of the important legal decisions and legislative acts that have brought us to the place where addiction is no longer a crime and medical treatment is recognized as having a useful role in dealing with the problem.

Physiology and pharmacology comprise part two. Dr. Platt, a psychologist, and Ms. Labate, a research associate, have done an impressive job of lucidly describing important findings and areas of controversy. Experts in pharmacology may argue with their content and organization, but for most psychiatrists these chapters provide a useful review and update. I learned, for example, that monoacetyl morphine, an intermediary metabolite of heroin, penetrates the blood-brain barrier and is absorbed more rapidly by cerebral tissue than morphine itself. I had always viewed as street folklore the statements of many addicts that heroin produces a faster high than morphine. Now research findings have confirmed that aspect of our patients' experiences.

Part three, Explaining Heroin Addiction, is the heart of this book. The authors, who have contributed to research exploding the myth of the addictive personality, carefully review the theories of addiction, psychopathology, and the psychosocial aspects of heroin addiction. Much of the material in this section of the book applies equally well to all chemical dependence, including alcoholism. Any physician working with drug-abusing patients will find the scope of his or her understanding increased by the material the authors present here.

The last section of the book, describing treatment approaches, was for me the most frustrating. The authors are very fair in their descriptions and provide particularly good coverage of the use of methadone in treating heroin addiction. However, they provide little synthesis or interpretation of the findings. In their description of psychotherapy as a treatment modality, they state, "The literature on psychotherapy with heroin addicts is both sparse and unencouraging." They end on this negative note rather than suggesting that psychotherapy will probably work only in the context of a structured treatment program which can also deal with the addict's behavior outside psychotherapeutic sessions. Vaillant cogently made this case in 1975 (1); this article is not cited by the authors.

My frustration is, of course, unfair. Specialists in history, pharmacology, or personality theory would be similarly frustrated with the other three sections of the book, which I found loaded with useful information. The authors have succeeded well in achieving their objectives. They have also produced a book that will serve as a useful stimulant and resource for any physician wrestling with the complex task of treating addiction.

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JOHN N. CHAPPEL, M.D.
Reno, Nev.

Schizophrenia: Biological and Psychological Perspectives, *edited by Gene Usdin, M.D. New York, N.Y., Brunner/Mazel, 1975, 144 pp., $8.95.*

The professional lives of most of those who read this review will be mainly devoted to the important matter of patient care. Few are likely to have much personal acquaintance with the investigations going forward in brain chemistry, the epidemiology of the schizophrenias, and psychopharmacology. Fewer still will read systematically the very specialized journals in which much of the original research is published. The American College of Psychiatrists, devoted to promoting postgraduate education in our profession, recognizes these facts and offers in this short, informative book four excellent review articles and three perspective-giving papers. The reviews are compact and reward a little study.

Dr. Arvid Carlsson's piece on the pharmacology of schizophrenia is a clear, uncluttered exposition of the interaction of antipsychotic agents, the neurotransmitters in brain synapses, and the importance of the major dopaminergic pathways. A clinician who wants to know how his or her pre-

scriptions work will find the main facts and theories spelled out here in 19 pages. Mandell's chapter expands on the current state of neurobiochemistry. He discusses the neurotransmitters, their discharge from presynaptic membranes, their uptake, and the inhibition of their action at postsynaptic receptor sites. He also reviews the factors that govern receptor function over longer periods, substrate uptake mechanisms, and delayed alterations in enzyme activities and amounts.

Chodoff and Carpenter discuss the psychogenic theories of schizophrenia, acknowledging the many problems of diagnosis and method that beset such research. The psychoanalytic hypotheses include those of conflict, thought disorder as a defense against intolerable affect, and schizophrenia as the consequence of a defect in structure resulting from defective internalization of objects and leading toward a fragile sense of self.

Chodoff and Carpenter mention the studies of Lidz, Wynne, and others dealing with disturbed families and their communication patterns as well as the destructive influences of parental egocentricity and narcissism. They suggest that the last several decades of psychogenetic inquiry have resulted in a consensus: schizophrenia most characteristically emerges in people who come from a disturbed family environment with faulty, idiosyncratic communicative processes, who are handicapped by poor early object relationships, and who are subject to an agonizing ambivalence in attitude toward interpersonal closeness.

Robert Cancro discusses the impact of genetic studies on the understanding of etiology and prevention. He sets out the main outlines of contemporary genetic evidence from studies of consanguinity, twins, and adoption clearly and without intruding too much detail. He also discusses the implications of genetic understanding for eugenic programs and prophylaxis.

A genetic factor in the prevalence of schizophrenia has now been demonstrated beyond reasonable doubt, but the exact nature of the factor is yet to be defined. Cancro speculates that more than one gene is involved and that more than one genetic constellation can set the stage for a schizophrenic breakdown when the patient falls into the correct evoking environment. Just what the characteristics of that environment might be, of course, we do not know, but Cancro mentions a range of psychological, social, family, and dietary possibilities. His chapter is a particularly fine one in that it demonstrates clearly how neither hereditary-chemical factors nor environmental-psychological ones alone are sufficient to explain the prevalence of the schizophrenias. The view that both genetics and environment contribute to the genesis of these psychoses is shared by other writers in this collection.

Klerman defends the medical or disease model of schizophrenia, pointing out parallels to such disorders as arthritis and hypertension. Although we have seen real advances in the institutional management of the schizophrenias in recent years, much remains to be done in the development of adequate aftercare. Although prolonged hospitalization is known to be harmful, there are insufficient rehabilitative, vocational, and residential resources available for adequate support of many discharged patients.

Fewer and fewer studies are now published on the utility of family, group, or individual psychotherapy; the lack of new evidence might partially account for the fact that no chapter devoted to psychotherapy alone appears in this book. In spite of the fact that many clinicians are convinced that psychotherapy is lifesaving in some cases and that it is useful in reducing morbidity, there are few studies that definitely prove this. The lack of a chapter on psychotherapy is regrettable, but most recent advances in schizophrenia studies are in the chemical-hereditary area. Many of the authors of the perspectives in this book agree that social therapies geared to the problems of the here and now are important in the manangement of the recovering patient, as is the prescription of appropriate antipsychotic drugs.

The book is rounded out with chapters by Roy Grinker, Sr., and Morris Lipton. Grinker comments on the evolution of schizophrenic research over the past 50 years, and Lipton discusses the impact of biological studies on theory and treatment.

The various articles are accompanied by useful bibliographies. *Schizophrenia: Biological and Psychological Perspectives* is an excellent general review for clinical psychiatrists who wish to keep informed as well as for residents and other physicians.

JOHN T. MALTSBERGER, M.D.
Boston, Mass.

The Experience of Dying, *edited by E. Mansell Pattison. Englewood Cliffs, N.J., Prentice-Hall, 1977, 335 pp., $10.95; $4.95 (paper).*

Some might say that the Age of Death emerged with the publication of Feifel's *The Meaning of Death* in 1959 (1). Since then, the field of thanatology has undergone a development all its own. The myths and taboos, the silence and neglect, and the fears and guilts that once enshrouded the topics of death and dying no longer encumber the enlightened clinician. Perhaps we have reached the stage, not dissimilar to Erik Erikson's idea of industry versus inferiority (2), in which the practitioner is receptive to instruction.

This book is of the type Cohen categorized as having a developmental-psychopathological perspective (3). Dr. Pattison's stated goal is "to provide a broader, in-depth portrayal of the dying process in its many personal forms." To accomplish that goal, he complements his erudition and his 10 years of clinical experience in dealing with dying patients with the thanatological expertise of 24 knowledgeable contributors. The 29 chapters of this book are addressed to those who must manage and care for the dying person. The topical bibliography provides the student with recent references to relevant issues in thanatology.

The first part of the book presents a theoretical background to understanding the dying process. Of particular interest is chapter 4, "The Experience of Dying," in which Dr. Pattison introduces the concept of the living-dying interval. Because each of us has certain life-span expectations according to which we plan and arrange our lives, we may be ill-prepared to encounter a shortening of those expectations. The reality of illness or accident may confront us with the crisis of knowledge of our own death. The detached and nebulous idea that all things eventually die becomes the personal and unavoidable idea, "I am dying now." The period between the crisis of knowledge and the point of death is the living-dying interval, more prevalent today because of major advances in medical therapeutics. Dr. Pattison divides this interval into three clinical phases: the acute crisis, the chronic living-dying, and the terminal phase. Within these three phases the practicing clinician can intervene and help the dying patient.

Parts two through seven of the book underscore the im-

portance of viewing the dying process throughout the life's cycle. Too little systematic scientific work has dealt with the cognitive meaning of death and the typical coping styles of the dying. Dr. Pattison's skill in selecting contributors able to elucidate the dying process, with its different reflections of the influence of such illness contexts as accidents, infections, malformations, organ transplants, and cancers, is both innovative and enlightening.

In part eight of this book, Styles of Dying, Dr. Pattison debunks the formalized stages of dying (4), expands on the notion of ego coping mechanisms in dealing with the stress of the living-dying interval, and, most importantly, gives the reader some guidelines for helping dying patients. In sum, that helping involves befriending the patient, providing "the corrective, supportive, inquisitive, challenging, accepting give and take that comprises our valued relationships with others."

In conclusion, aside from some irritating oversights by the copy editor—for example, Drs. E. Shneidman, A. Weisman, and E. Lindemann's names were misspelled—this book is both clinically informative and sensitively written. Dr. Pattison has done a useful service for all those who deal with the dying patient and his or her family.

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MICHAEL F. HEIMAN, M.D.
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The Basic Neurology of Speech, 2nd ed., *by Michael L.E. Espir, M.A., M.B., and F. Clifford Rose, M.B., D.C.H. Oxford, England, Blackwell Scientific Publications (Philadelphia, Pa., J.B. Lippincott Co., distributor), 1976, 143 pp., $12.00 (paper).*

This book is written primarily for speech therapists, but it provides concise and enlightening information for the psychiatrist interested in communication and its neurological aspects. One of the very attractive features of this book is its brevity. In less than 150 pages the authors succinctly present a difficult subject in a readily understandable fashion. The information they provide is not new, but it is usually scattered throughout larger textbooks.

The first part of the book describes the various types of speech and language disorders (stammering, dysarthria, and dysphonia) and their neuroanatomical and neurophysiological substrates. The second part gives a concise account of brain disorders that can influence speech and language, including a particularly good summary of the speech disturbances in epilepsy and dementia.

In conclusion, this is a valuable book for the psychiatrist who might tend to forget the many intermediate steps between thought and expression.

PHILIPPE E. CHANEL, M.D.
Washington, D.C.

The Alcoholisms: Detection, Assessment, and Diagnosis, *by George R. Jacobson, Ph.D. New York, N.Y., Human Sciences Press, 1976, 414 pp., $19.95.*

The author's purpose in this book is to select from the psychological tests used in studying alcoholism those which might be useful in identifying the prealcoholic person or the individual at risk, in assisting in taking preventive measures, and in detecting the alcoholic person at the earliest possible point in the development of the disorder. On this basis he reviews 12 tests that have been applied for these different purposes.

The following tests were chosen: the Alcadd Test (1949), the Manson Evaluation (1949), the Essential Readdictive Alcoholism Dimension (1961), the Iowa Alcoholism Intake Schedule (1965), the MacAndrew Alcoholism Scale (1965), the Alcoholism Assessment Interview (1965), the Alcohol Use Questionnaire (1969), the Bell Alcoholism Scale of Adjustment (1969), the Michigan Alcoholism Screening Test (1971), the Mortimer-Filkens Test (1971), the Criteria for the Diagnosis of Alcoholism (1972), and, finally, the Alcohol History Form—Alcoholism Severity Scale (1973).

The author describes each of these psychometric measures in detail in individual chapters, using a format that indicates the source of the most significant documents relating to the development of the test, its availability, a description and details of the test, the technique of its administration, and comments on the test as well as the training and experience necessary to properly administer, score, and interpret it. He includes a section on the development of each test and its reliability and validity. He expresses his own subjective evaluation of each test and its possible relationship to treatment planning and predicting outcome. He also makes suggestions as to which test might be particularly useful with special populations, such as drinking drivers, women, young people, poverty groups, and others. He discusses the advantages and disadvantages of each test and concludes with a helpful summary of each.

The author's scholarly and critical examination of these tests provides an overview of their formulation and applicability. The printing and format of the monograph make for ready accessibility. If there is any serious criticism of the presentation, it would be of its terseness. For those interested or involved in the study of the prevention and management of alcoholism, the survey provides a useful guide in assessing and selecting psychometric measures that might be most appropriate for a particular task. The book can be recommended as a useful addition to one's working library.

ALBERT A. KURLAND, M.D.
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Treatment and Rehabilitation of the Chronic Alcoholic. The Biology of Alcoholism, Vol. 5, *edited by Benjamin Kissin and Henri Begleiter. New York, N.Y., Plenum Press, 1977, 614 pp., $39.50.*

This fifth and final volume of a comprehensive review of the biological, psychological, and social aspects of alcoholism is devoted to the treatment of the alcoholic patient. It is one of the best books that I have read on this subject—is eminently readable and interesting in spite of the large amount of material presented. The many current treatment methods are well described and evaluated, and the historical background of treatment and extensive resource references

relating to this subject are provided. The wide range of information contained in this book is accurate, thorough, and up-to-date.

As the authors point out, the various remedies proposed for alcoholism often have strongly biased cohorts either for or against them. This suggests the complexity of this medical-social problem and the difficulty of obtaining independent appraisal of the relative effectiveness of different treatment modalities. There are many kinds of alcoholic patients; the therapist must individualize treatment to fit the needs of each patient. Usually a multipronged approach to the problem is required, as outlined in this book. Alcoholic patients constitute a markedly heterogeneous population with widely disparate needs; what helps one victim might not be indicated for another.

A broad spectrum of treatment modalities is available. Among those discussed in this book are medical; pharmacological (including disulfiram [Antabuse], lithium, antidepressants, and tranquilizers); psychotherapy; counseling; behavior modification (including aversive and operant conditioning and motivation); social, cultural, and religious therapy; family and group therapy; therapeutic communities; halfway houses; orientation lectures; and, of course, Alcoholics Anonymous. The authors stress the absolute need for therapists to be nonjudgmental, nonpunitive, and objective. They also stress the need for continuity of care.

The book quotes Keller's law: "The investigation of any trait in alcoholics will show that they have more or less of it." Keller concluded that "alcoholics are different in so many ways that it makes no difference." The special validity of this book is that all of the many treatments are as thoroughly evaluated as is possible with the current level of our knowledge. This enables the therapist to prescribe objectively and intelligently the treatment or treatments best suited to help his or her patient.

FREDERICK LEMERE, M.D.
Seattle, Wash.

Consciousness and Self-Regulation: Advances in Research, Vol. 1, *edited by Gary E. Schwartz and David Shapiro. New York, N.Y., Plenum Press, 1976, 385 pp., $18.95.*

Cognitive therapies—those focused on conscious thought content and the here and now—have elicited widespread interest. Persuasion, long considered a historical relic, has attained legitimacy through Jerome Frank and celebrity through Albert Ellis and Aaron T. Beck. "Rational thinking," self-taught or tutored, and ideation-oriented behavior therapy techniques are respectable in scientific circles. Indeed, psychology, we are told, has already "gone cognitive," and cognition has been hailed as the Cinderella of psychiatry.

The basic sciences underlying cognitive therapy would probably include the venerable psychological disciplines of introspectionism and associationism. To these would be added the areas encompassed by *Consciousness and Self-Regulation.*

As a result of their successful interdisciplinary seminars at Harvard, Gary Schwartz and David Shapiro were commissioned to edit a series of volumes devoted to the psychophysiology of consciousness, emotion, and self-regulation. The entire series is appropriately dedicated to William James, reverence for whom in this first publication does not preclude disagreement.

This first volume's nine chapters are mainly review articles by pioneers in their respective fields. Ernest R. Hilgard, on the basis of his classic hypnosis research, applies to pain perception his developing theory of multiple simultaneous streams of awareness. Karl H. Pribram recounts his 40 years' work in tracking the limbic system. Electroencephalographers trace out memory storage and retrieval, try to delineate hypnotic trance and altered states of consciousness, and monitor dreaming in search of psychological and physiological meanings.

Data and insights of direct relevance to psychiatric practice are concentrated in the two chapters devoted to clinical research. Reading Thomas D. Borkovec, one makes a startling discovery about anxiety, the key concept in the psychodynamic understanding of neurosis and other emotional disorders: patients, therapists, and researchers have been making the same error in believing that all anxiety is alike. Although all the answers are by no means at hand, at least some long-neglected questions are now being asked.

Borkovec subclassifies the experience of anxiety into physiological, subjective, and behavioral components. These components can then be further broken down, e.g., distinctive physiological profiles can be identified by means of Mandler's Autonomic Perception Questionnaire. Phobias themselves may have unique patterns: in snake phobias, characterized by determined avoidance of exposure to snakes, autonomic disruption is minor; in public speaking anxiety, by contrast, there is major autonomic disruption.

Therapeutic interventions can be shown to be specific in their effects. The mobilization of hope is a potent healing force only when autonomic factors are insignificant. When physiological arousal is the key sustainer of neurotic fear and behavior, cognitive interventions are relatively ineffective; in this instance such treatments as desensitization, flooding, and biofeedback are required. Drugs that alter the limbic system or autonomic nervous system might be indicated.

Why do some individuals capitulate to a level of autonomic arousal that is resisted easily by others? Borkovec should probably consider such differences in the capacity to endure unpleasant affects. A concept of ego strength would accord with Meichenbaum's principle, stated in the book's other clinical research chapter, that men are not moved by what they feel but by what they say to themselves about what they feel. This principle is a restatement of what might be called the cognitive creed: Epictetus' observation that "men are not moved by things but by the views which they take of them."

In his treatise on how to talk to oneself constructively, Donald Meichenbaum describes what he claims to be the final common pathway of successful psychotherapy—the patient's altering his or her internal monologue in order to facilitate adaptive behavior. Meichenbaum advocates the teaching of a generalized problem-solving approach that the patient can apply throughout his or her lifetime. An anxious individual with sweaty palms and tachycardia learns to reinterpret these phenomena as signals to employ coping techniques in which he has been trained (e.g., relaxation).

Each of the seven basic science chapters presents a new technical nomenclature and research methodology. For the psychiatric clinician this is rugged foreign terrain. On the whole, to traverse it requires more exertion than it is worth. Without considerable further study the reader will not learn enough about any field to be able to devise his or her own practical applications or hypotheses. He or she will have to be content with the smaller joy of sifting out choice bits of fact and conjecture that might be useful.

My personal selection of useful bits of information include the following:

There are many functional differences between the more concrete and verbal left cerebral hemisphere and the more dreamy, abstract right.

The basal ganglia control not only motor function but sensory input.

Seemingly irreversible deficits in brain function might one day be recouped. Information appears to be stored diffusely throughout the brain. Following destruction of a primary storage center, information could theoretically be recalled from secondary centers that were previously silent due to a signal-to-noise ratio lower than that for the primary center.

Sensory-deprived subjects are more amenable to attitude change than control subjects.

REM deprivation has not proved to be disruptive for the dreamer. Nor is it unquestioned that REM sleep restores the mind while NREM sleep restores the body.

Hypnotic susceptibility can be augmented by instructing subjects in how to experience hypnosis and by correcting anxiety-inducing misconceptions about hypnosis.

It can be predicted that Schwartz and Shapiro's later volumes will contain fewer speculations and more conclusions. Meanwhile, however, the first volume of *Consciousness and Self-Regulation* is not the place to start learning about cognition. For psychiatrists, less strain and more immediate profit will attend the examination of Beck's familiar if limited perspective (1) and Michael J. Mahoney's shrewdly critiqued compendium (2).

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JOSEPH S. SILVERMAN, M.D.
Altoona, Pa.

The Impact of Biology on Modern Psychiatry, *edited by Elliot S. Gershon, M.D., Robert H. Belmaker, M.D., Seymour S. Kety, M.D., and Milton Rosenbaum, M.D. New York, N.Y., Plenum Press, 1977, 270 pp., $25.00.*

This book comes from a scientific symposium held in Jerusalem in December 1975 to honor the 80th anniversary of the Jerusalem Mental Health Center Ezrath Nashim. The proceedings have the usual mixture of internationally known scientists reviewing work from their laboratories and includes generally less well known scientists presenting findings from specific projects. The uniqueness and value of this publication comes primarily from the latter, since the work of a number of the contributors is readily available in other publications. The proceedings are especially useful in calling attention to important work emerging from several institutions in Israel. This symposium is a collection of individual reports with little attempt toward thematic organization; therefore, I will briefly describe the contents rather than discuss them.

The book is divided into three sections. The first is titled Neurochemical Studies in Human Psychopathology. Dr. Herman M. van Praag provides an excellent overview of the work of his laboratory relevant to cerebral dopamine metabolism in relationship to pathogenesis and treatment of psychotic disorders. Dr. Goran Sedvall reviews the work conducted in the Karolinska Institutet examining CNS biochemical mechanisms relevant to antipsychotic drug action in humans. Dr. Samuel Gershon reviews the relationship of psychopharmacological agents to psychiatric diagnosis. The remainder of this section is composed of reports from several workers in Jerusalem. Drs. Ebstein and Belmaker compare lithium and haloperidol action on human peripheral β-adrenergic adenylate cyclase. Dr. Elizur and colleagues examine lithium and electrolyte distribution in relationship to neurotoxic lithium effects, and Dr. Samuel reports on a new method using oxygen-18 to measure monoamine turnover. There are also reports on levels of antibodies to viruses in various psychiatric illnesses by Drs. Rimon and Halonen and a report on a tissue-binding factor in the serum of schizophrenic patients by Dr. Witz and coworkers.

The second section is titled Catecholamines and Behavior. The six papers here are all interesting, but they do not constitute a coherent conceptual arrangement. They range from Dr. Youdim's discussion of factors influencing CNS biogenic monamine activity to Dr. Korczyn's reports on "Combined Use of Neuroleptic Drugs." Arnold Mandell provides a delightful piece critiquing the old and suggesting a new biochemical theory for psychiatry.

The final section, Genetic Studies in Psychiatry, presents an overview of current genetic strategies relevant to the major psychoses. This section has thematic organization, which makes for easy reading and utility as resource material. The genetics of schizophrenia is discussed by Seymour Kety, affective illness by Elliott Gershon, schizo-affective illness by Julien Mendlewicz, and diagnostic issues in twin studies by Margit Fischer. Drs. Belmaker and Ebstein relate genetically influenced enzymatic activity to the biogenic amine hypotheses of psychiatric disorders.

The Impact of Biology on Modern Psychiatry is disappointing in its contrast between title and content. The title suggests that the reader will receive a historical review of developments in neurobiology and genetics, their influence on psychiatric research, and the eventual impact on psychiatry in general. However, the reader will find a conventional symposium dealing with biological issues in psychiatry without any perspective on the growth of biological psychiatry, the relationship of neurobiology to the development of psychiatry, or consideration of implications for the future. One can merely note that the symposium itself is an example of the increasing influence of biology on modern psychiatry.

WILLIAM T. CARPENTER, JR., M.D.
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Brain Metabolism and Cerebral Disorders, 2nd ed., *edited by Harold E. Himwich. Holliswood, N.Y., Spectrum Publications (New York, N.Y., Halsted Press, John Wiley & Sons, distributor), 1976, 361 pp., $28.00.*

For those of us who knew Harold Himwich, this edition conjures up fond memories of a most imaginative and creative human being. He was the prototype of the truly multidisciplined scientist whose insights into events in the laboratory and the needs in the clinic bridged this gap and pointed the way for the young to follow.

In contrast to the first edition of this important book, in which all of the chapters were from the same pen, half of the chapters in this edition were written by Dr. Himwich. Nevertheless, these are the chapters central to the theme that

"ontogeny recapitulates phylogeny" first expressed by the late great neuroscientist Hughlings Jackson. It is not in the anatomical but in the functional sense that the theme is threaded through these pages. Himwich championed no single discipline; rather, he emphasized that a knowledge of the developing nervous system called on diverse disciplines to achieve full comprehension. That goal has yet to be achieved, but progress in no modest measure is attributable to Himwich's keen insights and heuristic writings and is exemplified in this second edition of *Brain Metabolism and Cerebral Disorders*.

Unlike many texts written in the popular trend that contain an assortment of papers presented in symposium form at some exotic place, for this book the editor wisely chose contributors whose philosophy of science is consistent with his own and whose expertise contributes significantly.

The three chapters on cerebral metabolism written by the editor serve as a reminder to the young investigator of the many important observations on the brain made at a time when less sophisticated methodology and little knowledge of brain metabolism was available. Many animal data are included in these chapters, which are complemented by a chapter on human brain metabolism by W. Sachs. Dr. Sachs pioneered tracer methods for assessing the relative participation of alternative metabolic pathways in the brains of normal and chronically ill patients. The studies on carbohydrate metabolism are especially informative.

Recognizing that metabolism is intimately associated with brain blood flow, Himwich includes four chapters devoted to a discussion of the anatomical and dynamic aspects of this subject in health and disease. To help in understanding the anatomy of the circulatory tree, neurochemists especially should appreciate the chapter by Professor A.G. Baptista, who presents those morphological characteristics which are distinctive in the human. He blends this with a background concerning the circulation and distribution of blood to the brain. With this background, the chapter on "Regulation of Cerebral Blood Flow" by D.H. Ingvar and N.A. Lassen is very readable and should prove valuable to those less familiar with the fundamental finding that cerebral blood flow (CBF) is ultimately controlled by oxidative metabolism of the nervous system. An area not covered in this chapter concerns the use of short-lived (about 10 minutes) positron-emitting isotopes. Like gamma-emitters, their activity can be detected with the same crystal multidetectors, but such isotopes can be incorporated into metabolically active substrates and provide information not available with current isotope techniques. In spite of this limitation, the chapter provides a meaningful basis for greater in-depth inquiry into a vast array of original papers on the subject.

The chapter by Oldendorf on the blood-brain barrier and on cerebral flow and metabolism in epilepsy, anoxia, ischemia, and edema is most important to the neurologist and neurosurgeon. With the background provided by the chapters by Baptista and Ingvar and Lassen, even the uninitiated is able to gain considerable insights into these most formidable problems.

It is perhaps most appropriate that the book concludes with chapters by Dr. Himwich on the somatic and autonomic divisions of the CNS. From his many years of inquiry into insulin as a means of treatment for mental disorders, he provides us with a tool to delineate the phyletic organization and function of the CNS. It is entirely appropriate as well to conclude this book with a description by Professor Barbeau of the rational approach to parkinsonism, a degenerative disease that appears generally after middle life. Professor Barbeau provides a synthesis of modern neurochemical concepts with a neurophysiological understanding of the underlying deficits. For this he draws on a large population of patients he has personally treated. Despite the progress made since the introduction of L-dopa, the author puts the problem in perspective when he states that this is a generalized disease of pigmented neuron degeneration not restricted to the nigrostriatal pathway.

There is still considerable knowledge to be gained about the developing and aging brain. This book provides a most provocative status report of neuroscience at the time the author took leave of his many friends, colleagues, and students with whom he unselfishly shared his enthusiasms and zest for this subject.

JOHN J. O'NEILL, M.D.
Philadelphia, Pa.

Human Ecology and Cognitive Style: Comparative Studies in Cultural and Psychological Adaptation, *by John W. Berry. New York, N.Y., Sage Publications (Halsted Press, John Wiley & Sons, distributor), 1976, 242 pp., no price listed.*

The author is an associate professor in the Department of Psychology, Queen's University, Kingston, Ont., Canada, and is also an associate editor of the *Journal of Cross-Cultural Psychology*. This book is one of a series titled Cross-Cultural Research and Methodology and would be considered a fair sample of contributions of its genre. It is a report of a decade's work. The author attempts a semiquantitative description of an extremely large conceptual canvas, reporting data on 21 sample groups from 11 different cultures. From these data he attempts to construct a theoretical model integrating ecological, cultural, and behavioral dimensions of behavior.

Berry uses ethnographic and psychological methods, combining anthropological and psychological approaches. The result is likely to be incomprehensible to psychiatrists without anthropological training. Further, it would interest only those with a narrowly focused research interest. The "cognitive styles" referred to in the title allude to the field-dependent-field-independent dimension of Witkin and associates, roughly subsumed under the theory of psychological differentiation. The principal finding reported is that members of preliterate, nomadic, loose cultural groups tend to be field independent, but members of tight, sedentary, agricultural groups tend to be field dependent in their cognitive styles.

Overall, Berry's presentation suffers from serious faults and limitations that most readers will find, at the least, very confusing. His model appears to be more complex than the data, and his subject matter is too obscure to be of interest to more than a handful of anthropologically oriented researchers in cross-cultural psychology.

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Books Received

This listing acknowledges the receipt of recent books. Books of particular interest to the readers of this journal will be reviewed as space permits, and copies of the reviews will be sent to the publishers. Books cannot be returned to the publishers.

Primary Prevention of Psychopathology, Vol. I: The Issues, *edited by George W. Albee and Justin M. Joffe. Hanover, N.H., University Press of New England, 1977, 399 pp., $20.00.*

Understanding Psychotherapy: Comparative Perspectives, *by George J. Allen. Champaign, Ill., Research Press Co., 1977, 271 pp., $6.95 (paper).*

Psychopharmacology: From Theory to Practice, *edited by Jack D. Barchas, M.D., Philip A. Berger, M.D., Roland D. Ciarranello, M.D., and Glen R. Elliott, Ph.D. New York, N.Y., Oxford University Press, 1977, 538 pp., $17.95; $10.95 (paper).*

The Implementation Game: What Happens After a Bill Becomes a Law, *by Eugene Bardach. Cambridge, Mass., MIT Press, 1977, 316 pp., $17.95.*

Evaluation of New Drugs in Clinical Psychopharmacology. Drugs Under Experimental and Clinical Research, Vol. 1, No. 1-2, April 1977, *edited by A. Bertelli, G.B. Cassano, P. Castrogiovanni, J. Levine, and J.R. Wittenborn. Barcelona, Spain, J.R. Prous, 1977, 462 pp., $50.00.*

Community Mental Health: A General Introduction, *by Bernard L. Bloom. Brooks/Cole Publishing Co. (Wadsworth Publishing Co.), 1977, 316 pp., $10.95.*

Artificial Intelligence and Natural Man, *by Margaret A. Boden. New York, N.Y., Basic Books, 1977, 537 pp., $17.50.*

Celebrate Yourself: Making Life Work for You, *by Dorothy Corkille Briggs. Garden City, N.Y., Doubleday & Co., 1977, 213 pp., $7.95.*

Behavioral Assessment: New Directions in Clinical Psychology, *edited by John D. Cone and Robert P. Hawkins. New York, N.Y., Brunner/Mazel, 1977, 418 pp., $17.50.*

Anti-Oedipus: Capitalism and Schizophrenia, *by Gilles Deleuze and Félix Guattari; translated by Robert Hurley, Mark Seem, and Helen R. Lane. New York, N.Y., Richard Seaver (Viking Press), 1977, 395 pp., $16.95.*

Surviving in Dentistry: The Sources of Stress, *by J.E. Dunlap, D.D.S. Tulsa, Okla., Dental Economics/PPC Books, 1977, 123 pp., $13.00.*

The Understanding of the Brain, 2nd ed, *by John C. Eccles. New York, N.Y., Blakiston (McGraw-Hill Book Co.), 1977, 244 pp.; $5.95 (paper).*

Current Developments in Psychopharmacology, Vol. 4, *edited by Walter B. Essman and L. Valzelli. New York, N.Y., SP Books (Spectrum Publications), 1977, 264 pp., $25.00.*

New Career Options for Women: A Counselor's Sourcebook, *by Helen S. Farmer, Ph.D., and Thomas E. Backer, Ph.D. New York, N.Y., Human Sciences Press, 1977, 400 pp., $16.95.*

New Career Options: A Woman's Guide, *by Helen S. Farmer, Ph.D., and Thomas E. Backer, Ph.D. New York, N.Y., Human Sciences Press, 1977, 96 pp., $2.95 (paper).*

Biological Bases of Psychiatric Disorders, *edited by Alan Frazer, Ph.D., and Andrew Winokur, M.D., Ph.D. New York, N.Y., SP Books (Spectrum Publications), 1977, 262 pp., $15.00.*

Five Lectures on Psycho-Analysis, *by Sigmund Freud; translated and edited by James Strachey. New York, N.Y., W.W. Norton & Co., 1977, 73 pp., $6.95.*

A Sexual Profile of Men in Power, *by Sam Janus, Ph.D., Barbara Bess, M.D., and Carol Saltus. Englewood Cliffs, N.J., Prentice-Hall, 1977, 187 pp., $9.95.*

Strindberg and Van Gogh: An Attempt at a Pathographic Analysis with Reference to Parallel Cases of Swedenborg and Hölderlin, *by Karl Jaspers; translated by Oskar Grunow and David Woloshin. Tucson, Ariz., University of Arizona Press, 1977, 214 pp., $12.50; $4.95 (paper).*

Primer of Lithium Therapy, *by James W. Jefferson, M.D., and John H. Greist, M.D. Baltimore, Md., Williams & Wilkins Co., 1977, 206 pp., $10.50 (paper).*

Stuttering: The Disorder of Many Theories, *by Gerald Jonas. New York, N.Y., Farrar, Straus and Giroux, 1977, 68 pp., $7.95; $2.95 (paper).*

Medicine and Money: Physicians as Businessmen, *by Joseph LaDou, M.D., and James D. Likens, Ph.D. Cambridge, Mass., Ballinger Publishing Co. (J.B. Lippincott Co.), 1977, 200 pp., $15.00.*

Telegarbage: What You Can Do About Sex and Violence on TV, *by Gregg Lewis. Nashville, Tenn., Thomas Nelson, 1977, 164 pp., $2.95 (paper).*

Alcoholism and Drug Dependence, *edited by J.S. Madden, Robin Walker, and W.H. Kenyon. New York, N.Y., Plenum Press, 1977, 469 pp., $39.50.*

Frontiers of Medicine, *edited by Gordon M. Meade, M.D. New York, N.Y., Plenum Press, 1977, 221 pp., $25.00.*

ANNUAL INDEX

Following is the comprehensive index for volume 134 of the *Journal*, covering all material from January 1977 through December 1977. The complete citation for each article in the author index is listed under the name of the first author; joint authors are listed alphabetically with a cross-reference to the full citation. Book reviews appear in the author index under the name of the reviewer and in the subject index under the heading Books Reviewed; the books are arranged alphabetically by the surname of the book's first author.

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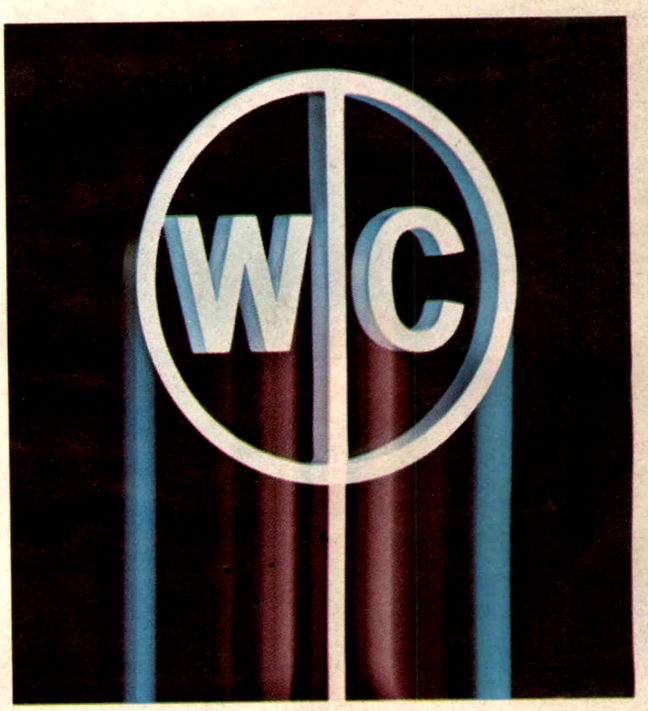
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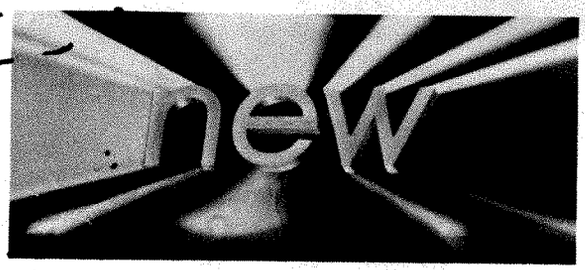
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■ **New Verstran**
The simplicity of a single scored 10 mg tablet.

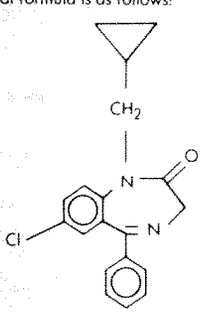
■ **New Verstran**
The versatility of fully effective divided daily or single nighttime dosage.

■ **New Verstran**
Side effects are characteristic of benzodiazepine drugs.*

Verstran* (prazepam) Ⓒ IV

Caution: Federal law prohibits dispensing without prescription.

Description: Verstran (prazepam); a benzodiazepine derivative, is identified chemically as 7-chloro-1-(cyclopropylmethyl)-1,3-dihydro-5-phenyl-2H-1, 4-benzodiazepin-2-one. The molecular weight is 324.8 and the structural formula is as follows:



Clinical Pharmacology: Studies in normal subjects have shown that Verstran (prazepam) has depressant effects on the central nervous system. Oral administration of single doses as high as 60 mg and divided doses up to 100 mg t.i.d. (300 mg total daily dose) were without toxic effects.

Single oral doses of Verstran in normal subjects produced peak blood levels at 6 hours postadministration, with significant amounts still present after 48 hours. Verstran was slowly absorbed over a prolonged period, rather constant blood levels were maintained, and excretion was prolonged. The mean half-life of prazepam measured in subjects given 10 mg t.i.d. for one week was 63 (± 15 s.d.) hours before and 70 (± 10 s.d.) hours after multiple dosing—a nonsignificant difference. Human metabolism studies showed that prior to elimination from the body, prazepam is metabolized in large part to 3-hydroxyprazepam and oxazepam.

Indications: Verstran (prazepam) is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

Contraindications: Verstran (prazepam) is contraindicated in patients with a known sensitivity to the drug and in those with acute narrow angle glaucoma.

Warnings: Verstran (prazepam) is not recommended in psychotic states and in those psychiatric disorders in which anxiety is not a prominent feature.

Patients taking Verstran should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery, including motor vehicles.

Because Verstran has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, including phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants. The effects of alcohol may also be increased.

Physical and Psychological Dependence: Withdrawal symptoms similar in character to those noted with barbiturates and alcohol have occurred following abrupt discontinuance of benzodiazepine drugs. These symptoms include convulsions, tremor, abdominal and muscle cramps, vomiting and sweating. Addiction-prone individuals, such as drug addicts and alcoholics, should be under careful surveillance when receiving benzodiazepines because of the predisposition of such patients to habituation and dependence.

Precautions:
Use in Pregnancy and Lactation:
An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. Verstran (prazepam) a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug. In view of their molecular size, Verstran and its metabolites are probably excreted in human milk. Therefore, this drug should not be given to nursing mothers.

In those patients in whom a degree of depression accompanies the anxiety, suicidal tendencies may be present, and protective measures may be required. The least amount of drug that is feasible should be available to the patient at any one time.

Patients taking Verstran for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed. Hepatomegaly and cholestasis were observed in chronic toxicity studies in rats and dogs.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

Pediatric Use:
Safety and effectiveness in patients below the age of 18 have not been established.

Adverse Reactions: The side effects most frequently reported during double-blind placebo-controlled trials employing a typical 30 mg divided total daily dose and the percent incidence in the Verstran (prazepam) group were: fatigue (11.6%), dizziness (8.7%), weakness (7.7%), drowsiness (6.8%), lightheadedness (6.8%), and ataxia (6.0%). Less frequently reported were: headache, confusion, tremor, vivid dreams, slurred speech, palpitation, stimulation, dry mouth, diaphoresis, and various gastrointestinal complaints. Other side effects included: pruritus, transient skin rashes, swelling of feet, joint pains, various genitourinary complaints, blurred vision, and syncope. Single nightly dose, controlled trials of variable dosages showed a dose-related incidence of these same side effects. Transient and reversible abnormalities of liver function tests have been reported, as have slight decreases in blood pressure and increases in body weight.

These findings are characteristic of benzodiazepine drugs.

Overdosage: As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

If vomiting has not occurred spontaneously, it should be induced. Immediate gastric lavage is also recommended. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though unlikely, may be controlled with Levophed* (levarterenol bitartrate) or Aramine* (metaraminol bitartrate). Caffeine and Sodium Benzoate Injection, USP, may be used to counteract central nervous system depressant effects.

Dosage and Administration: Verstran (prazepam) is administered orally in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 20 to 60 mg daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a divided daily dose of 10 mg to 15 mg. (See Precautions.)

Prazepam may also be administered as a single dose daily at bedtime. The recommended starting nightly dose is 20 mg. The response of the patient to several days' treatment will permit the physician to adjust the dose upward or, occasionally, downward to maximize antianxiety effect with a minimum of daytime drowsiness. The optimum dosage will usually range from 20 to 40 mg.

Drug Interactions: If Verstran (prazepam) is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monamine oxidase inhibitors or other antidepressants.

If Verstran is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

How Supplied: Verstran (prazepam) 10 mg light blue, scored tablets in bottles of 100 (N 0047-0276-51).

STORE BETWEEN 59°-86° F (15°-30° C).

*Please see this page for warnings, precautions and adverse reactions.

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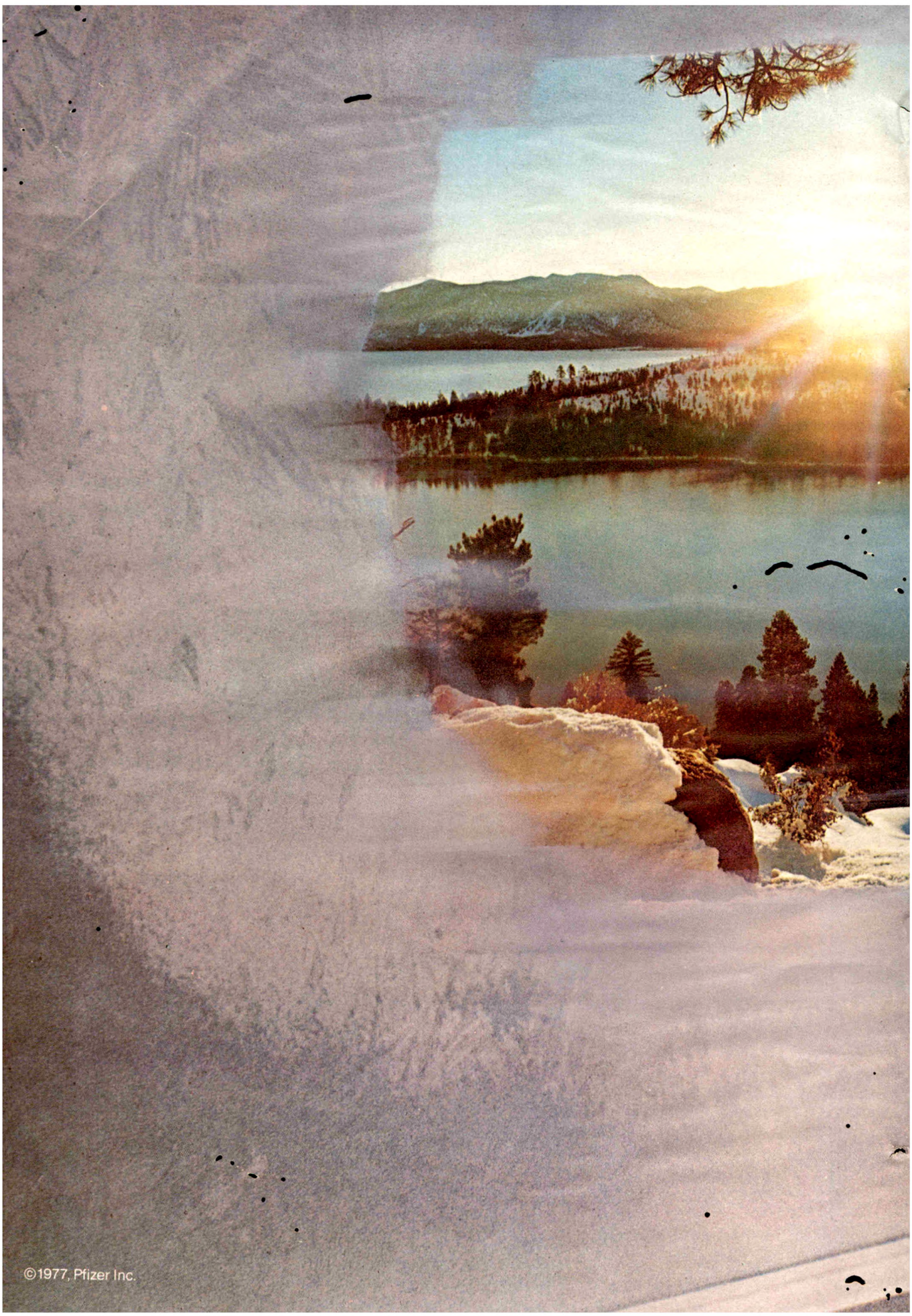
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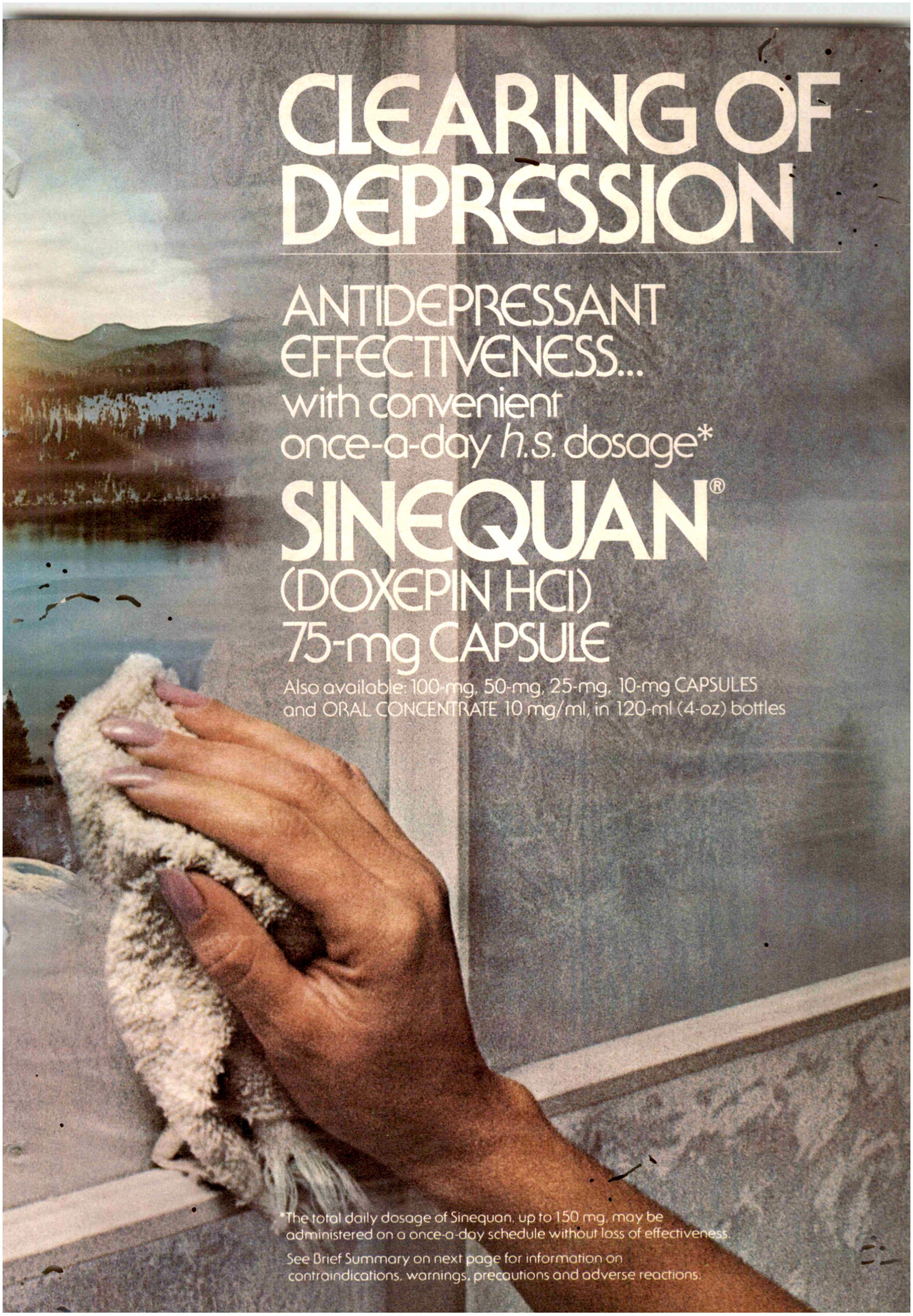
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CLEARING OF DEPRESSION
ANTIDEPRESSANT EFFECTIVENESS... with convenient once-a-day h.s. dosage*
SINEQUAN®
(DOXEPIN HCl)
75-mg CAPSULE
Also available: 100-mg, 50-mg, 25-mg, 10-mg CAPSULES and ORAL CONCENTRATE 10 mg/ml, in 120-ml (4-oz) bottles
*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.
See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

IN THE TREATMENT OF CLINICAL DEPRESSION—

SINEQUAN® 75 mg (DOXEPIN HCl) CAPSULE

PROVIDES ANTIDEPRESSANT EFFECTIVENESS

IN ADDITION TO OTHER, OFTEN BENEFICIAL, PROPERTIES...

CONVENIENT ONCE-A-DAY h.s. DOSAGE

which may increase patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and early-morning awakening often associated with depression.

MARKED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have occasionally been reported. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind.

Warnings. The once-a-day dosage regimen of SINEQUAN in patients with intercurrent illness or patients taking other medications should be carefully adjusted. This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus. Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established. There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established.

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors. Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug. The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved.

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively.

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug.

Patients should also be cautioned that their response to alcohol may be potentiated.

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy.

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE: Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN.

Anticholinergic Effects: Dry mouth, blurred vision, constipation, and urinary retention have been reported. If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage.

Central Nervous System Effects: Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued. Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular: Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic: Skin rash, edema, photosensitization, and pruritus have occasionally occurred.

Hematologic: Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura.

Gastrointestinal: Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects.)

Endocrine: Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other: Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects.

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting dose of 25 mg t.i.d. is recommended. Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day.

In more severely ill patients an initial dose of 50 mg t.i.d. may be required with subsequent gradual increase to 300 mg/day if necessary. Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day.

As an alternative regimen, the total daily dosage, up to 150 mg, may be given on a once-a-day schedule without loss of effectiveness. This dose may be given at bedtime.

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A. Signs and Symptoms

1. Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.
2. Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes.

B. Management and Treatment

1. Mild: Observation and supportive therapy is all that is usually necessary.
2. Severe: Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy. If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed. The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary. EKG monitoring may be required for several days, since relapse after apparent recovery has been reported. Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy; however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN.

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to 10 mg, 25 mg, 50 mg, 75 mg, and 100 mg doxepin in bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). SINEQUAN (doxepin HCl) 25 mg and 50 mg are also available in bottles of 5000. SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg. Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages. For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended.

More detailed professional information available on request.

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The American Psychiatric Association announces publication of two volumes reporting the Conference on Education of Psychiatrists

held at Lake of the Ozarks, Missouri, June 9-15, 1975. Organized by the A.P.A. in cooperation with the American Academy of Child Psychiatry, the American Association of Chairmen of Departments of Psychiatry, and the American Association of Directors of Psychiatric Residency Training.

1. PSYCHIATRIC EDUCATION: PROLOGUE TO THE 1980's, Library Bound, 544 pages, $15.00.

This is a substantive, highly readable, report of the Conference, written by Anne H. Rosenfeld with an Editorial Board comprising: Ewald W. Busse, M.D., Chairperson of the Conference, Walter E. Barton, M.D., Bernard Holland, M.D., Donna Norris, M.D., Melvin Sabshin, M.D., Jeanne Spurlock, M.D., and Robert L. Robinson, M.A. (Staff Consultant).

The volume contains ten chapters covering the topical structure of the Conference: Goals and Values; Competence, Evaluation, and Quality Control; The Resident; Faculty; Educational Methods: Means in the Service of Ends; The Program Environment; Research; The Economics of Residency Training; Psychiatry in Its Social Context; and Retrospect and Prospect. It also contains nine appendices describing the organization of the Conference and its participants, together with selected model programs and related informational materials of value.

2. THE WORKING PAPERS OF THE 1975 CONFERENCE ON EDUCATION OF PSYCHIATRISTS Paper bound, 432 pages, $12.50.

Sixty-two leading psychiatric educators served on seven preparatory commissions, one for each of seven topic areas, and prepared advance working papers as a basis for Conference deliberations. The topics and Commission Chairpersons were: Social Issues and Needs (James N. Sussex, M.D.); Goals and Objectives (Morton F. Reiser, M.D.); The Resident (David R. Hawkins, M.D.); Content, Methodology, Faculty and Environment (Milton Greenblatt, M.D.); Research (Daniel X. Freedman, M.D.); Economics (Herzl Spiro, M.D.); Evaluation and Quality Control (Eric Pfeiffer, M.D.). The Chairpersons, together with the Conference Chairperson, served as the Editorial Board for this volume.

Papers commenting on the reports of the commissions, published here for the first time, were presented at the Conference by Melvin Sabshin, M.D., Eugene B. Brody, M.D., Jeanne Spurlock, M.D., Jack R. Ewalt, M.D., Robert J. Stoller, M.D., James R. Comer, M.D., and Bernard Holland, M.D.

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Basic tool of primitive psychiatry



Fertility protective statues: For pregnant women, the statues insured a healthy, beautiful child, and protected both mother and child from the "evil eye." For sterile women, the statues' magical power aided fertility. Ashanti, Ghana.

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Basic tool of modern psychiatry

Thorazine®

brand of **chlorpromazine**

Tablets: 25 and 50 mg of the HCl

- Effective control of psychotic symptoms
- Especially useful in agitated, violent or anxious schizophrenic patients
- Unsurpassed clinical experience
- 18 convenient dosage forms and strengths

Before prescribing, see complete prescribing information in SK&F literature or *PDR*. The following is a brief summary.

Indications

Based on a review of this drug by the National Academy of Sciences–National Research Council and/or other information, FDA has classified the indications as follows:

Effective: For the management of manifestations of psychotic disorders. For control of the manifestations of manic-depressive illness (manic phase).

Probably effective: For the control of moderate to severe agitation, hyperactivity or aggressiveness in disturbed children.

Possibly effective: For control of excessive anxiety, tension and agitation as seen in neuroses.

Final classification of the less-than-effective indications requires further investigation.

Contraindications: Comatose states, presence of large amounts of C.N.S. depressants, or bone marrow depression.

Warnings:

Caution is recommended when 'Thorazine' is administered for the treatment of vomiting in children.

Antiemetics are not recommended to treat uncomplicated short-term vomiting in children or vomiting of unknown etiology.

The possibility of extrapyramidal reactions from 'Thorazine' may confuse the diagnosis of Reye's syndrome or other encephalopathy. Although unconfirmed, some suspicion exists that centrally-acting antiemetics may contribute to or adversely affect the course of Reye's syndrome, and should be avoided in suspected cases.

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Avoid using in patients hypersensitive (e.g., blood dyscrasia, jaundice) to any phenothiazine. Caution patients about activities requiring alertness (e.g., operating vehicles or machinery) especially during the first few days' therapy. Avoid concomitant use with alcohol. May counteract antihypertensive effect of guanethidine and related compounds.

Use in pregnancy only when essential. There are reported instances of jaundice or prolonged extrapyramidal signs in newborn whose mothers had received chlorpromazine.

Precautions: Use cautiously in persons with cardiovascular, liver or chronic respiratory disease, or with acute respiratory infections. Due to cough reflex suppression, aspiration of vomitus is possible. May prolong or intensify the action of C.N.S. depressants, organophosphorus insecticides, heat, atropine and related drugs. (Reduce dosage of concomitant C.N.S. depressants.) Anticonvulsant action of barbiturates is not intensified. Antiemetic effect may mask signs of toxic drug overdosage or physical disorders. Discontinue high-dose, long-term therapy gradually.

Patients on long-term therapy, especially high doses, should be evaluated periodically for possible adjustment or discontinuance of drug therapy.

Adverse Reactions: Drowsiness, cholestatic jaundice, agranulocytosis, eosinophilia, leukopenia, hemolytic anemia, thrombocytopenic purpura and pancytopenia; postural hypotension, tachycardia, fainting, dizziness and, occasionally, a shock-like condition; reversal of epinephrine effects; EKG changes have been reported, but relationship to myocardial damage is not confirmed; neuromuscular (extrapyramidal) reactions; pseudoparkinsonism, motor restlessness, dystonias, persistent tardive dyskinesia, hyperreflexia in the newborn; psychotic symptoms, catatonic-like states, cerebral edema; convulsive seizures; abnormality of the cerebrospinal fluid proteins; urticarial reactions and photosensitivity, exfoliative dermatitis, contact dermatitis; lactation and breast engorgement (in females on large doses), false positive pregnancy tests, amenorrhea, gynecomastia; hyperglycemia, hypoglycemia, glycosuria; dry mouth, nasal congestion, constipation, adynamic ileus, urinary retention, miosis, mydriasis; after prolonged substantial doses, skin pigmentation, epithelial keratopathy, lenticular and corneal deposits and pigmentary retinopathy, visual impairment; mild fever (after large I.M. dosage); hyperpyrexia; increased appetite and weight; a systemic lupus erythematosus-like syndrome; peripheral edema.

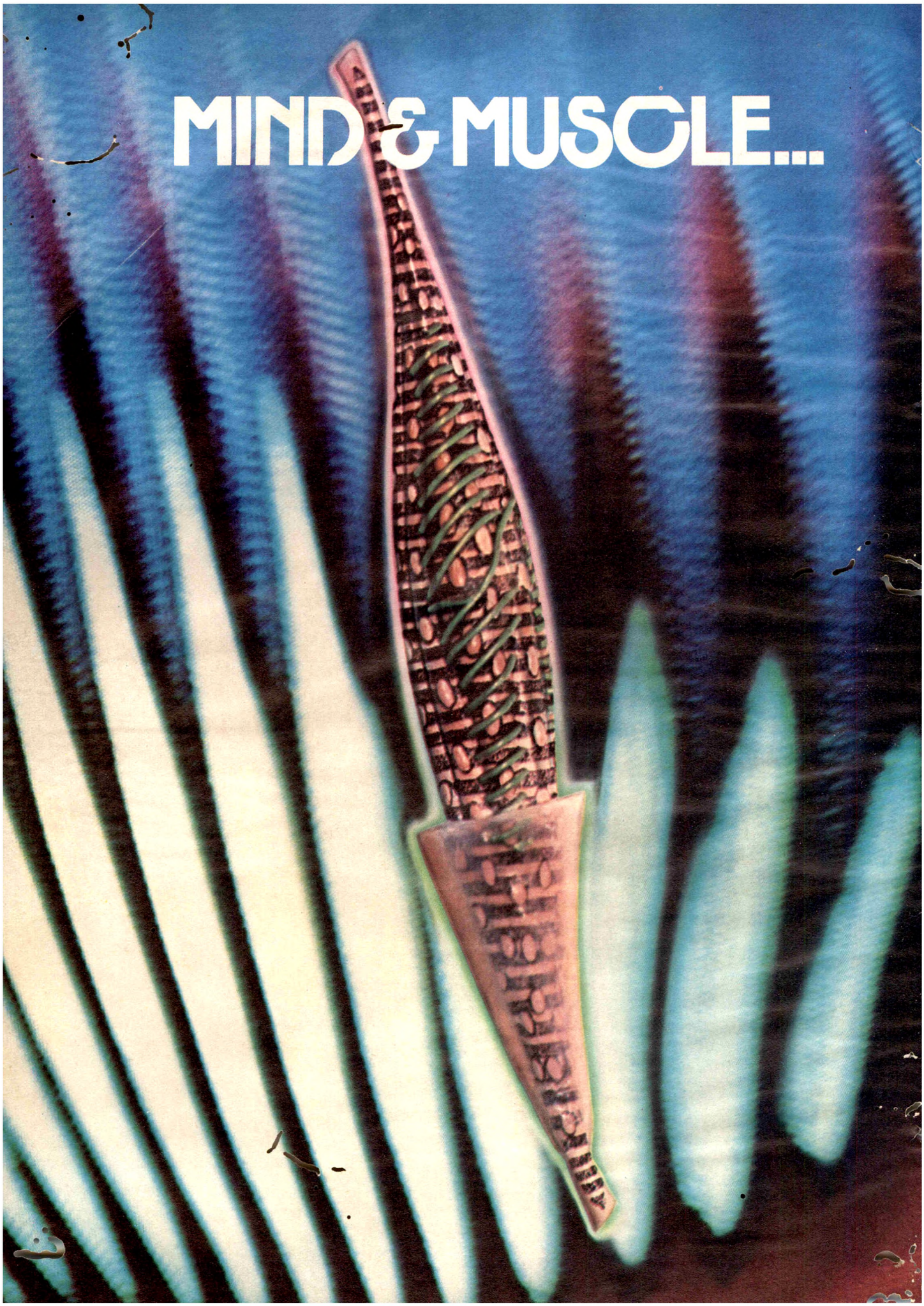
NOTE: Sudden death in patients taking phenothiazines (apparently due to cardiac arrest or asphyxia due to failure of cough reflex) has been reported, but no causal relationship has been established.

Supplied: Tablets, 10 mg., 25 mg., 50 mg., 100 mg. and 200 mg., in bottles of 100; Single Unit Packages of 100 (intended for institutional use only). Spansule® capsules, 30 mg., 75 mg., 150 mg., 200 mg. and 300 mg., in bottles of 50; in Single Unit Packages of 100 (intended for institutional use only). Injection, 25 mg./ml.; Syrup, 10 mg./5 ml.; Suppositories, 25 mg. and 100 mg. Concentrate (intended for institutional use only), 30 mg./ml. and 100 mg./ml.

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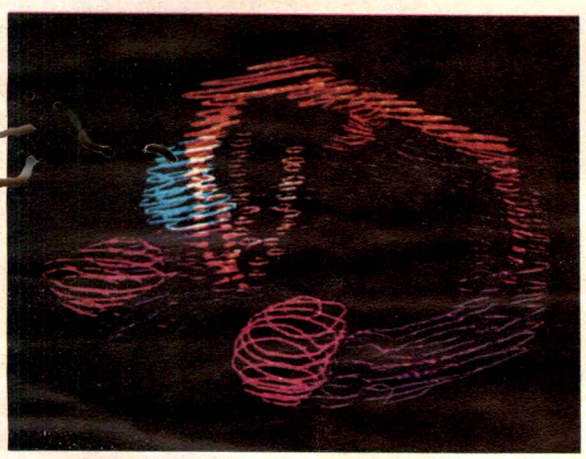
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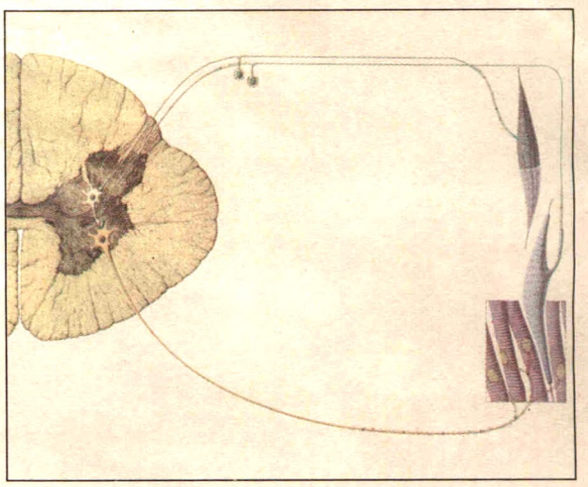
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Preliminary studies in both animals and humans have suggested that Valium (diazepam) may also work at the spinal level by enhancing presynaptic inhibition, a mechanism believed to diminish spasm in skeletal muscle. Clinically, adjunctive Valium has significant skeletal muscle relaxant properties.

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2-MG, 5-MG, 10-MG SCORED TABLETS

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ROCHE

VALIUM®
(diazepam)

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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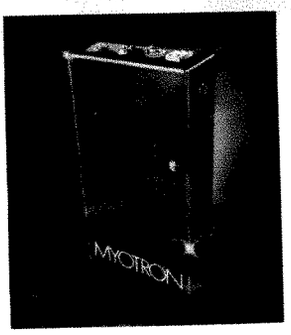
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in clinically significant depression...

penetrating the symptom barrier to productive psychotherapy

symptoms often interfere with therapy

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meeting therapeutic goals

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ELAVIL should not be used during the acute recovery phase following myocardial infarction; in patients hypersensitive to it; in those who have received an MAOI within two weeks; or in children under 12. Patients with cardiovascular disorders should be watched closely. Safe use during pregnancy and lactation has not been established. The drug may impair mental or physical abilities required in the performance of hazardous tasks and may enhance the response to alcohol. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Hospitalize as soon as possible any patient suspected of having taken an overdose.

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• For a brief summary of prescribing information, please see following page.

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addendum

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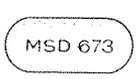


A single 50-mg tablet
Minimum starting dosage



Dosage may be increased by 25 or 50 mg as necessary until a total of 150 mg per day is reached.

A single 150-mg tablet
Maximum daily dosage



ELAVIL MAY ALSO BE PRESCRIBED IN DIVIDED DAILY DOSES

The 25-mg tablet



This strength may prove useful when therapy is initiated with divided daily doses in adult outpatients. Starting dosage is usually 75 mg daily. If necessary, this dosage may be increased gradually to a total of 150 mg a day. Increases are made preferably in the late afternoon or bedtime dose.

The 10-mg tablet



This strength may prove useful for patients who require lower doses, e.g., adolescent and elderly patients. For these patients who can not tolerate higher doses, 10 mg three times a day with 20 mg at bedtime may be satisfactory.

A sedative effect may be apparent before the antidepressant effect of ELAVIL is noted. An adequate therapeutic effect may take as long as 30 days to develop.

NOTE: The usual maintenance dosage of ELAVIL is 50 to 100 mg per day which may be given in a single dose preferably at bedtime. In some patients 40 mg per day is sufficient. This drug is not recommended for patients under 12 years of age.

Contraindications: Known hypersensitivity. Should not be given concomitantly with monoamine oxidase inhibitor since hyperpyretic crises, severe convulsions, and deaths have occurred. When used to replace a monoamine oxidase inhibitor, allow a minimum of 14 days to elapse before initiating therapy with amitriptyline HCl. Initiate dosage of amitriptyline HCl cautiously with gradual increase in dosage until optimum response is achieved. Not recommended during the acute recovery phase following myocardial infarction.

Warnings: May block the antihypertensive action of guanethidine or similarly acting compounds. Should be used with caution in patients with a history of seizures or a history of urinary retention, or with angle-closure glaucoma or increased intraocular pressure; in patients with angle-closure glaucoma, even average doses may precipitate an attack. Patients with cardiovascular disorders should be watched closely; arrhythmias, sinus tachycardia and prolongation of the conduction time have been reported, particularly with high doses; myocardial infarction and stroke have been reported with drugs of this class. Close supervision is required for hyperthyroid patients or those receiving thyroid medication. May impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle. In patients who use alcohol excessively, potentiation may increase the danger inherent in any suicide attempt or overdosage. Safe use during pregnancy and lactation has not been established; in pregnant patients, nursing mothers, or women who may become pregnant, weigh possible benefits against possible hazards to mother and child. Not recommended for patients under 12 years of age.

Precautions: Schizophrenic patients may develop increased symptoms of psychosis; patients with paranoid symptomatology may have an exaggeration of such symptoms; manic depressive patients may experience a shift to the manic phase. In these circumstances, the dose of amitriptyline HCl may be reduced or a major tranquilizer, such as perphenazine, may be administered concurrently.
When given with anticholinergic agents or sympathomimetic drugs, including epinephrine combined with local anesthetics, close supervision and careful adjustment of dosages are required; paralytic ileus may occur in patients taking tricyclic antidepressants in combination with anticholinergic-type drugs. Use cautiously in patients receiving large doses of ethchlorvynol, since transient delirium has been reported on concurrent administration. May enhance the response to alcohol and the effects of barbiturates and other CNS depressants. The possibility of suicide in depressed patients remains until significant remission occurs. Potentially suicidal patients should not have access to large quantities of this drug. Prescriptions should be written for the smallest amount feasible. Concurrent electroshock therapy may increase the hazards associated with such therapy; such treatment should be limited to patients for whom it is essential. When possible, discontinue the drug several days before elective surgery. Both elevation and lowering of blood sugar levels have been reported. Use with caution in patients with impaired liver function.

Adverse Reactions: *Note:* Included in this listing are a few adverse reactions not reported with this specific drug. However, pharmacological similarities among the tricyclic antidepressant drugs require that each reaction be considered when amitriptyline is administered. *Cardiovascular:* Hypotension, hypertension, tachycardia, palpitation, myocardial infarction, arrhythmias, heart block, stroke. *CNS and Neuromuscular:* Confusional states; disturbed concentration; disorientation; delusions; hallucinations; excitement; anxiety; restlessness; insomnia; nightmares; numbness, tingling, and paresthesias of the extremities; peripheral neuropathy; incoordination; ataxia; tremors; seizures; alteration in EEG patterns; extrapyramidal symptoms; tinnitus; syndrome of inappropriate ADH (antidiuretic hormone) secretion. *Anticholinergic:* Dry mouth, blurred vision, disturbance of accommodation, increased intraocular pressure, constipation, paralytic ileus, urinary retention, dilatation of urinary tract. *Allergic:* Skin rash, urticaria, photosensitization, edema of face and tongue. *Hematologic:* Bone marrow depression including agranulocytosis, leukopenia, eosinophilia, purpura, thrombocytopenia. *Gastrointestinal:* Nausea, epigastric distress, vomiting, anorexia, stomatitis, peculiar taste, diarrhea, parotid swelling, black tongue, rarely hepatitis (including altered liver function and jaundice) *Endocrine:* Testicular swelling and gynecomastia in the male, breast enlargement and galactorrhea in the female, increased or decreased libido, elevation and lowering of blood sugar levels. *Other:* Dizziness, weakness, fatigue, headache, weight gain or loss, increased perspiration, urinary frequency, mydriasis, drowsiness, alopecia. *Withdrawal Symptoms:* Abrupt cessation of treatment after prolonged administration may produce nausea, headache, and malaise; these are not indicative of addiction.

Overdosage: Hospitalize as soon as possible all patients suspected of having taken an overdose. Treatment is symptomatic and supportive. In addition, the intravenous administration of 1 to 3 mg physostigmine salicylate is reported to reverse the symptoms of tricyclic antidepressant poisoning. Because physostigmine is rapidly metabolized, the dosage should be repeated as required, particularly if life-threatening signs such as arrhythmias, convulsions, and deep coma recur or persist after the initial dosage of physostigmine.

How Supplied: Tablets containing 10 mg and 25 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100, 1000, and 5000; tablets containing 50 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100 and 1000; tablets containing 75 mg and 100 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 100; tablets containing 150 mg amitriptyline HCl, in single-unit packages of 100 and bottles of 30 and 100; for intramuscular use, in 10-ml vials containing per ml: 10 mg amitriptyline HCl, 44 mg dextrose, 1.5 mg methylparaben and 0.2 mg propylparaben as preservatives, and water for injection q.s. 1 ml.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486

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By Louis S. Reed, Ph.D.
Consultant in Health Economics, American Psychiatric Association

This book reports six studies that add to the growing body of data demonstrating the feasibility of covering mental illness under health insurance. Although in some ways it may be considered a supplement to APA's 1972 book **HEALTH INSURANCE AND PSYCHIATRIC CARE: UTILIZATION AND COST** (Reed, Myers, and Scheidemandel), all of the data are new. The studies cover the following areas:

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ACTIONS—Pharmacologically, **TRANXENE** (clorazepate dipotassium) has the characteristics of the benzodiazepines. It has depressant effects on the central nervous system. The primary metabolite, nordiazepam, quickly appears in the blood stream. The serum half-life is about 2 days. The drug is metabolized in the liver and excreted primarily in the urine.

INDICATIONS—**TRANXENE** is indicated for the symptomatic relief of anxiety associated with anxiety neurosis, in other psychoneuroses in which anxiety symptoms are prominent features, and as an adjunct in disease states in which anxiety is manifested.

TRANXENE is indicated for the symptomatic relief of acute alcohol withdrawal.

The effectiveness of **TRANXENE** for long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should reassess periodically the usefulness of the drug for the individual patient.

CONTRAINDICATIONS—**TRANXENE** is contraindicated in patients with a known hypersensitivity to the drug, and in those with acute narrow angle glaucoma.

WARNINGS—**TRANXENE** is not recommended for use in depressive neuroses or in psychotic reactions.

Patients on **TRANXENE** should be cautioned against engaging in hazardous occupations requiring mental alertness, such as operating dangerous machinery including motor vehicles.

Since **TRANXENE** has a central nervous system depressant effect, patients should be advised against the simultaneous use of other CNS-depressant drugs, and cautioned that the effects of alcohol may be increased.

Because of the lack of sufficient clinical experience, **TRANXENE** is not recommended for use in patients less than 18 years of age.

Physical and Psychological Dependence:
Withdrawal symptoms (similar in character to those noted with barbiturates and alcohol) have occurred following abrupt discontinuance of clorazepate. Symptoms of nervousness, insomnia, irritability, diarrhea, muscle aches and memory impairment have followed abrupt withdrawal after long-term use of high dosage.

Caution should be observed in patients who are considered to have a psychological potential for drug dependence.

Evidence of drug dependence has been observed in dogs and rabbits which was characterized by convulsive seizures when the drug was abruptly withdrawn or the dose was reduced; the syndrome in dogs could be abolished by administration of clorazepate.

Usage in Pregnancy:
An increased risk of congenital malformations associated with the use of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) during the first trimester of pregnancy has been suggested in several studies. TRANXENE, a benzodiazepine derivative, has not been studied adequately to determine whether it, too, may be associated with an increased risk of fetal abnormality. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. The possibility that a woman of childbearing potential may be pregnant at the time of institution of therapy should be considered. Patients should be advised that if they become pregnant during therapy or intend to become pregnant they should communicate with their physician about the desirability of discontinuing the drug.

Usage during Lactation:
TRANXENE should not be given to nursing mothers since it has been reported that nordiazepam is excreted in human breast milk.

PRECAUTIONS—In those patients in which a degree of depression accompanies the anxiety, suicidal tendencies may be present and protective measures may be required. The least amount of drug that is feasible should be available to the patient.

Patients on **TRANXENE** for prolonged periods should have blood counts and liver function tests periodically. The usual precautions in treating patients with impaired renal or hepatic function should also be observed.

In elderly or debilitated patients, the initial dose should be small, and increments should be made gradually, in accordance with the response of the patient, to preclude ataxia or excessive sedation.

ADVERSE REACTIONS—The side effect most frequently reported was drowsiness. Less commonly reported (in descending order of occurrence) were: dizziness, various gastrointestinal complaints, nervousness, blurred vision, dry mouth, headache, and mental confusion. Other side effects included insomnia, transient skin rashes, fatigue, ataxia, genitourinary complaints, irritability, diplopia, depression and slurred speech.

There have been reports of abnormal liver and kidney function tests and of decrease in hematocrit.

Decrease in systolic blood pressure has been observed.

DOSAGE AND ADMINISTRATION—
For symptomatic relief of anxiety:
TRANXENE is administered orally. The capsules may be given in divided doses. The usual daily dose is 30 mg. The dose should be adjusted gradually within the range of 15 to 60 mg. daily in accordance with the response of the patient. In elderly or debilitated patients it is advisable to initiate treatment at a daily dose of 7.5 to 15 mg.

TRANXENE capsules may also be administered as a single dose daily at bedtime; the recommended initial dose is 15 mg. After the initial dose, the response of the patient may require adjustment of subsequent dosage. Lower doses may be indicated in the elderly patient. Drowsiness may occur at the initiation of treatment and with dosage increment.

TRANXENE-SD tablets (22.5 mg.) may be administered as a single dose every 24 hours. This tablet is intended as an alternate dosage form for the convenience of patients stabilized on a dose of 7.5 mg. capsules three times a day. TRANXENE-SD tablets should not be used to initiate therapy.

TRANXENE-SD HALF STRENGTH tablets (11.25 mg.) may be administered as a single dose every 24 hours.

For symptomatic relief of acute alcohol withdrawal:
Recommended schedule: 1st 24 hours, 30 mg **TRANXENE** initially, followed by 30 to 60 mg in divided doses; 2nd 24 hours, 45 to 90 mg in divided doses; 3rd 24 hours, 22.5 to 45 mg in divided doses; 4th day 15 to 30 mg in divided doses. Thereafter gradually reduce to 7.5 to 15 mg daily, and discontinue as soon as condition is stable. Maximum daily dose is 90 mg. Avoid excessive reductions in total drug on successive days.

DRUG INTERACTIONS—If **TRANXENE** is to be combined with other drugs acting on the central nervous system, careful consideration should be given to the pharmacology of the agents to be employed. Animal experience indicates that **TRANXENE** prolongs the sleeping time after hexobarbital or after ethyl alcohol, increases the inhibitory effects of chlorpromazine, but does not exhibit monoamine oxidase inhibition. Clinical studies have shown increased sedation with concurrent hypnotic medications. The actions of the benzodiazepines may be potentiated by barbiturates, narcotics, phenothiazines, monoamine oxidase inhibitors or other antidepressants.

If **TRANXENE** (clorazepate dipotassium) is used to treat anxiety associated with somatic disease states, careful attention must be paid to possible drug interaction with concomitant medication.

MANAGEMENT OF OVERDOSAGE—Overdosage is usually manifested by varying degrees of CNS depression ranging from slight sedation to coma. As in the management of overdosage with any drug, it should be borne in mind that multiple agents may have been taken.

There are no specific antidotes for the benzodiazepines. The treatment of overdosage should consist of the general measures employed in the management of overdosage of any CNS depressant. Gastric evacuation either by the induction of emesis, lavage, or both, should be performed immediately. General supportive care, including frequent monitoring of the vital signs and close observation of the patient, is indicated. Hypotension, though rarely reported, may occur with large overdoses. In such cases the use of agents such as Levophed® (levarterenol) or Aramine® (metaraminol) should be considered.

While reports indicate that individuals have survived overdoses of **TRANXENE** as high as 450 to 675 mg., these doses are not necessarily an accurate indication of the amount of drug absorbed, since the time interval between ingestion and the institution of treatment was not always known. Sedation in varying degrees was the most common physiological manifestation of **TRANXENE** overdosage. Deep coma when it occurred was usually associated with the ingestion of other drugs in addition to **TRANXENE**.

CLINICAL PHARMACOLOGY—Studies in healthy men have shown that **TRANXENE** has depressant effects on the central nervous system. Prolonged administration of single daily doses as high as 120 mg. was without toxic effects. Abrupt cessation of high doses was followed in some patients by nervousness, insomnia, irritability, diarrhea, muscle aches, or memory impairment.

Absorption–Excretion:
After oral administration of **TRANXENE**, there is essentially no circulating parent drug. Nordiazepam, its primary metabolite, quickly appears in the blood stream. In 2 volunteers given 15 mg. (50 μC) of ^{14}C-**TRANXENE**, about 80% was recovered in the urine and feces within 10 days. Excretion was primarily in the urine with about 1% excreted per day on day 10.

HOW SUPPLIED—**TRANXENE** (clorazepate dipotassium) is supplied as capsules in three dosage strengths:

3.75 mg. capsules (gray with white cap) in bottles of 100 (NDC 0074-3417-13) and 500 (NDC 0074-3417-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3417-11).
7.5 mg. capsules (gray with maroon cap) in bottles of 100 (NDC 0074-3418-13) and 500 (NDC 0074-3418-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3418-11).
15 mg. capsules (all gray) in bottles of 100 (NDC 0074-3419-13) and 500 (NDC 0074-3419-53). Also available in ABBO-PAC® unit dose packages of 100 capsules (NDC 0074-3419-11).

TRANXENE-SD single dose tablets are supplied in two dosage strengths:

TRANXENE-SD 22.5 mg. tablets (tan-colored) in bottles of 100 (NDC 0074-2997-13).

TRANXENE-SD HALF STRENGTH 11.25 mg. tablets (blue-colored) in bottles of 100 (NDC 0074-2699-13).

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four good reasons to make it

Tranxene® CLORAZEPATE DIPOTASSIUM

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1. Tranxene has been shown equivalent to diazepam in anti-anxiety effectiveness, in controlled studies with hundreds of patients.

2. Tranxene is remarkably well tolerated; excessive sedation is seldom a problem.

3. Tranxene offers oral dosing options unmatched by another tranquilizer: not only conventional divided dose forms, but also economical single-dose tablets for once-a-day use at any convenient hour.

4. Tranxene is physically different in form and appearance from other widely used agents. (Fewer patients will know or recognize it.)

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ABBOTT

Please see overleaf for prescribing information.

